W. B. MEACHAM.

Digitized by the Internet Archive
in 2025

https://archive.org/details/diseasesofnoseth0000will_e6k6

DISEASES

OF THE

NOSE, THROAT AND EAR

MEDICAL AND SURGICAL

BY

WILLIAM LINCOLN BALLENGER, M.D.

PROFESSOR OF OTOLOGY, RHINOLOGY, AND LARYNGOLOGY, COLLEGE OF PHYSICIANS AND SURGEONS DEPARTMENT OF MEDICINE, UNIVERSITY OF ILLINOIS, CHICAGO; FELLOW OF THE AMERICAN LARYNGOLOGICAL ASSOCIATION; FELLOW OF THE AMERICAN LARYNGOLOGICAL, RHINOLOGICAL, AND OTOLOGICAL ASSOCIATION; FELLOW OF THE AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY, ETC.

FOURTH EDITION, REVISED AND ENLARGED

ILLUSTRATED WITH 536 ENGRAVINGS AND 33 PLATES

LEA & FEBIGER

PHILADELPHIA AND NEW YORK

1914.

Entered according to Act of Congress, in the year 1914, by
LEA & FEBIGER
in the Office of the Librarian of Congress. All rights reserved.

PREFACE TO THE FOURTH EDITION

THE distinguishing feature of this new edition will be found in its chapters on the Labyrinth. Great labor has been bestowed in marshalling the facts and formulating them for teaching purposes. Thirteen original colored plates now illustrate the physiological and pathological manifestations of nystagmus. A careful study of these alone will suffice to convey a clear idea of the subject. The new matter on the Labyrinth amounts to over one hundred pages. The author is indebted to Dr. J. R. Fletcher for the section on General Diagnosis of Labyrinth Disease, which he has revised. Twelve drawings illustrate the Neumann and the Hinsberg labyrinth operations.

The call for a new edition has been utilized to subject the entire book to a searching revision and to bring it fully to date. Among other new matters may be mentioned the full description of Mosher's fronto-ethmoid operation, with five drawings, showing each step. Mosher's technique is a distinct advance in the surgery of the sinuses. Autogenous vaccines in the treatment of hay fever, as advocated by Dr. T. M. Farrington, are given place, though this remedy has not yet fully proved its value. Space is given to it with the hope that others will subject it to a thorough trial, as it offers a fruitful field for research. Dr. Alfred Lewy has rewritten the section on Functional Tests of Hearing. Otosclerosis has been extensively revised and brought fully to date. Haynes' operation on the cisterna magna is fully described, and five drawings illustrate the technique. Vaccine therapy has been revised, and the His leukocyte-extract therapy is given in detail. It forms a distinct advance in the treatment of certain forms of infectious diseases, especially of the nasal sinuses and meninges. Meningitis has been largely rewritten, with much new material. The section on Abscess of the Brain has been revised by Dr. Howard Charles Ballenger. The use of salvarsan in the treatment of syphilis of the brain and auditory nerve is described with great fulness. It forms an important addition to this edition. Dr. George McBeam's theory of the causation of paracusis Willisii is given in full. In a word, every

line of the book has been revised, all obsolete matter has been eliminated, and much new text has been incorporated, with many new illustrations and plates, all of which were drawn by the author. He believes that the work, thus brought to date, affords a well-balanced presentation of its closely related specialties, and that it covers the field. He hopes, therefore, that it will continue to enjoy the favor which has called for four large editions in six years.

The author takes this opportunity of thanking Dr. C. E. Robb, who has read the proof, and Dr. Howard Charles Ballenger, his professional associate, who has also rendered valuable assistance in the proof-reading and in the revision of the section on Intracranial Complications in Aural Disease. Dr. Robb has also carefully revised the entire text for the avoidance of errors.

W. L. B.

CHICAGO, 1914.

CONTENTS

PART I

THE NOSE AND ACCESSORY SINUSES

CHAPTER I

THE CLINICAL ANATOMY AND PHYSIOLOGY OF THE NOSE AND ACCESSORY SINUSES 17

CHAPTER II

THE NOSE, THROAT, AND EAR IN RELATION TO GENERAL MEDICINE 26

CHAPTER III

THE OFFICE EQUIPMENT 36

CHAPTER IV

THE ETIOLOGY OF DEFORMITIES AND DEVIATIONS OF THE SEPTUM NASI 57

CHAPTER V

THE CHOICE OF SEPTUM OPERATIONS. THE SURGICAL CORRECTION OF OBSTRUCTIVE MALFORMATIONS OF THE SEPTUM 67

CHAPTER VI

THE ETIOLOGY OF INFLAMMATORY DISEASES OF THE NOSE AND ACCESSORY SINUSES 110

CHAPTER VII

THE METHODS FOR PROMOTING THE REACTION OF INFLAMMATION 122

CHAPTER VIII

INFLAMMATORY DISEASES OF THE NOSE 130

CHAPTER IX

THE INDIVIDUAL SINUSES 161

CHAPTER X

GENERAL CONSIDERATIONS IN REFERENCE TO THE SINUSES 176

CHAPTER XI

THE SURGERY OF THE ACCESSORY SINUSES 199

CHAPTER XII

NASAL NEUROSES. NASAL HYDRORRHEA. CEREBROSPINAL RHINORRHEA 253

CHAPTER XIII

NEOPLASMS OF THE NOSE 269

CHAPTER XIV

EPISTAXIS (NASAL HEMORRHAGE). RHINOSCLEROMA. FURUNCULOSIS. SCREW-WORMS 284

CHAPTER XV

SURGICAL CORRECTION OF EXTERNAL NASAL DEFORMITIES . 291

CHAPTER XVI

CHRONIC GRANULOMATA OF THE NOSE, THROAT, AND EAR . 304

PART II

THE PHARYNX AND FAUCES

CHAPTER XVII

DISEASES OF THE EPIPHARYNX AND BASE OF THE TONGUE 331

CHAPTER XVIII

INFLAMMATORY DISEASES OF THE MESOPHARYNX AND FAUCES 352

CHAPTER XIX

FUNCTIONAL NEUROSES OF THE PHARYNX 364

CHAPTER XX

NEOPLASMS OF THE PHARYNX 368

CHAPTER XXI

DISEASES OF THE FAUCES AND TONSILS 379

CHAPTER XXII

INFLAMMATORY DISEASES OF THE TONSIL 395

CHAPTER XXIII

SURGERY OF THE TONSILS 414

CHAPTER XXIV

NEOPLASMS OF THE TONSIL 442

PART III

DISEASES OF THE LARYNX

CHAPTER XXV

INFLAMMATORY DISEASES OF THE LARYNX AND EPIGLOTTIS 447

CHAPTER XXVI

PACHYDERMIA LARYNGIS. MALFORMATIONS AND DEFORMITIES. PROLAPSE OF THE VENTRICLES. STENOSIS. SUBGLOTTIC STENOSIS 500

CHAPTER XXVII

NEUROSES OF THE LARYNX 508

CHAPTER XXVIII

THE SINGING VOICE 525

CHAPTER XXIX

DEFECTS OF SPEECH 536

CHAPTER XXX

NEOPLASMS OF THE LARYNX 544

CHAPTER XXXI

FOREIGN BODIES IN THE LARYNX, TRACHEA, BRONCHI, AND ESOPHAGUS 577

PART IV

THE EAR

CHAPTER XXXII

THE CLINICAL ANATOMY AND PHYSIOLOGY OF THE EAR . . 601

CHAPTER XXXIII

THE FUNCTIONAL TESTS OF HEARING 616

CHAPTER XXXIV

GENERAL ETIOLOGY OF DEFECTIVE HEARING 630

CHAPTER XXXV

FOREIGN BODIES IN THE EAR. CERUMINOUS PLUGS IN THE MEATUS 635

CHAPTER XXXVI

MALFORMATIONS AND NEOPLASMS OF THE AURICLE . . . 645

CHAPTER XXXVII

DISEASES OF THE AURICLE AND EXTERNAL MEATUS . . . 654

CHAPTER XXXVIII

MALFORMATIONS AND DISEASES OF THE MEMBRANA TYMPANI 669

CHAPTER XXXIX

DISEASES OF THE EUSTACHIAN TUBES 683

CHAPTER XL

THE PRINCIPLES AND METHODS OF TYMPANIC INFLATION . 692

CHAPTER XLI

INFLAMMATORY DISEASES OF THE TYMPANUM 703

CHAPTER XLII

OTOSCLEROSIS; SPONGIFYING OF THE BONY CAPSULE OF THE LABYRINTH 735

CHAPTER XLIII

ACUTE AND CHRONIC SUPPURATIVE OTITIS MEDIA. CHOLESTEATOMA 742

CHAPTER XLIV

THE SEQUELÆ OF SUPPURATIVE OTITIS MEDIA, MASTOIDITIS, AND CHOLESTEATOMA. SUPPURATION OF THE LABYRINTH 765

CHAPTER XLV

PRINCIPLES OF TREATMENT AND GENERAL CONSIDERATIONS IN SUPPURATIVE OTITIS MEDIA 774

CHAPTER XLVI

GENERAL PATHOLOGY OF OTITIS MEDIA AND MASTOIDITIS . 784

CHAPTER XLVII

INTRACRANIAL AND JUGULAR PYOGENIC DISEASES OF OTITIC ORIGIN 789

CHAPTER XLVIII

SURGERY OF THE TEMPORAL BONE 819

CHAPTER XLIX

THE LABYRINTH: ITS PHYSIOLOGY, FUNCTIONAL TESTS AND DISEASE 861

CHAPTER L

LABYRINTHITIS: ILLUSTRATIVE CASES 917

CHAPTER LI

SURGICAL DISEASE OF THE LABYRINTH 926

CHAPTER LII

SURGERY OF THE LABYRINTH 953

CHAPTER LIII

FACIAL PARALYSIS 993

CHAPTER LIV

NON-SURGICAL DISEASES OF THE LABYRINTH 1000

CHAPTER LV

DEAF-MUTISM 1023

DISEASES OF NOSE, THROAT, AND EAR

PART I

THE NOSE AND ACCESSORY SINUSES

CHAPTER I

THE CLINICAL ANATOMY AND PHYSIOLOGY OF THE NOSE AND ACCESSORY SINUSES

THE NOSE

The Nasal Chambers.—The nose is divided, by the nasal septum, into two chambers, the right and the left. The nasal chambers are for respiratory, olfactory, phonatory, and gustatory purposes. The inspiratory current passes upward from the vestibules to the middle and superior meatuses, and is thence deflected downward and backward by the middle turbinals and the roof of the nose to the choanæ, into the epipharynx. The expiratory current is deflected from the vault of the epipharynx into the choanæ, and thence forward through the middle and inferior meatuses to the vestibules of the nose (Fig. 2).

The practical clinical application of the foregoing facts lies in the different effects of stenosis in the inferior and in the superior portions of the nasal chambers. An obstructive deformity of the lower portion of the septum may interfere somewhat with the expiratory current, as it blocks the inferior meatus while the middle and superior meatuses are free, and the expiratory current, therefore, passes through the nasal chamber upon the obstructed side, with but little or no impediment. The obstruction in the lower portion of the nasal chamber does not materially interfere with the inspiratory current, as its course is normally higher in the nasal passage. There are exceptions, however, to this rule. If, for example, the deformity of the septum extends well forward into the vestibule of the nose it will materially interfere with the inspiratory current, as it blocks the entrance to the nose. (See Chapter IV.)

The Septum.—This subject is fully discussed in connection with the deformities and malformations of the septum. (See Chapter IV.)

The Turbinated Bodies.—The turbinated bodies, three in number, are located upon the outer wall of the nasal chambers, and are known

as the inferior, middle, and superior turbinated bodies (Fig. 2), of which only the inferior and middle are of clinical importance. These are characterized by the presence of venous plexuses in the submucous tissue of the membrane, known as "swell bodies," or the erectile tissue of the nose. The erectile tissue is chiefly distributed along the inferior border of the inferior turbinal, and on the posterior ends of the inferior and middle turbinals. Its function is supposed to be that of warming the inspired air and of regulating the amount of serous secretion. Either process is of vital importance to the lower respiratory tract. The lower respiratory tract does not secrete enough moisture for physiological purposes (protective), nor is it capable of warming the inspired air sufficiently to bring it to the body temperature without injury to its mucous membrane. It is important that the heating and humidifying apparatus of the nose should be in good physiological condition. When, therefore, the vasomotor nerves which regulate the erectile tissue are disturbed in their function, the preparation of the inspired air for the lower air tract is imperfectly performed. The lower air tract is exposed to the irritating influence of the inspired air, and irritation of the lining mucosa and of the endothelial cells which line the air vessels of the lungs may result in bronchitis, while the transfusion of the gases, oxygen, and carbon dioxide may be disturbed in the air vesicles. The processes of tissue metabolism or the chemistry of nutrition are perverted.

In addition to the foregoing conditions resulting from the disturbed functions of the "swell bodies," the patient may experience either a sense of "stuffiness" of the nose or of a foreign body, or the reverse, an unduly open nose. If, for example, there is an anterior or vestibular obstruction from any cause, the negative pressure thus brought about causes an engorgement of the "swell bodies," with the resultant disagreeable symptoms already described. This condition is known as rhinitis with turgescence. If, on the contrary, the patient is anemic, the "swell bodies" may become collapsed and the nasal chambers unduly patulous. This condition is known as rhinitis with collapse of the erectile tissue. The turbinated bodies are of clinical interest, for the further reason that they divide the nasal chambers into three partial chambers or meatuses. The inferior meatus is the space between the floor of the nose and the inferior turbinal. The middle meatus is the space between the inferior and middle turbinals. The superior meatus is the space above the middle turbinal. The meatuses are of great clinical interest on account of the accessory nasal sinuses opening into them.

The Meatuses.—The inferior meatus is of clinical importance, as the nasal orifice of the tear duct opens in its anterior portion, and because it is a part of the expiratory air tract.

The Middle Meatus.—The middle meatus is of great clinical importance because the frontal, anterior ethmoidal, and the maxillary sinuses open into it. The frontal and the anterior ethmoidal cells drain into the infundibulum in 50 per cent. of the cases. The bulla ethmoidalis and the cells in the middle turbinal do not drain into the infundibulum, but open directly into the middle meatus. The bulla is often quite

large and bulges so much toward the septum that it encroaches upon the infundibulum and entirely obstructs it. It thereby interferes with the drainage of the frontal, maxillary, and the anterior ethmoidal cells. The cells opening into the middle meatus are referred to for convenience as Series I.

When pus is present in the middle meatus it is significant of empyema of one or more of the cells comprising Series I, namely, the frontal sinus, the anterior ethmoidal, and the maxillary sinuses (antrum of Highmore).

The Superior Meatus.—The superior meatus is of clinical interest because the posterior ethmoidal and the sphenoidal cells (Series II) open into it. This meatus cannot be directly inspected on account of its hidden position above the middle turbinal. It may, however, be examined with a probe. When pus flows into it from the posterior ethmoidal and sphenoidal sinuses, and the olfactory fissure is not completely closed, it may be seen lying between the septum and the middle turbinal (the olfactory fissure).

The superior meatus is of still further clinical interest because the terminal filaments of the olfactory nerve are distributed there. (See Olfactory Nerves.)

The Sinuses Residual Organs.—The nasal accessory sinuses in man are the remains of the olfactory organ, hence they have a low recuperative power after operations. I have repeatedly observed the slow and sometimes incomplete repair after operations, even after the most thorough exenteration, especially of the ethmoidal cells. I attribute this to the fact that the structures in man have ceased to perform the function they were originally designed to do. Through long ages of retrogression the tissues have lost some of their vitality and do not regenerate with the same degree of vigor as those structures which still perform their functions.

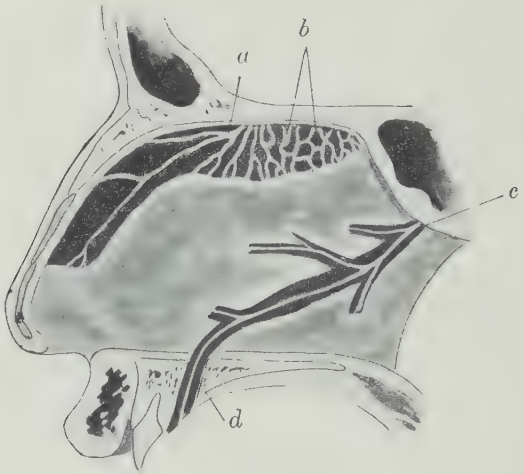
The Nerve Supply of the Nose.—The Sensory Nerves.—The sensory nerves of the nasal septum, the N. ethmoidalis anterioris and the N. nasopalatinus, send their filaments to the anterior and posterior portions of the septum, respectively. The N. ethmoidalis anterioris passes through the anterior portion of the cribriform plate (Fig. 1), thence forward and downward to the vestibule. The N. nasopalatinus extends forward and downward on the septum to the canalis incisivus, anastomoses with that of the other side, and ends in the mucous membrane of the hard palate.

The sensory nerve supply of the outer walls of the nose is derived from the N. ethmoidalis anterioris and from branches of the ganglion sphenopalatinum. The N. ethmoidalis anterioris supplies the anterior portion of the lateral walls in front of the turbinated bodies, and the turbinated bodies are supplied by branches of the sphenopalatine ganglion (Fig. 2). The hard and soft palates are also supplied from this ganglion. These anatomical facts may be utilized in injecting cocaine for anesthetic purposes (Killian) and in injecting alcohol in the treatment of hyperesthetic rhinitis (O. J. Stein).

Vasomotor branches are also supplied to the vessels of the mucous

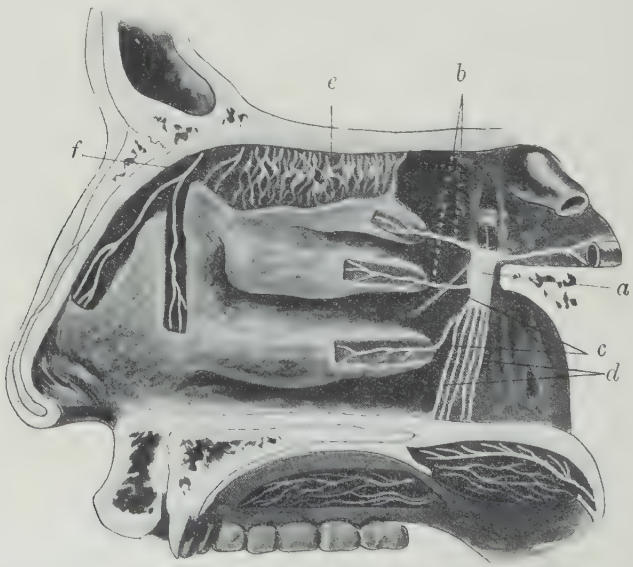
membrane and erectile tissue of the turbinated bodies from the ganglion sphenopalatinum, and are under the control of the vasomotor centres of

FIG. 1



Nerve supply of the septum nasi: *a*, N. ethmoidalis anterioris; *b*, N. olfactorii; *c*, N. nasopalatinus; *d*, canalis incisivus. (After Spalteholz.)

FIG. 2



Nerves of the lateral wall of the nose: *a*, ganglion sphenopalatinum; *b*, rami nasales posteriores superiores laterales; *c*, rami nasales posteriores inferiores laterales; *d*, Nn. palatini; *e*, Nn. olfactorii; *f*, rami nasales interni, N. ethmoidalis anteriores. (After Spalteholz.)

the medulla; there is probably a connection with the nuclei of the vagus through association fibers (Watson Williams).

The distribution of the accessory nerves over the septum and the outer walls of the nose, and especially the branches from the sphenopalatine ganglion over the turbinals, at once suggests the reason for the sensitiveness of these areas when the mucous membrane is inflamed, or is so swollen that it impinges against the septum. It also suggests the reflex phenomena, as asthma, often observed when there is inflammation or other disease of these regions. The association fibers, referred to above, connecting the sphenopalatine ganglion with the vagus establish a physiological relationship between the upper and the lower respiratory tracts, hence the asthma of nasal origin. I have repeatedly seen cases in which the asthma promptly disappeared after the removal of nasal polypi, or after an exenteration of the ethmoidal labyrinth for sinuitis. The irritation of the terminal filaments of the turbinal branches from the sphenopalatine ganglion was thus removed, and the reflex stimulus through the ganglion to the vagus and thence to the bronchial muscles ceased to be given off; hence, the bronchial spasm (asthma) was cured.

The vascular engorgement present in chronic rhinitis with turgescence is due to a paresis of the vasomotor constrictor muscles supplied by the branches of the sphenopalatine ganglion.

The Olfactory Nerve.—The olfactory nerve descends through the lamina cribrosa (cribriform plate) from the under surface of the olfactory bulb and is distributed in the mucous membrane covering the upper portion of the superior turbinal and a corresponding portion of the septum (Figs. 1, 2, and 3). Formerly it was thought that the distribution of the olfactory nerve in man covered a much more extensive area, the upper and median surfaces of the middle turbinal and a corresponding area of the septum being included in the alleged area of distribution. In many of the lower animals the nerve has a wider distribution; the sinuses communicate more freely with the nasal chambers and are utilized for the spread of the terminal olfactory nerve filaments. In man they are the remains of the organ of smell, and only communicate with the nasal cavities through small ostei or cell openings, as they are no longer needed for olfaction.

To return to the olfactory nerve. It is obvious that if the middle turbinal and the septum are in apposition, the inspired air does not reach the olfactory region, and anosmia or loss of the sense of smell results. It follows that if the obstruction to the olfactory fissure is overcome, either by the removal of the middle turbinal or by the correction of the deviation of the septum, air is admitted to the olfactory region and the sense of smell is restored, provided the nerve has not undergone degeneration.

Inasmuch as the distribution of the olfactory nerve is limited to the superior turbinal and the corresponding portion of the septum, the middle turbinal and the ethmoidal cells may be removed in their entirety without interfering with its distribution. In such operations the superior turbinal should be left intact in so far as it is compatible with a complete exenteration of the ethmoidal cells.

The Blood Supply of the Nose.—The middle meningeal artery gives off the sphenopalatine branch, which, when it reaches the posterior

portion of the lateral wall of the nose, subdivides into the lateral posterior nasal arteries. These are distributed over the middle and inferior turbinals and the middle and inferior meatuses. The superior turbinal and the anterior portion of the outer wall of the nasal chamber are supplied by the posterior ethmoidal and the anterior ethmoidal arteries respectively (Plate I, Fig. 1).

As the posterior lateral nasal arteries are of considerable size, it is to be expected that the removal of either the middle or inferior turbinated bodies may be attended by considerable hemorrhage. As a matter of fact, the removal of the middle turbinal is usually followed by more or less bleeding for twenty-four hours. There is a free anastomosis between the lateral nasal arteries and the anterior ethmoidal artery; hence, after the removal of the turbinated body bleeding may come from both sources though but one artery is injured.

The septum is supplied by the A. nasales posteriores septi, a branch of the A. sphenopalatina, through the foramen sphenopalatinum. It has three main branches: one supplies the posterior, another the inferior, and the other the middle and posterior portions of the septum.

The A. ethmoidalis anterior and the A. ethmoidalis posterior are distributed to the anterior and the superior portions of the septum (Plate I, Fig. 2). Severe hemorrhage occasionally attends or follows an operation upon the septum, especially when the operative field includes the middle branch of the A. nasales posteriores septi.

THE PHYSIOLOGY OF THE NOSE

The functions of the nose are olfactory, phonatory, respiratory, gustatory, and the ventilation of the nasal accessory sinuses. The gustatory function in man is probably of least importance, the olfactory of secondary importance, the phonatory of tertiary importance, while the respiratory, and ventilating functions are of the greatest importance.

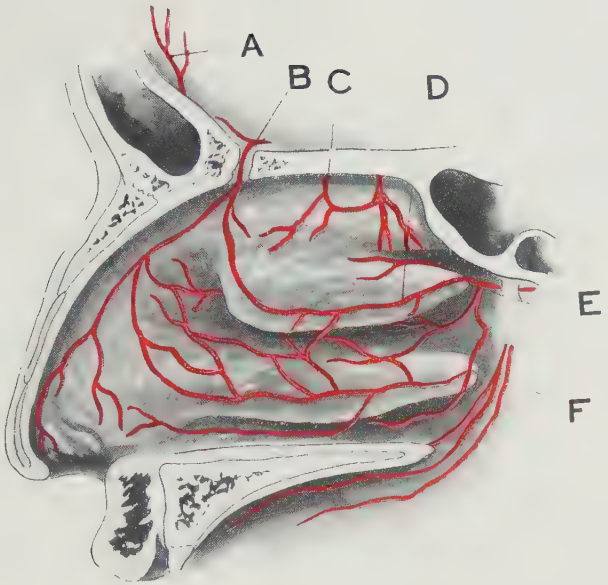
The Sense of Smell.—The olfactory nerve, or organ of smell, is located in the upper portion of the nasal chambers. The olfactory nerve (Fig. 3) is distributed over the attic of the nose as far downward as the upper margin of the middle turbinated body and on the septum over a corresponding area. A knowledge of the area of distribution of this nerve is of practical importance in the diagnosis, prognosis, and treatment of certain diseases of the nose. If there is anosmia, or loss of the sense of smell, the question arises as to whether the impairment is due to a degenerative change in the nerve itself, or to an obstruction to the entrance of the odoriferous particles or emanations to the terminal cells of the olfactory nerve.

The lesions may, however, be intracranial, in which case there may be no evidence of either an obstructive lesion or of degenerative changes in the attic of the nose.

The loss of the sense of smell, while not comparable to the loss of the nasal respiratory function, is, nevertheless, attended by considerable

PLATE I

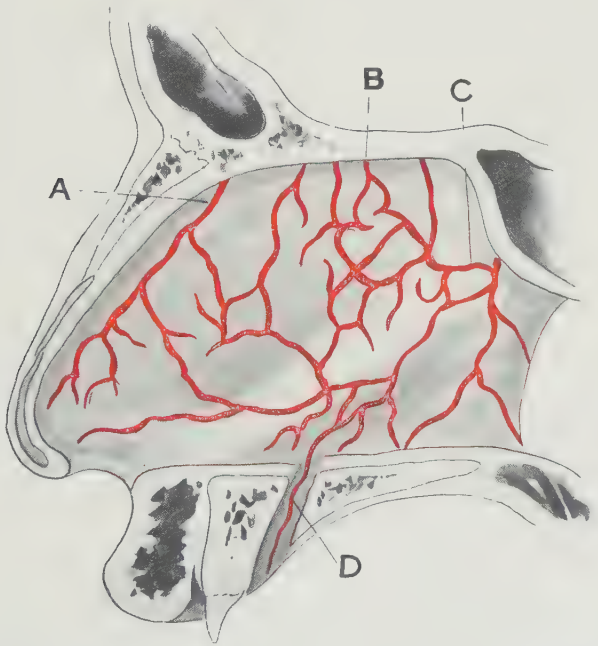
FIG. 1



The Arterial Supply of the Lateral Wall of the Nose.

A, a. meningea anterior; *B*, a. ethmoidalis anterior; *C*, a. ethmoidalis posterior; *D*, aa. nasales posteriores laterales; *E*, a. sphenopalatina; *F*, aa. palatinæ major et minores.

FIG. 2



The Arterial Supply of the Septum Nasi. (After Spalteholz.)

A, a. ethmoidalis anterior; *B*, a. ethmoidalis posterior; *C*, aa. nasales posteriores septi; *D*, anastomosis with a. palatina major.

inconvenience. The pleasure experienced by the recognition of certain odors is longed for by those affected by anosmia. More than this, they have lost one of the senses whereby they are protected from harm by certain substances, as ammonia, etc. By its aid we are warned of the near approach of decaying matter, or other foul-smelling and unsanitary substances. In the lower animals the sense of smell is of much greater utility in seeking food and in detecting the approach of hunters and animals intent upon their destruction.

Phonation.—The function of the nose in speaking and singing is so important that Jeane de Reske has said that the more he studies the voice the more he is convinced that it is a question of the nose. I have often noted that popular public speakers had well-developed nasal resonance, while speakers lacking resonance had difficulty in holding the attention of their audiences. While the initial tone is produced by the vibrations of the vocal cords, the voice is decidedly unpleasant and unmusical if it is not rich in overtones from the resonance chambers of the nose, throat, and chest. (See *The Singing Voice.*) The nasal chambers and accessory cavities are of prime importance in voice production, and any obstruction from swelling of the mucous membrane, deflection, or other lesion of the septum so materially alters the quality of the voice as to make it disagreeable and inartistic.

FIG. 3

Showing the area of distribution of the olfactory terminal nerve cells in the human nose. The triangular flap is the septum turned upward; the area of distribution is limited to the region of the superior turbinal, and a corresponding area of the septum, the middle turbinal receiving few or no olfactory cells.

Nasal Respiration. — As before stated, the respiratory function of the nose is the most important. The nasal chambers are more than mere tubes through which air is drawn into the lungs; they produce certain changes in the air which prepare it so that the normal transfusion of oxygen and carbon dioxide may take place through the walls of the air vesicles. The respiratory functions of the nose are threefold, namely: (*a*) To temper, (*b*) humidify, and (*c*) filter the inspired air.

Experiments have demonstrated that no matter what the temperature of the air may be before it is inhaled, it is raised or lowered, as the case may be, to near the body temperature. The delicate structures of the

deeper respiratory tract are thereby protected against the great variations and extremes of temperature.

It has also been shown that the air in passing through the nasal chambers receives moisture from the nasal mucous membrane. The mucosa of the lower respiratory tract and the epithelial walls of the air vesicles of the lungs are thus protected from the varying humidity of the atmosphere. In passing through the nose the air is raised (usually) in temperature, thus expanding it and increasing its capacity to absorb moisture. The "swell bodies," or erectile tissue of the nose, and the serum-secreting glands of the nasal mucosa give off moisture, which is rapidly taken up by the expanded air and carried to the lower respiratory tract, where the serum-secreting organs are much less developed. It has been estimated that approximately one pint of serum is thus transferred from the nasal cavities to the lower respiratory tract in twenty-four hours.

The part of the nasal structures which secrete most of the serum is generally supposed to be the "swell bodies," or erectile tissue, located chiefly along the free border of the inferior turbinated bodies, and on the posterior ends of the middle and inferior turbinated bodies. The latter portions sometimes become enlarged and form the so-called mulberry hypertrophies. It is probable that the mucous glands also secrete some of the serum. The "swell bodies" are under the control of the vasomotor nervous system, which, under normal conditions, regulates the supply of moisture to meet the demands. If the air is dry the "swell bodies" enlarge and become just active enough to fully saturate the expanded air in the nose; whereas if the atmosphere is humid they are less active. When an obstructive lesion, or catarrhal inflammation, is present the "swell bodies" and glands do not respond normally to the atmospheric conditions, hence the air is not properly humidified in its passage through the nose. The treatment of these conditions should be, therefore, so directed as to restore the "swell bodies" and glands to their normal activity. In order to do this, it may be necessary to give stability to the vasomotor nervous system by judicious bathing, outdoor exercise, etc. In addition, local massage of the mucous membrane and other treatment may be necessary. Surgical interference should always be accomplished with respect to the location of the "swell bodies," care being exercised to avoid their destruction, except in those cases in which they have undergone considerable hypertrophy. The surgery of the middle turbinated body may be practised with much greater freedom, because it does not have so much to do with the respiratory functions of the nose. The inferior turbinated body, however, should be treated surgically only when its secretory function is largely destroyed, or when it is so enlarged by hypertrophic or hyperplastic changes that it obstructs nasal respiration.

That the nose is a filter is evident upon inspection of the secretions and the vibrissæ of the vestibule, as they are loaded with dirt. The vibrissæ guarding the atrium of the nostrils act as a coarse filter, the larger particles lodging on them, the smaller ones entering the nasal cavities, where they are caught upon the irregular surface of the moist

mucous membrane. The lower air tract is thus protected from the irritation which would otherwise result. F. C. Cobb, under the direction of Frederick Coolidge, of Harvard University, has shown by a long series of experiments that the secretions posterior to the vestibules of the nose are sterile, thus demonstrating the great physiological importance of the vibrissæ and the sterilizing quality of the nasal secretions.

The Gustatory Function of the Nose.—The real gustatory or taste sense (sweet, sour acid, bitter, and salt) is supplied by the distribution of the glossopharyngeal and the fifth nerves to the fauces and the base of the tongue, whereas the delicate flavors which give so much pleasure to the consumption of foods and drinks are appreciated through the olfactory nerve. If the nostrils are closed and the eyes covered it is almost impossible to distinguish between coffee and water of the same temperature, as the aromatic flavor cannot be appreciated by the nose when closed.

Ventilation of the Sinuses.—I have assumed a fifth function of the nose —the ventilation of the accessory sinuses—which has not heretofore been described under the physiology of the nose. It is obvious to anyone who has had an abundant opportunity of observing inflammation of the sinuses, that ventilation is a prime requisite for the maintenance of the mucous membrane of these cavities in a healthy condition. Any interference with the ventilation of these cavities lowers the resistance of the mucous membrane and the diminished amount of oxygen allows the secretion to undergo rapid decomposition.

Summary: The functions of the nose are fivefold, namely:

1. Olfactory, located in the attic of the nose.
2. Phonatory, enriching the voice by overtones.
3. Respiratory.

(*a*) The air is warmed or tempered to or nearly to the body temperature in passing through the nose, thereby preventing shock and irritation to the mucosa and air vesicles of the lower respiratory tract.

(*b*) The air is expanded by the warmth of the nasal chambers, and its capacity to absorb the moisture thrown off by the "swell bodies" and mucous glands is increased. The mucosa and air vesicles are thus moistened, or, at least, their moisture is not absorbed (the air being already saturated in its passage through the nose), and irritation is prevented. The nose keeps the inspired air in a state of saturation.

(*c*) The air is filtered in its passage through the nose by the vibrissæ and the moist mucous membrane. The irritation to the mucosa and air vesicles which would otherwise occur is thus prevented.

4. The gustatory (olfactory) sense complements the sense of taste.
5. The ventilation of the accessory sinuses maintains the normal resistance of the mucous membrane and prevents the rapid decomposition of the secretions.

CHAPTER II

THE NOSE, THROAT, AND EAR IN RELATION TO GENERAL MEDICINE

The writings of William Meyer, of Copenhagen, William Daly, of Pittsburg, and E. P. Friedreich, of Leipsic, have given a breadth to rhinology, laryngology, and otology which they did not have in the days when practice along these lines was regarded as a "specialty." With this broader view they are now regarded as the pursuit of the practice of general medicine and surgery, with special reference to the diagnosis and treatment of diseases in general, and those of the nose, throat, and ear in particular.

A proper comprehension of the relation of the nose, throat, and ear to general medicine and surgery will be facilitated by a brief analysis of the interdependence and coördination of the various organs and parts of the body.

ELEMENTARY FACTS

(*a*) **The Breathway.**—The upper respiratory tract is the channel in which the air is prepared for the interchange of gases which takes place in the air vesicles of the lungs. The nose is especially concerned in the process of humidifying, warming, and filtering the inspired air, and it is obvious that any disease or obstruction that interferes with these physiological processes will affect the transfusion of gases through the capillaries of the walls of the air vesicles. The absorption of oxygen by, and the elimination of carbon dioxide from, the blood will not occur in normal ratio. The blood will be deficient in oxygen and surcharged with carbon dioxide. As oxygen is essential to the processes of assimilation and nutrition, its lessened quantity in the blood gives rise to certain disturbed conditions of the digestive, the assimilative, and the nutritive functions. The presence of an excess of carbon dioxide also adds to these disturbances. It is well known that the excessive accumulation of carbon dioxide in the blood acts as a poison to the leukocytes, thus interfering with their functional activity. A normal amount of carbon dioxide in the blood favors the assimilative, nutritive, and leukocytic processes, and it is only after a greatly increased amount of it is present that there are marked disturbances. It not only interferes with the activity of the leukocytes, but also with other cellular structures of the body as well. The combined effect, therefore, of an increased amount of carbon dioxide and a diminished quantity of oxygen in the blood is to produce general anemia, indigestion, malassimilation, malnutrition, and infectious processes.

The xanthin group of toxins, including indican, are thrown into the circulation and give rise to certain nervous phenomena, as restlessness, peevishness, headache, mental depression, aprosexia, and a general feeling of malaise.

The digestive disturbances are still further increased by the ingestion of the infected secretions from the epipharynx and the tonsils. Putrefactive as well as pathogenic bacteria are swallowed with the secretions from the nose and throat, and give rise to what is commonly known as chronic dyspepsia, or indigestion. It is probable that the putrefactive germs are more potent in this connection than the streptococci and the staphylococci. The conditions of the nose and throat which most commonly give rise to this kind of discharge are nasal stenosis, atrophic rhinitis, chronic rhinitis, sinuitis, epipharyngeal catarrh, and chronic follicular tonsillitis.

There are certain conditions of the stomach and of the intestinal tract which affect the mucous membrane of the upper respiratory tract. If, for example, there is chronic indigestion, there is also malassimilation and faulty metabolism. The imperfect products of indigestion are incompletely oxidized and are thrown into the circulation, where they irritate the mucous membrane of the nose, as well as the vasomotor nerves, thus causing local congestion and overnutrition. The secretions of the glands of the mucous membrane of the upper respiratory tract are also thereby modified, thus predisposing to, or at least intensifying, the catarrhal disease present. In the same way hyperacidity and subacidity of the stomach may indirectly irritate the mucosa of the nose and throat. One of the most potent influences exerted by the products of indigestion is through the reflex nervous system, pharyngitis, hypersensitiveness, sneezing, etc., being the direct expression of this condition.

In atony of the stomach there is a putrefactive formation of gases, which act reflexly and through the circulatory system on the mucous membrane of the upper respiratory tract and cause phenomena quite similar to those just mentioned. Another condition which is quite similar in many respects to the foregoing is that which occurs in gout or lithemia. In connection with this disease the larynx and the pharynx are particularly affected. In the pharynx there may be itching behind the pillars of the fauces, associated with a similar irritation in the external meatus of the ear. Some observers regard these signs as characteristic of gout.

When such symptoms appear, the administration of calomel and the bicarbonate of soda, followed in twelve hours by a saline purge, will give marked relief. After this, teaspoonful doses of the phosphate of soda should be given two or three times daily for a few weeks.

Vomiting and eructation of gases from the stomach exert an irritating effect upon the mucous membrane of the pharynx, the epipharynx, and the nose. The irritation is due to biochemical as well as mechanical causes. Catarrhal inflammation in the epipharynx is thus perpetuated, and may finally extend to the Eustachian tube and the middle ear, and cause tinnitus and deafness.

(*b*) **Intimate Relations between Organs.**—All the organs of the body are more or less intimately connected by the vascular, the lymphatic, and nervous systems, hence disturbances in one more or less affect the others. The bloodvessels and the lymph channels carry toxic and infective material to all the organs of the body, including the nose, throat, and ear, and thus influence the functional and the pathological processes in these organs. While the data considered under this subject somewhat overlap those considered under (*a*), it is well, nevertheless, to emphasize certain features more prominently in this connection.

Anemia is a condition of the blood due to various causes, and often gives rise to collapse of the erectile tissue of the nose. This is usually spoken of as "rhinitis with collapse of the turbinated bodies." (See Rhinitis with Collapse of the "Swell Bodies.")

On the other hand, another condition of the nasal mucous membrane which may *cause anemia* instead of being a result of it, as related in the preceding paragraph, is atrophic rhinitis. It is characterized by anemia which is probably due to the absorption of toxic material from the nose, and to the loss of the respiratory functions of the nose.

If the lymphatic vessels are charged with infective material, which is finally transferred to the bloodvessels and tissues of the entire body, a state of general toxemia is induced, the nose, throat, and ear participating in the disturbed processes. On the other hand, one of the commonest clinical pictures is that wherein the lymphatic glands are enlarged by suppurative disease of the ear, nose, and throat. This subject is discussed more fully in the chapter on the Clinical Anatomy of the Tonsils. I wish, however, to emphasize the influence of suppurative diseases of the ear upon the lymphatic glands of the neck. As the ear is more intimately connected with the lymphatic glands of the posterior triangle of the neck, it is to the glands in this region that we should look for enlargement in inflammatory disease of this organ.

The close approximation of the mucous membrane of the nose and ear to the contents of the cranial cavity may also give rise to serious consequences by the conveyance of infective material thereto. Brain abscess, meningitis, septic thrombophlebitis, etc., may be thus caused, although the usual channel of invasion is through a necrotic area in the floor of the cranial cavity.

The nervous system, when disturbed in its function, necessarily influences the upper respiratory tract, as well as other parts of the body. We may thus have vasomotor rhinitis and asthma, as well as certain functional disturbances of the ear and the larynx as a result of a disturbance of the general nervous system.

Hysteria probably comes under this heading, and while it is not demonstrable histologically, it may have a histological basis. Hysteria of the nose, throat, and ear, as in other parts of the body, is characterized by a disturbance of those functions which are more particularly under the control of the mind, the involuntary functions not being affected. In the larynx, for instance, the normal respiratory movements are not disturbed, as they are involuntary; whereas the movements of the larynx

which are concerned in the production of speech, being under the control of the mind, are voluntary, and are affected.

Hay fever, laryngeal cough, sneezing, bronchial asthma, anesthesia and hyperesthesia of the mucous membranes of the ear, nose, and throat are *reflex* phenomena, which may result from the irritation of the nervous system by the toxic material in the circulation.

Another very important disease generally regarded as due to infection of the blood is *rheumatic fever*, or acute articular rheumatism. The gateway of infection is often through the tonsils, or some portion of Waldeyer's ring. The throat symptoms of this disease are a reddened pharynx, with a defined or circumscribed inflammation of the larynx, redness and swelling in the arytenoid region, and sometimes fixation of the arytenoid cartilages. Pain and difficulty in phonation and deglutition may also be present in rheumatic fever. The physician should not only look upon the tonsils as the portals of infection, but he should look to the pharynx and larynx for some symptoms of the rheumatism. Acute rheumatic fever also gives rise to certain symptoms which are not commonly recognized. For example, it may cause nosebleed in children, and in some cases is undoubtedly the cause of chorea.

Malaria is another disease affecting the blood which gives rise to certain symptoms in the ear, nose, and throat. Mastoid pain, and, indeed, mastoid suppuration, has been observed in which the malarial element was prominent. In view of some recent observations, it may be questioned, however, whether these cases were distinctly malarial in their origin. We now know that there are certain septic conditions which give rise to symptoms so nearly like those due to the plasmodium of malaria that it may be questioned whether these cases were truly malarial, or whether they were septic. It is known, however, that the malarial poison may cause nasal hydrorrhea and vasomotor rhinitis.

The bloodvessels and lymphvessels are channels of communication between the *throat* and the *appendix*. In certain cases of appendicitis it has been shown that streptococcus infection was present both in the throat and in the appendix. Another possible source of communication in these cases is through the alimentary tract.

(*c*) **The Digestive Tract.**—The digestive tract, which prepares the food for tissue building, is affected by the putrefactive and the pathogenic microörganisms from the nose, throat, and ear. The primary treatment should be addressed to the relief of the diseased condition of the upper respiratory tract, rather than to the stomach and the intestines. The presence of dyspepsia, or other functional disturbances of the stomach and the intestines, should lead to the examination of the nose and throat, with special reference to the discharges from them, which may be swallowed by the patient. On the other hand, if there is an irritable state of the nasal, pharyngeal, and laryngeal mucous membranes, which is not explained by any local source of irritation, careful attention should be given to the condition of the stomach and the intestines, or to the organs of digestion and assimilation in general, with a view to determining whether they are properly performing their functions. If they

are not, the nutritive properties of the food are thrown into the circulation imperfectly or insufficiently prepared for their purposes. The irritation thus carried to the nasal mucous membrane and to the nerves supplying it may be the chief cause of the local disturbances. It is obvious that under these circumstances the treatment should be addressed to the correction of the disorders of the digestive tract, rather than to the nose, throat, and ear.

(*d*) **Excretory Organs.**—The function of the excretory organs is to throw off the refuse material formed during the processes of nutrition. The refuse consists not only of the material not needed for the nutrition of the body, but also of the toxic material and the half-way products of oxygenation already referred to. Hence, any impairment of the functions of these organs results in an excess of toxic material in the blood and the lymphatic vessels, thereby causing congestion, irritation, hypertrophy, hyperplasia, or altered secretions in the upper respiratory tract. This feature of the subject is intimately associated with those in the preceding paragraphs.

The *skin* and the *kidneys* are the chief excretory organs of the body. We will dismiss the skin with a brief reference to the fact that eczema lupus, etc., affecting other portions of the body may also involve the external nose and external ear. Or, the pathogenic processes may begin with the skin of the nose or the external ear, and extend to other parts of the body. We will also incidentally state that erysipelas of the nose may involve the nasal mucous membrane, and that erysipelas of the skin over the mastoid process may extend to the middle ear and the mastoid cells, or even to the cranial cavity through the lymphatics and the bloodvessels of this region.

The *kidneys*, however, are the excretory organs which chiefly interest us in this connection. They may be diseased by prolonged infection in remote parts of the body, as in the nasal sinuses or the alveolar processes —pyorrhea alveolaris. Bright's disease may manifest its earliest symptoms in the mucous membrane of the throat. The throat symptom complained of is dryness. This same symptom may also be present in diabetes. Diabetes is mentioned here not because it is a disease of the kidneys, but because its chief symptom is to be found in the examination of the excretions from the kidneys.

When a patient complains of persistent dryness of the pharynx his urine should be tested for albumin, casts, and sugar. In some cases albumin will not be found at first, but after a few years its presence may be detected.

Edema of the glottis, causing laryngeal stenosis, is often due to uremia developing as a result of Bright's disease. In the milder forms of uremia bronchial asthma and hemorrhage of the upper air passages are sometimes found to be the chief expression of the disease. In the more pronounced uremic conditions there may be aphasia from edema of the brain.

(*e*) **Proximity of Organs.**—The close proximity of the organs of the head favors a correlated pathological activity. The eye is near the

nose and has immediate communication with it through the tear duct, as well as through the lymphatics, the bloodvessels, and the nervous system; hence disease in one often gives rise to certain symptoms in the other. Experiments with certain colored solutions dropped into the eye have shown the coloring matter within a very short time in the nasal mucous membrane. The instillation of bacteria yields the same results. Clinically, it is not uncommon to observe an inflammatory condition in the eye simultaneously with or following a similar process in the nose. I have often had cases referred to me by ophthalmologists who were unable to prescribe satisfactory glasses until after I had corrected the nasal condition, usually involving the middle turbinated body or the ethmoid cells. The proximity of the nose to the ear, as well as the physiological communication between them *via* the Eustachian tube, gives rise to a very intimate relation between these organs.

It is well known that inflammation of the epipharynx sometimes extends through the Eustachian tube, by continuity of tissue, to the middle ear. This condition may develop until there is suppurative otitis media, mastoiditis, and even intracranial complications. Adenoids are also a fruitful source of mischief to the ear and the mastoid process. They may mechanically obstruct the Eustachian tube, or the epipharyngitis which almost invariably accompanies them may cause the ear disease. The removal of adenoids in children is often followed by immediate relief of deafness and of suppurative inflammation of the middle ear.

While the stomach is not so closely related to the ear as the epipharynx, nevertheless it has a close pathological and anatomical connection through the esophagus. In vomiting, foreign matter may be forced into the Eustachian tube and the middle ear, and may cause otitis media and its attending complications. From this same organ eructations of gas may also cause irritation in the epipharynx and the Eustachian tubes.

The nasal discharges, especially when there is *empyema* of the *accessory sinuses* of the nose, usually pass backward into the epipharynx and cause irritation and inflammation in this region. They also pass to the larynx and cause more or less trouble there. Stenosis of the nose interferes with the functions of that organ, and thus allows the air to pass into the epipharynx, the larynx, and the bronchial tubes insufficiently warmed, insufficiently moistened, and imperfectly filtered. Irritation of the mucosa of the lower respiratory tract is thus caused and gives rise to catarrhal inflammation.

The ear is separated from the cranial cavity by a partition of bone which in places is not more than one-sixteenth to one-eighth of an inch in thickness. Chronic suppuration within the middle ear and the mastoid cavity often results in necrosis of this thin plate of bone, thus opening a channel of communication between the middle ear and the cranial cavity. The sequels or complications of mastoiditis, such as meningitis, brain abscess, septic thrombophlebitis, etc., may thus result from ear disease.

The nose is but slightly separated from the cranial cavity, and through

the ophthalmic veins may cause thrombophlebitis of the cavernous sinus, which is usually fatal.

(*f*) **Infections.**—Systemic infections from the upper respiratory tract have already been more or less considered in this chapter as well as in the one on the Tonsils as Portals of Infection; hence the subject will not be elaborated here.

(*g*) **The Central Nervous System.**—It is obvious, inasmuch as the central nervous system supplies the innervation of the nose, throat, and ear, that in disease of the central nervous system the parts which it supplies must be affected. In other words, in certain diseases of the central nervous system some of its characteristic symptoms may be found in the upper respiratory tract.

In tabes dorsalis there may be certain motor laryngeal disturbances, which may be either bilateral or unilateral. There may be ataxic movements of the vocal cords. Laryngeal crises, as spasmodic cough, may be present.

Ear symptoms in tabes are rare. The cochlear and vestibular nerve endings may, however, be congested. In this event there will be diminished or entire absence of bone conduction and hearing for the higher tones. Dizziness, nausea, and nystagmus may also be present in exceptional cases.

In multiple sclerosis a tremulous voice, which is easily fatigued, and is deep and hoarse in character, may be present. Muscular palsy of the laryngeal muscles is rare. The ear symptoms in this disease are tinnitus, and loss of hearing by bone conduction through the sclerotic degeneration of the nuclei.

The symptoms found in paralysis agitans are about the same as those found in multiple sclerosis.

(*h*) **The Lymphatic System.**—There are certain constitutional symptoms due to infections through the lymphatic system which should be especially singled out, although they have already been referred to in Section (*a*) of this chapter.

We now recognize that a fever, characteristic of childhood, which has heretofore been regarded as one of the ill-defined malarial infections, is due to an infection through the adenoid growths in the epipharynx. The fever usually runs an irregular course of about ten days, and is characterized by an afternoon temperature of 100° to 104°, with restlessness, peevishness, sharp pains through the ears at night, anemia, general debility, loss of appetite, coated tongue with indentations from the teeth, constipation, and cervical adenitis. Mouth breathing is not essential as a factor in causing the infection. A small amount of lymphatic tissue in the epipharynx is a sufficient portal for the entrance of the bacteria. The presence of this type of fever is almost always an indication for the removal of the adenoids. If the child is known to be tuberculous, some consideration may be given to the matter before removing them, for if the removal is imperfectly done, it may give rise to a recrudescence of the tuberculous infection, which may extend to the lungs and lead to a fatal issue.

Another disease which may express itself through certain pathological changes in the ear, nose, and throat is syphilis. The nose may be the primary seat of the lesion, the infection taking place in the removal of crusts from the septum with the finger. The tonsils are occasionally the seat of the primary lesion or chancre through the use of infected instruments in the throat. The author has seen cases in which both tonsils were the seat of chancre as a result of the instruments used in lancing a peritonsillar abscess.

In one case there was the characteristic initial lesion in the left tonsil, with the cervical bubo on the same side, which was followed a few days later by the characteristic skin eruption. The source of the infection in this case was the dirty instruments used in lancing a peritonsillar abscess. I first saw the case six weeks after the tonsils were lanced. The patient had been complaining of sore throat for two or three weeks. The tonsils and the bubo were still very much in evidence and the eruption on the skin had just begun to show. In the course of another week the *corona veneris* developed. The copper-colored eruption on the face showed more plainly at a distance of twelve or fifteen feet than it did when viewed near by.

Secondary syphilis may manifest itself by mucous patches in the buccal cavity, by hyperemia of the larynx, hoarseness, and syphilitic coryza, with scanty, thick secretion from the nose. Syphilitic coryza is not always recognized by the family physician, it being regarded as a simple obstinate cold in the head. The scanty thick discharge, with stenosis of the nose, should, however, excite suspicion of the true nature of the disease.

I once saw a case in which there was a marked arrest of development of the bones of the face because when in childhood the syphilitic coryza developed the family physician regarded it as an ordinary cold. He treated the patient for the same without success, and was finally surprised to find the nasal bones and the septum giving way. The soft palate and the pharynx later became involved and rapidly melted away under the blighting influence of the Spirochæta pallida. The patient is now thirty-six years old, and has the most pronounced "frog" face I have ever seen. Adhesive bands bind the soft palate to the pharyngeal wall, making it difficult for him to speak distinctly, though he is now successfully engaged in business.

The *tertiary manifestations* of syphilis are syphilitic pharyngitis and laryngitis, with a raucous voice. Syphilitic lesions of the tonsils, presenting a dirty grayish necrotic surface resembling diphtheria, are occasionally observed. Syphilitic gummata are not excessively destructive in character. Syphilitic papillomata of the tonsils and the soft palate are elsewhere described.

Recent investigations have discredited the oft-repeated statement that the skin and the mucous membranes of the animal organism are insurmountable barriers to microörganisms so long as the epithelial coat is intact. Bono and Frisco report that the researches undertaken at the Institute of Hygiene at Palermo have established the fact that germs

deposited on the intact skin or mucosa are found soon afterward in the lymphatic ganglia of the respective regions. If the germs are so numerous or so virulent as to overcome the resistance offered by the lymphatic ganglia, general infection follows. If not, there is merely a local reaction on the part of the ganglia, which become tumefied and undergo various modifications in their structure proportional to the number of germs which reach them.

Diseases of the Eye Due to Nasal Lesions.—To establish the relationship between the nasal mucous membrane and the eye, microorganisms were placed on the nasal mucous membrane both with and without obliteration of the nasolacrymal canal. The result of the experiments showed the penetration of the germs into the vitreous and the aqueous humors of the eye on the same side. (Bono and Frisco.)

"None of the animals exhibited any signs of general infection. One or two colonies, at most, could be derived from the blood in the heart, the liver, the spleen, and the lymphatic ganglia of the neck, and occasionally from the anterior auricular, the submaxillary, the deep jugular, and the carotid lymphatic ganglia. This fact, considered in connection with the presence of large numbers of germs in the aqueous and the vitreous humor, and the absence of general infection, warrants the conclusion that the bacteria penetrated directly into the eye from the nasal and the conjunctival mucous membranes, and that they also arrived secondarily in the eye through the blood, but reduced in numbers and virulence. Part of the germs were retained by the ganglia connected with the anterior lymphatic vessels of the eyeball and its appendages. In further experiments with instillations of India ink it was possible to trace the exact route followed by the particles from the conjunctival lymphatics along Schlemm's canal into the anterior chamber and thence into the vitreous. From the lymphatics of the nasal mucosa the particles passed into the ethmoid cells and the lamina papyracea, thence into Ténon's capsule, and on into the eyeball. The practical results of these researches are particularly important in the pathology of the eye."

F. Mendel, after observing many cases, comes to the conclusion that the nasal infection and inflammation is transferred to the eye by the direct connection or continuance of the epithelium of the nasal mucous membrane to the conjunctiva, as well as by the intimate vascular association.

The ophthalmic artery gives off the anterior ethmoidal, which supplies the nose and the lacrymal canal. The venous supply of the nasal mucous membrane, by means of the lacrymal plexus, is in direct communication with the ophthalmic vein.

Heber Nelson Hoople, in a paper read before the American Laryngological, Rhinological, and Otological Association, in 1901, advances the theory that faulty pressure within the nose can cause asthenopia of both the ciliary and external ocular muscles. That is, mechanical pressure in a limited area of the nose, called by Mackenzie the reflex area, can cause muscular asthenopia. By muscular asthenopia he means the impairment of the efficiency of the ocular muscles in the performance of their ordinary functions.

The pressure to which Hoople refers is confined chiefly to the middle turbinal, especially in great enlargement of the middle turbinated body.

A concomitant symptom usually occurring in conjunction with the asthenopia is a browache or headache referred to the frontal region or to the occiput in rare instances.

He cites a number of cases in his own practice and in that of others in which the asthenopia disappeared as soon as the nasal pressure was overcome. The asthenopic cases referred to belong to the so-called normal type rather than to the excessive type.

He concludes that a moderate amount of pressure or mechanical irritation of the middle turbinated body against the adjacent septum will temporarily impair the function of the ciliary muscle; to a lesser or more variable degree it will also impair that of the external ocular muscles. If mechanical irritation (from congestion or swelling of the soft tissues) can impair the functions of these muscles, how much more would a continuous pressure from a septal spur or other deviation of the septum digging into the middle turbinal keep up this impairment.

The reason for the association of headache with asthenopia is that they have a common cause—pressure upon the sensorimotor branches of the trigeminus. So far as the sensory part is affected, a radiated or a reflex headache is produced; so far as the sympathetic fibers are affected a vasomotor reflex is produced. This is equally true where there are inflammatory conditions, as ethmoiditis. It matters little whether the pressure is from within the ethmoid cells and turbinal or from without these structures. The important point is that the same branches of these nerves are pressed upon, and, therefore, the same kind of disturbances should be expected to follow.

The asthenopic disturbance is probably due to irritation of the sympathetic fibers in this particular class of cases. That it is such in all cases is also probable. It could be inferred from other facts, *e. g.*, when treatment addressed to the uterus, the bladder, or the stomach has given relief to the asthenopic symptoms.

In the light of the foregoing views expressed by Hoople, asthenopia or disturbed function of the ciliary and external ocular muscles is usually due to intranasal pressure and irritation in the middle turbinal and ethmoidal regions, rather than to toxemia from infection of the sinuses. The speedy relief of the asthenopia following the divulsion or the removal of the offending middle turbinal seems to prove this view rather than the view referring the disturbance to toxemia.

In the cases referred to by Hoople the headaches were of the ocular rather than the sinus type, as they were induced, or aggravated, by the use of the eyes, and were relieved upon retiring for the night. Sinus headache is not always aggravated by using the eyes, and is often most pronounced upon awakening.

CHAPTER III

THE OFFICE EQUIPMENT

In the equipment of an office the chief point to be considered is facility in treating patients. The treatment and consultation rooms should be equipped for work rather than for entertainment. Everything for facility and thoroughness; nothing for show. "Bluff" is a confession of unfitness. Thorough knowledge and frankness of statement will inspire confidence and give an impression of mastery as no amount of "bluffing" will do.

The essential furnishings of the consultation room and treatment room should consist of the following:

(*a*) Treatment and operating chair. (*b*) A revolving stool for the surgeon. (*c*) A treatment table or cabinet. (*d*) A fountain cuspidor. (*e*) A linen cupboard. (*f*) A writing desk. (*g*) A sterilizer. (*h*) A revolving desk chair. (*i*) Two small chairs. (*j*) An adjustable bracket for the examination lamp. (*k*) A selection of instruments and apparatus for examinations, treatments, and operations.

The Treatment and Operating Chair.—This should be a revolving chair, as suggested by Dr. Robert Levy, as it is desirable to turn the patient from side to side in treating his ears, and for other reasons as well. The bottom should be on a central screw pin, so that it can be adjusted to different heights for children and adults. The back should be so constructed that it can be lowered to a horizontal position in case of faintness and when it is desirable to operate with the patient in a prone position. An adjustable head-rest should be attached to the back of the chair (Figs. 4 and 5). An ordinary chair may, of course, be used, but in the case of faintness, etc., the work is greatly facilitated and the comfort of the patient assured if the chair is of the adjustable type described.

The Treatment Table or Cabinet.—If an assistant is employed it is preferable to have the instruments in a separate cabinet in an adjoining sterilizing room or corner. The treatment cabinet may then consist of a metal enamelled frame with a plate-glass top, or it may be a double-decked table, with top and shelves about one foot apart. These tops afford ample room for the distribution of bottles containing remedies for topical applications and for the instruments of examination and operation.

The treatment table, or cabinet (Fig. 6), is an important piece of furniture. Its selection should depend largely upon whether the surgeon has an assistant to wait upon him. If he has, the cabinet need not be constructed to contain all instruments, as the assistant will bring

such as are necessary for each case. If he does not have an assistant, it is convenient to have the instruments in the cabinet within reach.

The Hot-water Basin.—A most excellent addition to the table is a basin, set in the centre of the upper glass top, with running hot water for the purpose of rinsing instruments during the course of treatments. If preferred, the hot-water basin may be attached to a special wall bracket (Fig. 7), as it is only intended as a convenience. It is also useful in cleansing and warming the laryngeal mirror during throat examinations.

FIG. 4

FIG. 5

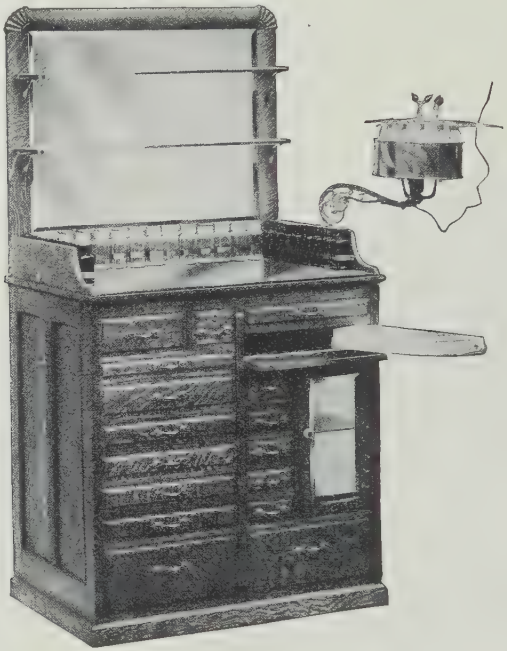
Operating chairs.

No matter how sterile the tongue depressor may be when first used, its introduction into the mouth the second or third time without cleansing is, to say the least, disgusting to the patient.

A basin of running hot water is, therefore, an invaluable and, I might add, an indispensable adjunct to the office equipment. It is not, however, indispensable in so far as the safety of the patient is concerned, as only his own secretions come in contact with the instrument used. If the fundamental principles of common cleanliness are to be recognized it is a

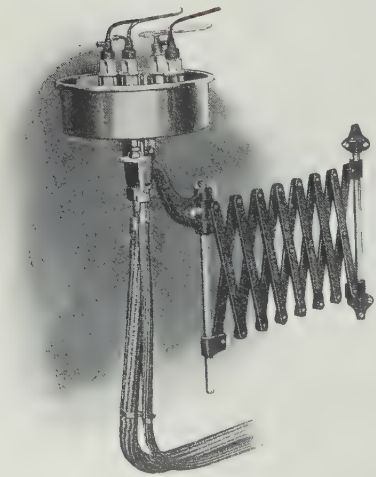
valuable and necessary office fixture. It is not a question of whether it pays, but rather one of common decency, and that always pays.

FIG. 6



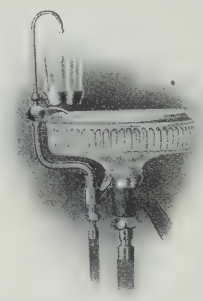
Pynchon's medicine and instrument cabinet.

FIG. 7



Clark's hot-water basin.

FIG. 8



Clark's fountain cuspidor.

A bowl of antiseptic solution is not a substitute for running hot water unless the bowl is refilled for each rinsing. The solution would otherwise

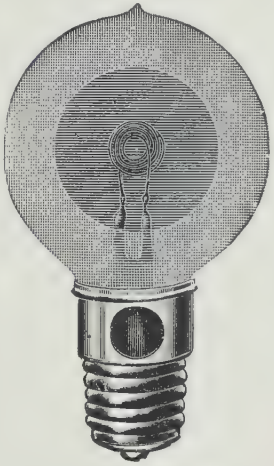
soon become thick with secretions and detritus, and the introduction of an instrument into it for rinsing purposes would be even more disgusting than no rinsing at all.

The Examination Lamp.—The examination lamp may be a kerosene, gas, or an electric lamp; the latter is preferable, because it gives off less heat and requires less attention. The lamp may or may not have a hood with a focussing lens, as the surgeon may elect. Personally, I prefer an electric lamp of 50 candle-power (Fig. 9). This should have a ground-glass surface, except a circular area on one side, where the glass should be clear.

It affords plenty of light, is simple, throws out little heat, and is inexpensive.

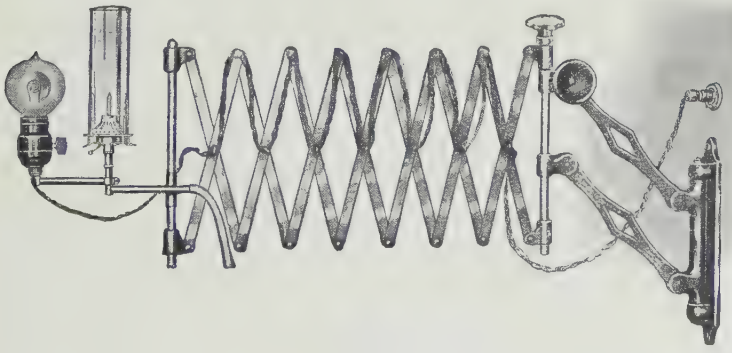
A wall bracket to support the lamp is an important item, inasmuch as it is constantly used. It should, therefore, be well constructed and accommodate itself to the varying conditions under which it is used. That is, it should be so constructed that the lamp can be raised and lowered and turned from side to side with the least trouble to the operator. It should be so well made that it will never get out of order, a state or condition into which many wall-lamp brackets are likely to fall. That shown in Fig. 10

FIG. 9



A 50 candle-power electric lamp with a rotating socket.

FIG. 10



Wall-lamp bracket.

has proved quite satisfactory in nearly every respect. A Kierstein head lamp (Fig. 11) is preferred by some operators.

Compressed-air Apparatus.—The compressed-air apparatus may be one of three types: (*a*) A hand bulb; (*b*) a tank pumped by hand or by some automatic device, as a water pump; or (*c*) a system of compressed

air supplied throughout the building by means of pipes from a central compressed-air tank. The latter is preferable when it can be obtained, as it requires no attention whatever. A compressed-air tank in the office automaticaly supplied by means of a hydraulic pump is the next most preferable arrangement. A hand pump is inconvenient and necessitates considerable labor. The hand bulb is suitable when eight pounds or less of pressure are required.

An Accessory Regulating Air Tank.—An accessory regulating air tank is a very convenient and valuable addition to the compressed-air system, as it enables the surgeon to use the amount of pressure required for various purposes. The nasal mucous membrane, for example, will not tolerate a higher pressure than ten pounds with the De Vilbiss spray tube, whereas the pharynx will tolerate from twenty to forty pounds' pressure. A nebulizer requires a higher pressure than the spray tube, and in inflation of the Eustachian tube and middle ear the pressure

FIG. 11

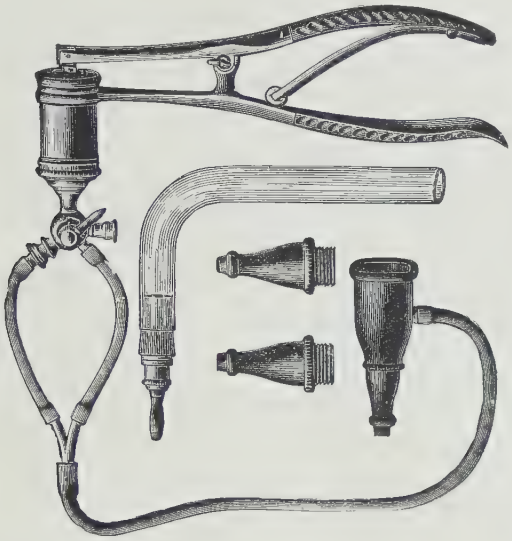
Kierstein lamp and head bracket.

required varies from eight to twenty pounds, according to the degree of obstruction present. Hence, a regulating air tank is a convenient if not a necessary apparatus. The tank should be connected with the main reservoir and the compressed air turned on until the gauge indicates the required pressure, say ten pounds. If at another time in the treatment but two pounds' pressure is needed the escape valve may be opened until the gauge indicates two pounds. There are many other ways in which such a regulating air tank may be used to advantage. The gauge regulators on the market are not nearly so satisfactory as the Pynchon and Hubbard regulating tanks, and are not recommended.

Massage Apparatus.—Ear Drum.—Pneumomassage, or the massage of the ear drum by the alternate rarefaction and condensation of the air in the external auditory meatus, is accomplished by means of a hand pump, as first devised by Delstanche, of Brussels (Fig. 12), or it may be operated by an electric motor, as first devised by Chevalier Jackson, of

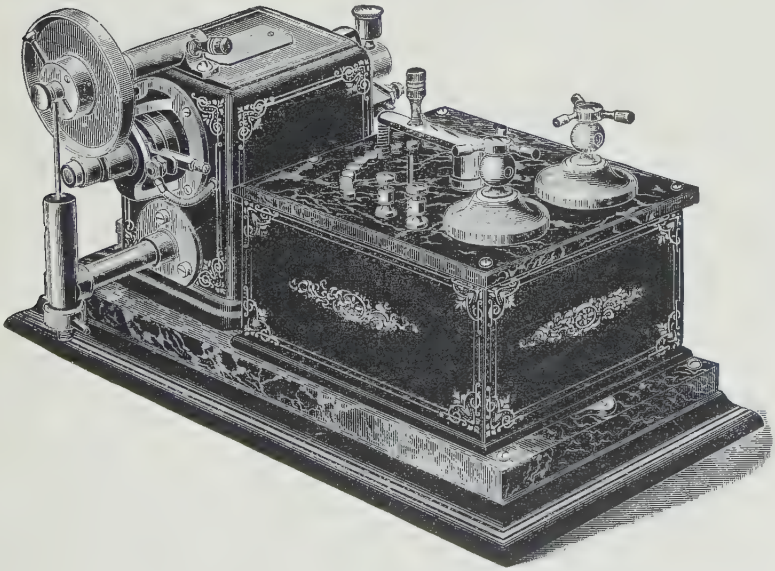
Pittsburg, and later, in 1893, improved by Pynchon (Fig. 13). The pneumomassage of the ear drum is recommended in deafness and ear

Fig. 12



Delstanche's rarefactor and artificial leech.

Fig. 13



The Victor electrocautery with Pynchon's pneumomassage pump.

noises of catarrhal origin, though its value has been greatly exaggerated. Delstanche was of such high repute that he was awarded the Lenval prize

for having designed the best instrument for relief of deafness, hence the procedure was adopted by aurists all over the world. Subsequent experience with it and its modifications has not justified the high expectations with which it was received. Pneumomassage has a place in aural practice, however, as by it the mucous membrane is brought into a more active and resistant state, and the labyrinth is also stimulated to greater functional activity. In a limited number of cases the ossicles of the ear are rendered more mobile and transmit sound better after its application. Tinnitus is also occasionally relieved by it. Such cases require rare skill and knowledge to determine what is best to do for them. Routine inflation and pneumomassage are almost without result except in a few cases. Accurate diagnosis is of first importance; then the treatment should be very carefully and intelligently prescribed. Few cases of deafness and tinnitus are relieved by pneumomassage.

Then, too, the massage apparatus should be regulated to suit each case. The length of the piston stroke, the frequency of the vibrations, and the length of time the massage should be used are questions to be settled according to the peculiarities of each case and the experience and judgment of the surgeon. Massage *per se* is of no value as a therapeutic agent. It is only when it is used with "brains" that it becomes of value. Surgeons who are uninformed and inexperienced are often tempted to furnish their offices with formidable-looking mechanical devices, with the belief that they are thus preparing themselves to adequately cope with disease. If they are intelligent observers, they soon learn that the "man behind the gun" is the first requisite for the attainment of success.

I have, however, found the hand apparatus of Delstanche of the greatest value as a diagnostic and therapeutic agent. With it the ear drum may be observed under compression and rarefaction, and points of adhesion and of atrophy are clearly demonstrated. When the air is rarefied in the meatus, the points of adhesion being fixed, the remainder of the membrane bulges outward, leaving no doubt as to the condition of the middle ear. If there is an atrophic area in the ear drum it bulges like a blister beyond the other parts of the membrane. If the otoscopic portion of the apparatus is provided with a magnifying lens the texture of the ear drum may be clearly demonstrated.

Aside from the diagnostic value of the Delstanche apparatus, its greatest usefulness is in the treatment of the exudative forms of middle-ear catarrh. It is in the protracted course of these cases that the adhesive processes form. The viscid exudate agglutinates the ear drum to the inner tympanic wall, becomes organized, and thus permanently fixes it to the inner wall of the middle-ear cavity. The timely and intelligent use of the Delstanche rarefactor, or other pneumomassage apparatus, may prevent permanent adhesions. The apparatus should in the beginning be used daily with a slow, long stroke of the piston. After the inflammatory process has abated and the exudate is less viscid and less profuse the treatment may be gradually reduced in frequency and finally abandoned. The length of the stroke (force of the suction) should

be gradually diminished, as a too long-continued stretching of the membrana tympani will render it abnormally lax from pressure (suction) atrophy.

Another device for the massage of the ear drum consists of a glass tube partially filled with metallic mercury (Fig. 14). The open end of the tube is shaped to fit the external meatus, and when not in use is closed with a rubber cork. Its application is simple, the uncorked end being placed firmly in the external meatus, and the patient instructed to move the head from side to side, allowing the mercury to drop against the ear drum. This procedure is repeated several times at each daily seance. According to Dr. Joseph C. Beck, its originator, the *rationale* of its use consists in the impact of the mercury against the malleus and ear drum, the force being transmitted to the entire ossicular chain and to the labyrinth. This stimulates the functional activity of these structures and improves the condition present. Dr. Beck has found its chief usefulness in the relief of the tinnitus rather than the deafness, a fact which to my mind is significant. That is, the mechanical shocks thus applied to the membrana tympani and transmitted to the labyrinth affect the circulation of the labyrinth, improve the nutrition, and increase the local leukocytosis. Dr. Beck has also noted that the improvement was usually transient, lasting only a few days or weeks after discontinuing the treatment.

FIG. 14

Beck's mercury massage.

The Electrocautery.—So much has been said within recent years about the use, or rather the uselessness, of the electrocautery (Fig. 13) that I feel impelled to defend it. It is still a very useful apparatus, and an office is incomplete without it. It is true that it has been too frequently, indiscriminately, and unintelligently used, but it still fills a place of great usefulness in the armamentarium of the specialist. Its usefulness in turgescent rhinitis has been greatly abridged by the improved methods of operating upon the nasal septum (notably the submucous resection), but even in this condition it still affords a means of temporarily overcoming the excessive swelling of the inferior turbinated bodies. It also affords a valuable means of treating chronic granular pharyngitis with lymphoid enlargements along the lateral and posterior walls of the pharynx. Still other uses could be described, but as they are mentioned in connection with the respective diseases, the two citations are sufficient to show that the electrocautery apparatus is not an obsolete instrument.

Spray Tubes.—The spray tubes and the medicated fluids used in them have also come under the ban as therapeutic agents. There was a time when the rhinologist and laryngologist was called the "spray specialist," more derisively a "squirt-gun doctor." Whatever grounds

there may have been for these characterizations it is certain that they do not apply to the specialist of the present time. Nearly all special surgeons now recognize the futility of attempting to cure diseases of the nose and throat by means of medicated water and oil. The etiology of the catarrhal and suppurative inflammations of the nose and throat is better understood, and the ideas concerning their treatment have undergone corresponding changes. It is being more and more recognized that mucous-lined cavities are subject to catarrhal and infective inflammation somewhat in proportion to the degree of obstruction to their drainage and ventilation. This one factor is probably the most significant etiological factor emphasized in recent years. Goodale and Jonathan Wright emphasize it in reference to the crypts of the tonsil. Heath has recently emphasized the same truth in reference to the mastoid antrum and the middle ear. (See *Meatomastoid Operation;* also the *Clinical Anatomy of the Nose,* and the *Inflammatory Diseases of the Nose and Accessory Sinuses.*)

FIG. 15

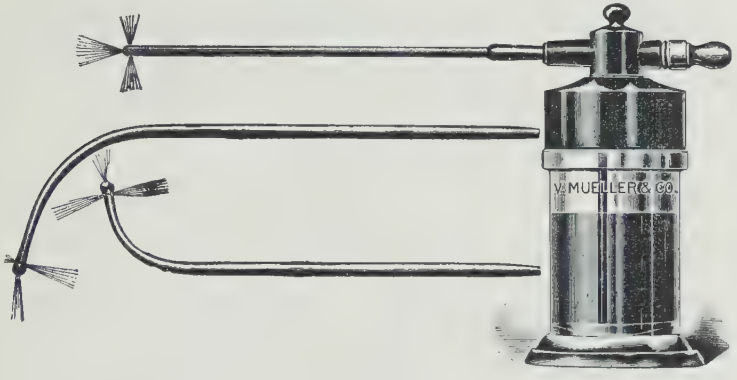
De Vilbiss' atomizer and nebulizer.

In view of this more modern conception of the etiology of the inflammatory diseases of the ear, nose, and throat, surgical procedures have largely replaced the topical and caustic applications once in popular favor. The spray tube, or atomizer, occupies a less conspicuous place than it did a few years ago (Fig. 15). An array of fifty or a hundred spray bottles, each with a different medicated or perfumed solution, is no longer a necessary part of an office outfit; indeed, such an array of spray formulæ is in some ways a confession of an antique, if not altogether obsolete, conception of medical practice. Spray tubes are, nevertheless, necessary adjuncts to the office outfit, as they should be used to cleanse the nasal and throat cavities before operating and treating acute and chronic inflammations.

George F. Hawley's spray tube (Fig. 16) is the best cleanser, as it throws out a coarse spray in every direction and softens and dislodges

the tenacious and dried secretions. The straight tip may be inserted into the sphenoidal sinus after the middle turbinate has been removed, and the secretions thoroughly washed out. The apparatus as a whole is an excellent substitute for other methods of irrigating the nose. The straight tip may be bent to conform to the requirements for reaching the frontonasal duct and maxillary sinus. Postnasal and laryngeal tips make it a universal instrument for irrigating the upper respiratory passages on account of the improved methods of topical and surgical treatment now in vogue.

Fig. 16



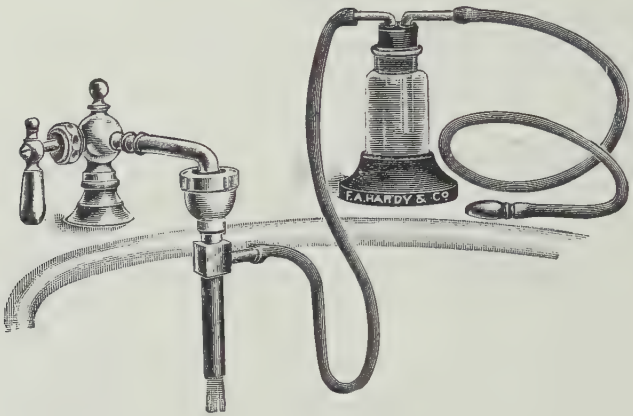
Hawley's spray tube.

The Mechanical Vibrator.—Some years ago the mechanical vibrator was mentioned as acting favorably upon tinnitus and deafness, but its more general use by English and American otologists has demonstrated its comparative uselessness for these purposes. At that time it was stated that when applied over the spinal column it seemed to act favorably upon the ear. I have tried it faithfully for this purpose, with no appreciable effect. Its chief field of usefulness is in reducing the swelling and sensitiveness of the glands of the neck and the headache accompanying the various sinus affections. But even these conditions are better and more pleasantly ameliorated by the leukodescent lamp. The vibratory or mechanical massage increases the lymphatic flow, improves the nutrition, and increases local leukocytosis. Hence, it relieves pain and tenderness, and reduces the activity of an inflammatory process, provided it can be applied to the parts. In this respect it acts upon the principle of Bier's constriction and negative pressure treatment and the leukodescent-light treatment; that is, it increases the local leukocytosis, improves the local nutrition, and thus diminishes the infectious process.

Negative Pressure Apparatus.—This apparatus consists of a device whereby the air pressure is reduced in the upper air passages, notably the nose and accessory sinuses (Figs. 17 and 18). The negative air pressure within the nose and accessory sinuses facilitates the discharge of the

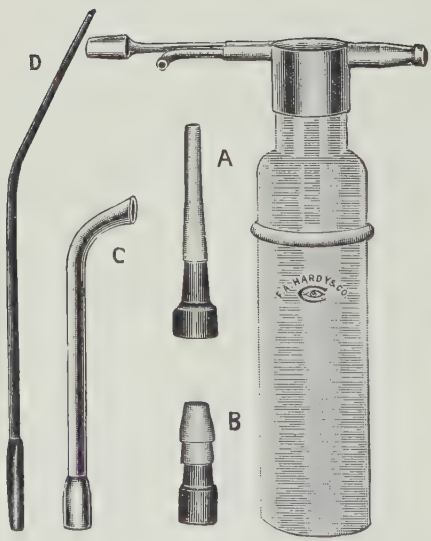
secretions and purulent accumulations, increases the local nutrition and leukocytosis, and acts favorably upon the inflammatory process. Its chief field of usefulness seems to be in the treatment of the subacute inflammations of the sinuses, though it exerts a favorable influence upon chronic sinuitis.

Fig. 17



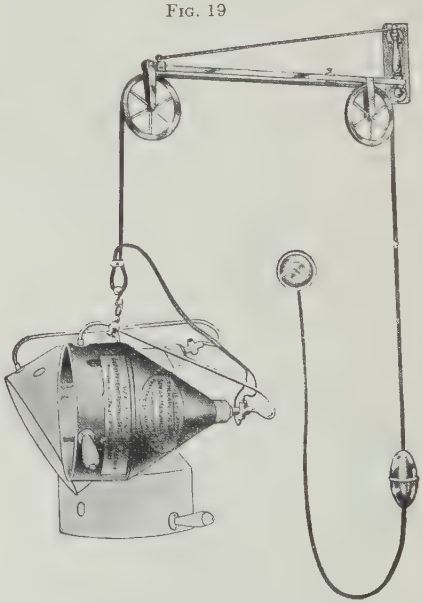
Brawley's vacuum aspirator.

Fig. 18



Pynchon's modification of Dabney's vacuum aspirator.

Fig. 19



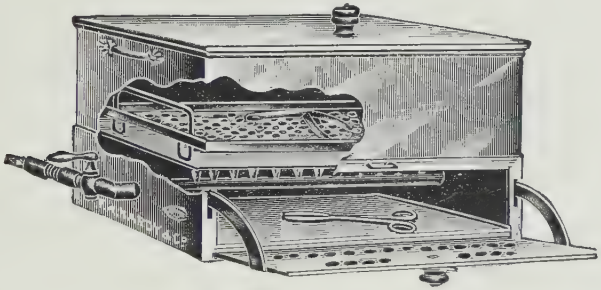
The leukodescent therapeutic lamp.

The Leukodescent Lamp.—The leukodescent lamp is a single incandescent globe of 500 candle-power (Fig. 19), around which is placed a reflector eighteen inches in diameter. The reflector focuses the rays of light, thus increasing their penetrating power. The therapeutic properties

of the leukodescent light are in the heat and chemical rays. The leukodescent light is rich in blue-violet rays, in addition to the light and heat rays. The blue-violet are very active chemical rays and increase the tissue metabolism and the leukocytosis, thus providing for the destruction of the pathogenic bacteria.

Clinically, I have found the leukodescent light of value in infectious and inflammatory processes. For instance, I have seen cases of chronic maxillary empyema with granulations cease discharging under its influence. The pain, tenderness, and swelling likewise disappeared. In no case, however, have I seen a cure by this mode of treatment. In acute sinuitis I have seen marked and rapid improvement follow its use. Infection of the mastoid wound rapidly improves under its use three times daily. Cervical adenitis usually responds readily to the rays. Pain of almost any origin is relieved and in many cases stopped by it. The pain of sarcoma is almost invariably checked. It seems to exert a slight control over an oozing postoperative hemorrhage. Its power to increase tissue metabolism and local leukocytosis reduces

FIG. 20



Pynchon's sterilizer and instrument dryer.

the bacterial activity. The latter is probably due more to the increased leukocytosis than to the bactericidal property of the rays. While they are bactericidal when applied continuously for ten minutes at a distance of thirteen inches in the laboratory, they are probably not bactericidal at eighteen inches for a few moments at short intervals in their clinical application. The rays are too hot to be tolerated constantly at close range, hence the effects produced in laboratory experiments cannot be duplicated in actual practice.

Lamps of less candle-power are correspondingly poor in the blue-violet rays, the 50 candle-power lamp having scarcely a trace of them. It has been shown that ten 50 candle-power lamps grouped have identically the same quality of rays as a single 50 candle-power lamp, and that the rays are in no way similar to those given off by a 500 candle-power lamp. A single 500 candle-power lamp should be chosen, as a lamp of less capacity is not sufficiently rich in the chemical rays to produce the best results.

A Sterilizer for Instruments and Gauze. An office outfit is not complete without a sterilizer of some kind. All instruments should be boiled

in a 2 per cent. solution of sodæ biboras for at least twenty minutes before they are used, for either examinations, treatments, or surgical operations. The instruments may be boiled in a porcelain-lined bucket or pan, or in a specially designed sterilizer, as shown in Fig. 20. The apparatus shown in the illustration is provided with a drying chamber in addition to the boiling tray, and is recommended on this account. Instruments are often damaged or altogether ruined because they are not dried after being sterilized. With this sterilizer they may be boiled and dried after an operation.

Topical Applications.—Topical remedies which should have place upon the treatment table are numerous, though individual preference may greatly modify their number and character. I shall only refer to those which have proved satisfactory in my practice.

Nitrate of Silver.—The following solutions of the nitrate of silver should be kept on the treatment table in blue-glass bottles, or in a cabinet within convenient reach of the surgeon or his assistant:

℞.—Argenti nitratis . . . . . gr. x
Aquæ des. . . . . ℥j—M.

This is approximately a 2 per cent. solution of the silver salt, and is useful when a mild but positive astringent action is required, as in simple subacute catarrhal inflammation of the upper respiratory tract. It may be applied with a spray tube, the essential parts of which are made of hard rubber and aluminum, or of glass. Other metals are acted upon by the silver salt, and are not suitable for the silver solutions on this account. The silver solution may also be appiled with a cotton-wound applicator. A camel's-hair brush is not recommended, on account of the difficulty of keeping it sterile.

℞.—Argenti nitratis . . . . . gr. xx
Aquæ des. . . . . ℥j—M.

This solution is approximately 4 per cent. in strength, and may be used as No. 1 when a more positive astringent and antiseptic action is required.

℞.—Argenti nitratis . . . . . gr. xl
Aquæ des. . . . . ℥j—M.

This solution is approximately 8 per cent. in strength, and is useful in the more chronic catarrhal inflammations of the upper respiratory tract. Solutions of greater strength than this are rarely indicated in chronic inflammations of the mucous membrane except when a caustic action is required. Greater strengths are apt to cause irritation and an aggravation of the local chronic inflammation.

In the very acute inflammations a much higher percentage of silver may be used.

℞.—Argenti nitratis . . . . . ℥j
Aquæ des. . . . . q. s. ad ℥j—M.

This is a 12½ per cent. solution, and is a valuable local remedy in acute lacunar inflammation of the tonsils. The more acute the attack and the more edematous the tissue the stronger the silver solution should be.

℞.—Argenti nitratis . . . . . ʒij
Aquæ des. . . . . q. s. ad ℥j—M.

This is a 25 per cent. solution, and is useful as a local application in acute infectious inflammations of the fauces. It is especially useful in acute lacunar tonsillitis, one application in the primary stage often being sufficient to abort the inflammatory process.

℞.—Argenti nitratis . . . . . gr. cccxxxij
Aquæ des. . . . . q. s. ad ℥j

This is a 90 per cent. solution, and is useful in acute lacunar tonsillitis in the most virulent and acute stage. It should only be applied when the inflammation is very recent and aggravated in type. The tissues should be succulent and highly inflamed. In such a case it is a specific remedy. I have never seen a case corresponding to the above description in which the second application of the remedy was necessary. Its use in this strength is not painful, but, on the contrary, relief immediately follows.

If this strength of solution were applied to a subacute inflammation the chemical trauma would probably aggravate the existing inflammatory process rather than relieve it. A solution of silver salt of this strength coagulates the mucous secretions and blanches the surface of the inflamed mucous membrane. It is also a powerful germicide. The inflammatory infiltration of the tissue is checked and the vitality of the infective bacteria is greatly impaired.

Caution should be observed in using silver nitrate. The salt in any strength has a marked irritating effect on the intrinsic muscles of the larynx. To avoid this accident the cotton-wound applicator should be freed of the excess of the solution by squeezing it with a liberal wad of cotton. When this is done the inflamed area should be lightly brushed with it.

The following rules are valuable: (*a*) The milder the inflammation the milder the solution (*b*) The more intense the inflammation the stronger the solution.

Guaiacol Solutions.—Solutions of guaiacol in olive oil are useful local remedies in acute inflammation of the fauces and pharynx.

The strengths recommended are 10, 25, and 50 per cent. of guaiacol in pure olive oil. The more severe the inflammation the stronger the solution required.

While guaiacol is not as efficient a remedy in acute tonsillitis as the stronger solution of silver, it is nevertheless very positive in its action, many cases requiring but a few applications to check the inflammatory process. It produces a pungent, hot sensation which lasts for about thirty seconds.

Compound Tincture of Benzoin.—The compound tincture of benzoin is a valuable local remedy in the throat when a mild but positive astringent and antiseptic remedy is indicated. It may be used in chronic granular pharyngitis during the mild exacerbations of the disease with good effect.

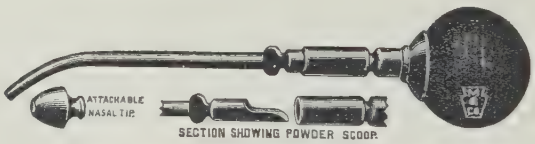
Its chief value is as an adjunct in dressing the nasal accessory cavities. The gauze should be moistened in the solution, the excess removed by squeezing, and packed in the nasal cavity. It prevents decomposition and stimulates healthy granulations. A plain gauze dressing in the nasal chambers, if allowed to remain more than twenty-four hours, often takes on a very offensive odor. If the gauze is moistened with the compound tincture of benzoin, it may remain in the nose seventy-two hours without acquiring an offensive odor.

A foul-smelling chronic otorrhea may be rendered sweet by mopping the cavity dry and applying a dressing of gauze moistened with the compound tincture of benzoin.

Subnitrate of Bismuth Powder.—This powder may be used with gauze dressings as a substitute for the compound tincture of benzoin. It also prevents decomposition, though not over so extended a period.

It may also be insufflated (Fig. 21) into the nose after an intranasal operation, where it forms a coating which acts as a mechanical and a chemical protection to the underlying tissue.

FIG. 21



Powder insufflator.

Ichthyol Solutions.—Ichthyol in aqueous and glycerin solutions may be used as a topical application in the nasal chambers where there is a foul or ozenic secretion. The nose should be packed with cotton or gauze saturated with the solution. Personally, I prefer to use a cork-screw applicator wound with cotton and dipped in the ichthyol solution. This is then introduced into the nasal cavity and the applicator removed with a reverse screw motion, leaving the ichthyol pad in the nose. This should be left in place for from ten to thirty minutes, according to the degree of infection and tumefaction of the tissue. If the secretions are profuse and dried in the nasal cavities, the aqueous solution should be used; if there is a state of sepsis and local tumefaction of the tissues, the glycerin solution should be used on account of its hygroscopic action.

Iodine Solutions.—Iodine in a glycerin menstruum is a valuable remedy in chronic granular pharyngitis, and in those cases of middle-ear catarrh associated with granular pharyngitis or atrophic rhinitis.

The following formulæ may be used in such cases:

℞.—Tr. iodini . . . . . ♏xlviij
Glycerini . . . . . q. s. ad ℥j—M.

℞.—Iodoformi . . . . . gr. j
Potas. iodidi . . . . . gr. x–xx
Morphia sulphatis . . . . . gr. j
Glycerini . . . . . ℥j—M.

℞.—Iodini . . . . . gr. v–xx
Potas. iodidi . . . . . gr. x–xxx
Ol. gaultheriæ . . . . . ♏v
Glycerini . . . . . ℥j—M.

℞.—Tr. iodidi,
Tr. ferri chl.,
Glycerini . . . . . āā q. s. ℥j—M.

The fourth formula is very astringent, and is used to promote even healing by granulation after tonsillectomy in adults. It is also of great value in the subacute type of granular pharyngitis.

Carbolic Acid.—Carbolic acid may be used in any strength from 10 to 95 per cent. aqueous or glycerin solution.

℞.—Carbolic acid . . . . . gr. xx
Glycerin . . . . . ℥j—M.

This is approximately a 4 per cent. solution, and may be used in subacute dry dermatitis of the external auditory meatus and in subacute otitis media.

℞.—Carbolic acid . . . . . ℥j
Glycerin . . . . . q. s. ad ℥j—M.

This is a 12 per cent. solution, and may be used in acute otitis media. It should be dropped into the meatus two or three times daily and a cotton plug introduced to prevent its escape (A. H. Andrews). It is claimed that if dropped into the meatus in the initial stage of acute suppurative otitis media it aborts the further progress of the inflammation in nearly every instance. On the other hand it is claimed that its frequent use causes a fibrosis and thickening of the ear drum, and thus causes permanent diminution of hearing. It may be said, however, that its frequent use is not often required to abort an attack of acute otitis media.

℞.—Carbolic acid . . . . . gr. ccclvj
Aquæ des. . . . . ♏xxiv—M.

This is a 95 per cent. solution of carbolic acid, and may be used when a superficial caustic effect is desired, as in infective granulomata of the middle ear and mastoid, either before or after operation. I have occasionally used it in cases of old, foul-smelling otorrhea to diminish the odor and to stimulate healthy granulation. (See Chemical Caustics.)

Alcohol.—Alcohol is also a valuable remedy for topical applications. I know of no better ingredient for a gargle than alcohol. It is astringent and antiseptic, and, when properly diluted, is grateful to an inflamed surface.

℞.—Alcohol,
Cinnamon water . . . . . āā ℥ij
Formaldehyde . . . . . ♏ij
Glycerin . . . . . ℥v
Aquæ des. . . . . q. s. ad ℥viij—M.

The above formula is a good gargle in acute tonsillar and pharyngeal inflammations and in the soreness following the removal of the tonsils. In very young children it may be used in a more diluted form.

In chronic otorrhea alcohol may be used in the following dilutions and mixtures:

℞.—Alcohol . . . . . 1 part
Aquæ des. . . . . 2 parts—M.

℞.—Alcohol . . . . . 1 part
Aquæ des. . . . . 1 part—M.

℞.—Alcohol . . . . . 2 parts
Aquæ des. . . . . 1 part—M.

℞.—Alcohol . . . . . 3 parts
Aquæ des. . . . . 1 part—M.

℞.—Alcohol . . . . . 95 per cent.

The alcohol dilutions given above are used principally in the treatment of chronic suppurative otitis media.

They constitute the so-called "alcohol treatment" of this disease: The meatus is first filled with the weakest solution, then mopped out, and each solution applied in series until the patient tolerates the 95 per cent. solution. If the strongest solution is applied at once it causes considerable pain and irritation, whereas if the strength is gradually increased unpleasant results are avoided.

Alcohol is a positive astringent and antiseptic remedy of considerable value.

℞.—Alcohol (95 per cent.) . . . . . ℥j
Boric acid . . . . . gr. xx—M.

℞.—Alcohol (95 per cent.) . . . . . ℥j
Iodoform . . . . . gr. v—M.

The addition of boric acid and iodoform is supposed to give the local antiseptic effect of these drugs. If an excess of either drug is added, and the solution is agitated just before the instillation of the solution, a precipitate of the partially suspended drug is deposited on the diseased mucous membrane.

These solutions should be used after having applied the weaker alcoholic solutions.

Ointments.—Various drugs may be prepared with an oily menstruum, preferably lanolin, as it has greater affinity for the mucous membrane than vaseline. Pure olive oil may also be used as a menstruum. The following mixtures are recommended:

℞.—Zinc oxide . . . . . gr. xlviij
Lanolin . . . . . ℥j—M.

℞.—Zinc oxide . . . . . gr. xlvij
Morph. sulph. . . . . gr. j
Atropine . . . . . gr. $\frac{1}{100}$
Lanolin . . . . . q. s. ad ℥j—M.

The first formula is soothing to an inflamed surface, and may be applied in those cases in which there is an irritating mucous or seromucous discharge in catarrhal sinuitis. It is also of use in the massage

of the nasal mucous membrane in rhinitis with collapse, and in turgescence of the "swell bodies." For this purpose a delicate silver applicator should be wound with a small wisp of cotton and dipped into the ointment. The nasal mucous membrane should then be gently massaged with the ointment, the probe being lightly held between the thumb and forefinger. The wrist movement, or the combined wrist and forefinger movement, should be used in performing the massage. The applicator should he held so lightly that if the cotton-wound applicator should strike a turbinated body or other obstruction the probe will slip through the fingers and do no damage.

The sensitiveness of the mucous membrane may be quickly removed by the above procedure.

The second mixture is of value when the nasal mucous membrane is sensitive and when there is an acute exacerbation of the inflammation. The morphine and atropine relieve the sensitiveness and reduce the congestion.

℞.—Ichthyol . . . . . gr. xlviii
Lanolin . . . . . ʒj—M.

The ichthyol ointment may be used in those cases where the secretions are dried in the nasal cavities to stimulate the glandular functions. It may be applied by massage, as described above.

Chemical Caustics.—Chemical caustics are largely replaced by the electrocautery, though there are instances in which the chemical caustics are preferable. The following are recommended:

Carbolic Acid (95 per cent.).—Where a superficial and diffused cauterization is desired, as in an unhealthy granulating surface, carbolic acid is an ideal caustic agent. It does not penetrate deeply, nor does it produce pain. It is also of value in cases of old suppuration of the ear, in which there is a foul odor and exuberant granulations. The ear should first be thoroughly freed from secretions with a cotton-wound probe and the carbolic acid applied afterward. After one minute has elapsed alcohol should be dropped into the meatus to check the action of the carbolic acid and to prevent its action upon the skin of the meatus and auricle during its removal. The carbolic acid should be dropped into the middle ear with a medicine dropper, care being exercised to avoid contact with the cutaneous surface.

Carbolic acid may also be used in the pharynx when a diffused superficial caustic action is desired, as in a mild case of granular pharyngitis, though in these cases it is usually preferable to puncture the follicles or nodules scattered over the pharyngeal wall with the galvanocautery.

Chromic Acid.—Chromic acid has long been a favorite chemical caustic in the nose, throat, and ear, though it has been largely replaced by the galvanocautery. A few crystals are engaged upon the end of a probe and held over an alcohol or gas blaze to drive off the water of crystallization, but not long enough to reduce them to an ash or cinder. The bead of acid thus formed is drawn across the area to be cauterized,

where it rapidly abstracts the water from the tissue and thus destroys or cauterizes its superficial layers.

It may be used in turgescent rhinitis, follicular pharyngitis (granular pharyngitis), and in any other condition requiring cauterization. It is not as deep in its penetration as is usually desired in either of these conditions, hence it is not as reliable as the galvanocautery.

In order to increase its efficiency, Norval H. Pierce and Max A. Goldstein have devised instruments for its subcutaneous use. The submucous method has not, however, appealed strongly to the profession, as the galvanocautery is easily and efficiently applied with equally good or even better results.

It should be remembered that chromic acid is quite irritating to the kidneys, and may cause albuminuria. Its extensive use is, therefore, contraindicated in cases already thus affected.

Technique.—(*a*) Local cocaine anesthesia. (*b*) Puncture the mucous membrane at the anterior end of the free border of the inferior turbinated body. (*c*) Introduce a probe or other elevator through the puncture and tunnel the substance of the mucous membrane, keeping near the periosteum. (*d*) Introduce the Goldstein concealed probe containing the bead of chromic acid into the depth of the tunnel. (*e*) Uncover the bead of chromic acid and withdraw it through the tunnel. This cauterizes the wall of the tunnel within the mucous membrane. If sloughing does not occur the result is very good (Fig. 106).

Trichloracetic Acid.—This is a valuable chemical caustic agent and is generally used in a 20 per cent. solution. It has been employed chiefly in tuberculosis of the larynx, in conjunction with curettage, and in hypertrophied and diseased tonsils, after splitting the walls of the crypts.

In laryngeal tuberculosis after the intralaryngeal removal of all the tuberculous tissue available by this route the operated area is swabbed with a 20 per cent. solution of trichloracetic acid, to destroy any remaining tuberculous tissue and to seal up the lymphatic openings to prevent the spread of the tuberculous process.

Kaufmann has recommended the free and deep incision of the crypt walls of the tonsils, especially of those crypts opening into the supratonsillar fossa, and applying a 20 per cent. solution of trichloracetic acid to the incised surfaces. More than one sitting is usually required for this purpose. The object of this procedure is to destroy the diseased epithelial lining of the crypts and to cause cicatricial contraction of the substance of the tonsil. In this way the tonsil is reduced in size and its non-resistant cryptic epithelium is destroyed.

The acid applications are very painful for a prolonged period of time. This, together with the fact that repeated applications are often necessary, renders the procedure an undesirable one. The complete removal of the tonsil by dissection is a more certain and desirable procedure, as both tonsils may be removed at one sitting.

Nitrate of Mercury.—A 10 per cent. solution of the nitrate of mercury may be used to cauterize deep sloughing syphilitic ulcers of the nose and

throat, as it excites healthy granulation, and thereby checks the sloughing and syphilitic ozena.

Antiseptic and Detergent Solutions.—The cleansing of the nose and throat with detergent sprays and washes is not as popular a procedure now as formerly. Experience has shown that such applications exert little curative action on catarrhal and other diseases. They do, however, promote temporary increase in the hyperemia and leukocytosis. Such solutions also stimulate the constrictor muscle fibers of the "swell bodies" of the turbinals, and thus temporarily reduce the turgescence. The antiseptic action is probably but slight and of little value. The three useful effects of the antiseptic and alkaline nasal washes are therefore as follows: (*a*) Detergent or cleansing effects. (*b*) Muscular contraction of the interlacing fibers of the "swell bodies." (*c*) Slight promotion of the reaction of inflammation. The detergent and stimulating solutions recommended are as follows: (1) Seiler's solution. (2) Dobel-Pynchon solution.

(2) ℞.—Powd. sod. bibor. (Squibb),
Powd. sod. bicarb. (Merck) . . . . . āā ʒij
Thymoline . . . . . Oss
Glycerin (C. P.) . . . . . Oiss

First mix and triturate the two salts and place them in a one-gallon bottle, adding one-half the quantity of glycerin; then let it stand twenty-four hours uncorked, with frequent agitations. Next add the remainder of the glycerin and continue the agitations for another twenty-four hours, with the bottle uncorked as before. Lastly, add the thymoline and let the solution stand twenty-four hours. One ounce of this mixture should be added to one pint of water, when it is ready for use.

The solutions may be used with an atomizer, a nasal douche, or a syringe. They may also be used as gargles, although the distinctly alkaline taste is usually disagreeable to the patient.

Oily Solutions for Use with a Nebulizer.—Aromatic and antiseptic drugs may be added to an oily menstruum and thrown into the respiratory tract with a nebulizing device. The action of such mixtures is as an emollient or protective agent, and as a stimulant to the mucous glands. They also cause contraction of the circular muscle fibers of the arterioles, and thereby reduce the congestion. The effects are transient, and afford relief without exerting a marked curative effect.

The following formulæ are recommended:

1. Chlorotone inhalant.

℞.—Chlorotone . . . . . gr. xv
Camphor . . . . . gr. xxx
Menthol . . . . . gr. xxx
Oil cinnamon . . . . . ♏v
Oil petrolatum . . . . . ʒij—M.

2. Acetozone inhalant.

℞.—Chlorotone . . . . . ♏vij
Acetozone . . . . . ♏xv
Oil petrolatum . . . . . q. s. ad ʒij—M.

The spray bottles and nebulizing bottles devised by De Vilbiss (Fig. 15) have proved more satisfactory than any others, as their construction is simple and they rarely need repairing or other attention.

Hawley's spray tube is also a useful device for washing the nasal cavities, and is often preferable to the spray tube, as it does not injure the epithelium of the nasal mucous membrane.

The air pressure allowable for spraying the various mucous surfaces with De Vilbiss' spray apparatus is as follows: (*a*) The nasal mucous membrane, 4 to 10 pounds. (*b*) The epipharynx (nasopharynx), 8 to 20 pounds. (*c*) The mesopharynx (oropharynx), 10 to 30 pounds. (*d*) The hypopharynx, and larynx, 10 to 30 pounds. The air pressure needed for De Vilbiss' nebulizing bottles, 10 to 40 pounds.

The Pynchon and Hubbard regulating tanks, elsewhere mentioned, are of great value in conjunction with the spray and nebulizing tubes. Hubbard's regulating tank is especially recommended, as it has a filtering device for cleansing the air. It also has an arrangement for heating the air.

Solutions which Produce Ischemia.—Solutions which produce local blanching of the mucous membrane are chiefly derived from the suprarenal glands of sheep. They produce a powerful contraction of the circular muscle fibers of the arteries, which lasts for several minutes. They are on this account of diagnostic and therapeutic value. They also reduce the amount of primary hemorrhage in operations.

The following formulæ are recommended:

℞.—Adrenalin chloride . . . . . 1 to 1000

℞.—Adrenalin chloride . . . . . 1 to 2000

℞.—Adrenalin chloride . . . . . 1 to 4000

It is rarely necessary to use the first formula except when there is a great deal of secretion and blood to dilute the solution. If applied to a clean mucous membrane the second and third formulæ are of sufficient strength to contract the vessels. Local ischemia is produced for diagnostic purposes in the various forms of rhinitis and in reducing the engorgement of the tissues to admit a view of the nasal chambers. Adrenalin is also used to check local oozing of blood after operations.

CHAPTER IV

THE ETIOLOGY OF DEFORMITIES AND DEVIATIONS OF THE SEPTUM NASI

According to Freeman, Trendelenburg was the first to describe the high-arched palate with deformity of the septum nasi, though he did not consider it due to lack of development of the maxillary bones. Loewy was of the same opinion, though he regarded the Gothic arch as of rachitic origin. Zuckerkandl does not accept the rachitic origin, as he believes that the lower jaw and not the upper exhibits the rachitic influence. However this may be, Freeman reminds us that it is common to find the Gothic arch associated with deviated septa. He shows that in 302 cases of high-arched palate, 290, or 96 per cent., were associated with deviated septa.

In studying the Mütter collection, Freeman found many straight septa associated with Gothic palates, thus demonstrating that a high arch is not necessarily a cause of septal deviation. Indeed, he believes that the faulty development of the superior maxillæ is a fruitful source of deviated septa, especially in dolichocephalic heads. The skulls were those of non-Europeans, in whom, as Zuckerkandl has pointed out, the deformities of the septum are much more infrequent than in Europeans. Mosher has recently called attention to the low position of the floor of the antrum of Highmore in skulls with the Gothic palate.

As the Gothic arch is naturally present in infants, it is easy to understand that anything which interferes with the development of the skull will prevent development of the hard palate and its consequent descent. Indeed, in such cases the later development of the alveolar processes and the eruption of the teeth will cause the arch to become more peaked. As the arch remains high, the septum in its further development must bend to make room for its growth. Welcker, in support of this view, has shown that those cases in which one maxillary bone descends, the other remaining high-arched, convexity of the septum is toward the descended maxilla.

According to Eugene S. Talbot, Morgagni believed that deviated septa were due to excessive development of the vomer, while Jarvis reported four cases in one family suggesting an hereditary influence. Talbot believes that direct hereditary influence is rare, though there may be a family development of the facial skeleton, as shown by Sachus' and Welcker's investigations.

According to Bosworth, the deformities of the septum are usually traumatic in origin. He points out that an injury to the nose need not be attended by an immediate and obvious deformity, but it may set up

a low-grade inflammation, which in a number of years finally results in an obstructive malformation of the septum. This is undoubtedly a frequent cause of septal deviations, especially of the anterior cartilaginous portion, which is exposed to traumatic influences. That it is a frequent cause of deformity of the bony portions (perpendicular plate and the vomer) is extremely doubtful, as they are protected from blows by the nasal and superior maxillary bones.

Talbot holds that deviations of the septum are due to the unequal development of the adjacent bones, more especially the turbinated bodies. Their development in turn depends upon the growth of the facial bones, which are modified as the facial angle increases and prognathism is lost. The turbinated body being displaced or enlarged toward the septum, the septum is crowded to the opposite side. The septum is not necessarily pushed over by direct contact of the turbinated bone, as the respiratory currents of air may cause it to deflect during the prepuberty period, when the vomer and perpendicular plate are soft and cartilaginous. Talbot believes that the underlying cause of septal deformities is a neurosis and degeneracy, in which conditions there may be an imballance of development of the various bones of the face, total collapse of the outer walls of the nose, associated with an arrest of the development of the bones of the face, jaws, dental arch, chest, and shoulders.

Summary.—1. Morgagni thought they were due to excessive development of the vomer; the vomer crowding upward against the descending perpendicular plate of the ethmoid caused septal deflection to one side, in order to allow of continued development.

2. Trendelenburg and Freeman think the chief cause of the deflection is in the persistent high or Gothic arch of the hard palate. The vomer and the perpendicular plate of the ethmoid are thereby crowded and deflected in order to find room for further complete development.

3. Jarvis believes the chief cause is heredity, and quotes observations in support of this theory.

4. Schaus and Welcker advance the hypothesis of a faulty development of the facial bones, including those of the nose.

5. Bosworth argues that traumatism is the chief cause of deflections.

6. Talbot takes the theory of Schaus and Welcker and carries it still farther, and says that malformations of the septum are due to neuroses or stigmata of degeneracy, which result in irregular development of the facial bones. He believes that pigeon chest, adenoids, and deformed nasal septa are all due to the same neurotic influences, which arrest development in some parts while in others there is an increase in the development.

It is difficult to arrive at a final conclusion concerning these theories, as data of almost any kind can be found by one who diligently searches for it. It is easy to say there is excessive development of the vomer, and to report so many thousands of observations on skulls in which this theory is substantiated. Trendelenburg and Freeman have satisfied themselves that the Gothic arch is the cause. They say the high arch of

childhood does not descend as it should, and that the space for the vomer and the ethmoid plate is thereby encroached upon and deflection results. Talbot and others have studied the so-called high arch and find that it rarely exists, also that in some instances there is lack of lateral development of the superior maxillæ, which gives rise to the Gothic arch, or what appears to be an abnormally high arch. Actual measurements show them to be no higher than normal. Then, too, Talbot claims that many hard palates which are lower than the average are attended by septal deformities. He does not deny that traumatism does in some instances account for septal deformities, but he does deny that it is the chief cause of deviations. He belives that consanguineous marriages predispose to the neuroses and that facial deformities result therefrom. He holds that the facial bones are transitory and more subject to developmental influence than most parts of the skeleton, hence are either arrested or overdeveloped in those tainted with the stigmata of degeneracy.

Dr. Talbot's views present the most rational explanation of this much mooted question that has yet been offered. He does not name the overdevelopment of a particular bone nor does he claim the failure of the palatine arch (roof of the mouth) to descend as being the cause of deviations of the septum. If these conditions are present he claims they are incidental signs of a neurosis or degeneracy. The factor which causes excessive development of the vomer or of a Gothic or narrow (not high) arched palate causes the deformed septum also.

In conclusion, I will epitomize the etiology of deformities of the nasal septum as follows, in the order of their importance.

(*a*) Neuroses or stigmata of degeneracy which causes either an arrest or an excessive development of the bones of the face, including the nose; one of the expressions of the neurosis being deformed septa (Talbot).

The theories of Trendelenburg, Freeman, Morgagni, Jarvis, Schaus, and Welcker are swallowed up in that of Talbot. The individual theories they advance imperfectly convey the true explanation, while Talbot's comprehends them all and strikes at the root of the matter.

(*b*) Bosworth's traumatic hypothesis is true as to a certain number of cases. That it explains a majority or even a large percentage of them is doubtful.

The phraseology used by Talbot may be objectionable, inasmuch as it assumes that there are "stigmata of degeneracy" present in all cases not due to traumatism. It would be better, perhaps, to say that deflections of the septum are usually due to an incoördination in the development of the bones of the face, including those of the nose.

A CLINICAL CLASSIFICATION OF DEVIATIONS OF THE SEPTUM NASI

Malformation and deviation of the nasal septum may be either developmental or traumatic in origin. When developmental, any or all portions of the septum may be involved, whereas if it is of traumatic origin the anterior or cartilaginous portion only is affected, except in rare cases.

The point of chief clinical interest, however, is in the type and location of the deformity rather than in its origin. Even the type and location of the deviation have to a considerable degree lost their clinical significance in so far as treatment is concerned, since the perfection of the submucous resection of the septum has been accomplished, and so many types of septal malformations are found to be amenable to it.

Cartilaginous Deviations.—When the deformity is limited to the cartilaginous portion of the septum it is one of three types, viz.:

(*a*) A deflection of the anterior portion generally known as the columnar cartilage (Fig. 22). The antero-inferior border of this cartilage is turned outward into the vestibule of the nose and obstructs the respiratory passage. This type of deviation is not as serious in its consequences as those that obstruct the nasal chamber in the region of the middle turbinated body, as it only interferes with the ventilation of the nasal chamber and accessory sinuses, the drainage being unimpaired, except in so far as it depends upon the mechanical aid of the air current in propelling the secretions to the epipharynx.

FIG. 22

Deviation of the anterior portion of the septal or columnar cartilage, which may be removed through Hajek's incision by sharp dissection.

(*b*) An angular deviation in an antero-posterior direction is serious in proportion to its proximity to the middle turbinal. If it is limited to the region of the vestibule or the inferior turbinate it is of less clinical importance, though its removal is still indicated. If it obstructs both the middle and the inferior meatuses its removal is of greatest importance, as it interferes with both the drainage and ventilation of the nasal chamber and the accessory sinuses of the nose.

(*c*) A perpendicular deviation of the cartilage only interferes with the ventilation, without blocking the drainage of the secretions except anteriorly, which is inconsiderable.

Osseous Deviations.—For clinical purposes osseous deviations of the septum may be divided into three types:

(*a*) A bony ridge or crest along the upper border of the crista nasalis and the vomer. The direction of this deformity is backward and upward, usually beginning anteriorly about one-half inch from the border of the inferior portion of the nasal opening, near the floor of the nose. A ridge in this location does not necessarily obstruct the normal inspiratory tract (middle and superior meatuses), nor does it greatly interfere with the drainage of the secretions. It does, however, encroach upon the inferior turbinated body, and thus causes irritation of this important physiological organ and produces a sense of stuffiness of the nose. It interferes also to some extent with the posterior drainage of the secretions. It also projects to some extent into the respiratory pathway and forms a favorable place for the desiccation of the secretions. Crusts are, there-

fore, generally found upon the anterior extremity of the ridge, and in blowing the nose become detached, tear the epithelium, and give rise to epistaxis. While the ridge may not cause nasal obstruction, it should be removed on account of the mechanical irritation of the inferior turbinal and the resulting turgescent and hypertrophic rhinitis.

(*b*) The perpendicular plate of the ethmoid bone is often convex or cup-shaped and impinges upon the middle turbinate upon the side of convexity. This is, perhaps, one of the most serious obstructive lesions of the septum, as it obstructs both the drainage and the ventilation of the superior meatus, and of the frontal, ethmoidal, and sphenoidal cells. Sufficient importance has not been given this type of deviation, hence I wish to lay special emphasis upon it. It is this type of deviation, more than any other, that gives rise to conditions which result in catarrhal and suppurative inflammation of the accessory sinuses. In the first place the secretions are retained, undergo decomposition, and impair the vitality of the mucous membrane. Infection and inflammatory reaction naturally follow. The ostei of the sinuses become closed from swelling of the mucosa, and this still further interferes with the drainage. Furthermore, the ventilation of the superior meatus and of the obstructed sinuses is partially or completely lost, and the decomposition of the secretions is thereby encouraged. The oxygen of the air within the obstructed sinuses is absorbed and rarefaction results.

FIG. 23

A compound deviation of the septum. The upper deviation is of the greater clinical importance, as it blocks the ventilation and drainage of the sinuses.

The blood of the lining mucous membrane is attracted to the parts by the negative pressure thus created, and catarrhal inflammation is promoted. If, in the course of events, active pus-producing microbes, such as the streptococci, staphylococci, diplococcus pneumoniæ, etc., find lodgement there, a suppurative inflammation of the sinuses results.

It is obvious that this type of deviation is of the greatest importance and that the indications for its removal are urgent.

(*c*) The combined deviation, including the ridge along the crest of the vomer and the convexity of the perpendicular plate of the ethmoid bone (Fig. 23), is a very common type of septal deformity, and often calls for correction at the hands of a surgeon. The indications for operative interference are given under (*a*) and (*b*) of Osseous Deviations, and need not be further discussed here. The indications are obviously more urgent than in either the simple ridge or the convex perpendicular plate of the ethmoid, as the ill effects of both deviations are to be reckoned

with. It should be noted that the convexity of the perpendicular plate of the ethmoid is usually on the side opposite to the ridge along the crest of the vomer, though it may be on the same side. It should also be noted that the cartilaginous portion of the septum is deviated with the perpendicular plate of the ethmoid, and should, of course, be included in the operative field.

(*d*) There are still other deformities of the osseous septum, as the so-called spurs on the anterior portion, which in reality are composed of the crista nasalis and cartilage in combination, though they may be true osteomata.

THE COMPLICATIONS AND SEQUELS OF OBSTRUCTIVE MALFORMATIONS OF THE SEPTUM

A review of the preceding paragraphs naturally leads to the conclusion that many of the catarrhal and suppurative inflammations of the nasal and accessory sinuses are often due either directly or indirectly to obstructive malformations of the septum.

The whole truth is not expressed in the above statement; nevertheless, the deduction is fundamental and should form the working basis in a large majority of cases. The etiology of the inflammatory diseases of the nose and accessory sinuses is given in Chapter VI.

The following morbid conditions within the nose and accessory sinuses are either directly or indirectly caused, or their course is often largely influenced, by a preëxisting deviation of the septum:

1. Acute rhinitis or coryza.
2. Chronic turgescent rhinitis.
3. Chronic hypertrophic rhinitis.
4. Chronic hyperplastic rhinitis.
5. Acute sinuitis, catarrhal and suppurative.
6. Chronic sinuitis, catarrhal and suppurative.
7. Polypoid degeneration of the mucosa of the nose and sinuses.
8. Atrophic rhinitis.

It is apparent, therefore, that deviations of the nasal septum should be a primary rather than a secondary subject in a systematic text-book on diseases of the nose. They are, therefore, herein discussed before taking up the consideration of the inflammatory diseases which are so largely dependent upon them.

Indications.—The indications for the correction, or the removal, of obstructive deviations of the septum are based upon the following considerations:

1. If the deviation of the septum does not interfere with (*a*) the functional activity of the "swell bodies" of the inferior turbinates, (*b*) the ventilation of the middle and superior meatuses and the accessory sinuses, and (*c*) the drainage of the same areas it should not be subjected to surgical treatment. In other words, deviations of the septum should never be corrected simply because they are departures from the median

line of the nose, but only when they obstruct ventilation and drainage, or interfere with the function of the "swell bodies."

2. The positive indications for the correction of deviated septa are present when the septum (*a*) interferes with the normal functional activity of the "swell bodies," or (*b*) prevents the normal ventilation and (*c*) drainage of the nasal chambers and accessory sinuses.

If, for instance, a ridge along the crest of the vomer is so prominent as to touch the inferior turbinate, or if it extends forward into the vestibule far enough to partially obstruct the inspiratory current of air, and thereby produces rarefaction of the air posterior to the obstruction, it should be removed. The same is true in reference to anterior angular deflections of the cartilaginous septum.

If the deviation is higher up, in the region of the middle turbinate, and interferes with the ventilation of the superior meatus and the accessory sinuses draining into it, it should be corrected.

If a septum is tested by the foregoing standards, with a negative result, it should not be subjected to surgical correction, no matter how great the deviation or deviations may be.

If, on the contrary, a septum is tested by the foregoing standards, with a positive result, it should be corrected by some surgical procedure.

THE SYMPTOMS OF DEVIATIONS OF THE SEPTUM

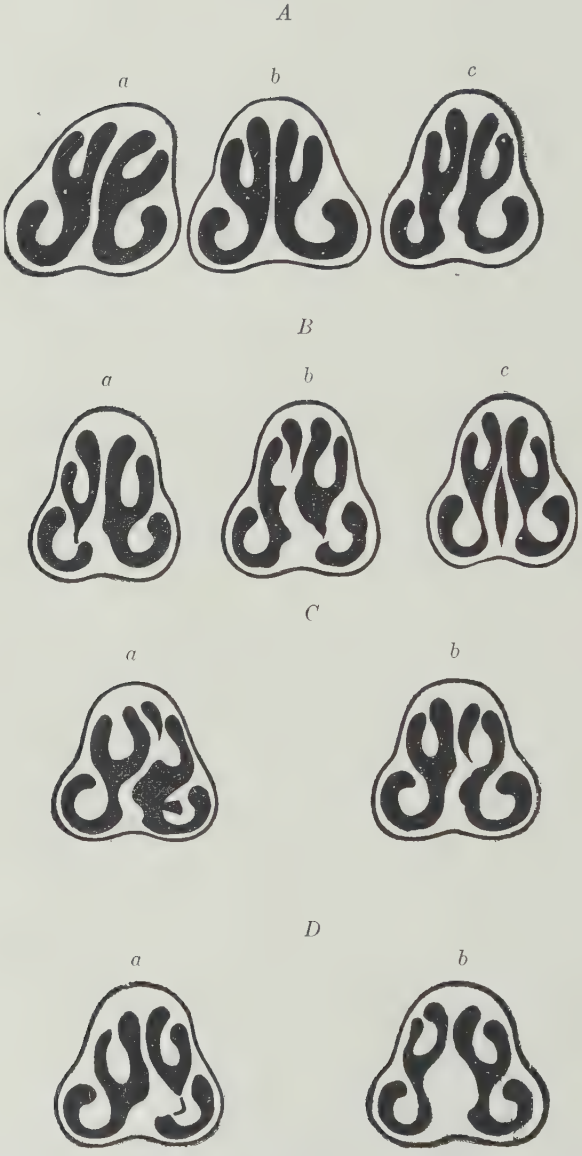
The Subjective Symptoms of Obstructed Deviations.—The subjective symptoms of nasal obstructions are (*a*) a sense of fulness, either in the lower or upper portion of the nasal chambers, according to the location of the deviation. If, for instance, the deviation impinges upon the "swell body" of the inferior turbinate there is a sense of stuffiness or fulness in the lower portion of the nose; whereas if it is in the region of the middle turbinate there is a sense of stuffiness or pressure through the bridge of the nose between the eyes.

(*b*) If the obstruction in the region of the middle turbinate is great enough, or has given rise to a catarrhal inflammation in the anterior ethmoidal cells, there may be pain, upon pressure, at the inner angle of the orbit under the floor of the frontal sinuses. When pain is elicited upon pressure in this region, it is very significant of anterior ethmoidal inflammation, and possibly of the frontal sinus as well.

(*c*) Frontal headache is frequently present in high deviations, and is most severe in the morning upon awakening. If of ocular origin it subsides at night and recurs during the day while using the eyes.

(*d*) Dizziness or vertigo is sometimes a direct expression of inflammation or irritation in the ethmoidal and the frontal sinuses. The dizziness is often exaggerated, or is produced by stooping forward or suddenly rising from the stooping posture, and is present when the eyes are closed. Dizziness or vertigo of ocular origin is often relieved when the eyes are closed, as the irritation from the light is thereby eliminated. Dizziness of nasal origin is aggravated by jarring the body.

FIG. 24



A. Types of non-obstructive septa: *a*, deviated from the median line; *b*, normal straight septum in the median line; *c*, deviation of the lower portion of the septum, with a concavity in the left nasal chamber, but with compensatory hypertrophy of the left inferior turbinated body.

B. Types of obstructive septa: *a*, ridge pressing against the inferior turbinate; *b*, ridge pressing against the left inferior turbinate and a convexity higher up on the right side obstructing the olfactory fissure on that side; *c*, a split septum causing double obstructive convexity of the septum.

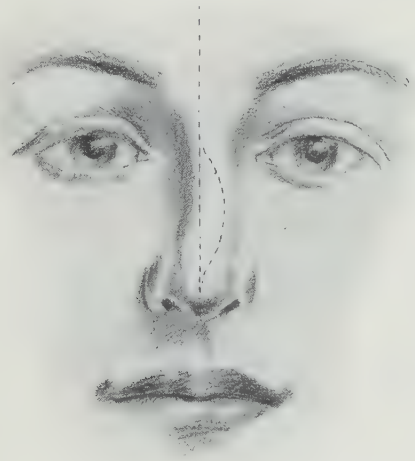
C. *a*, an S-shaped septum causing obstruction in the inferior portion of the nasal chamber on the right side and the superior portion of the chamber on the left side; *b*, a high, angular deviation of the septum causing obstruction of the olfactory fissure of the left side.

D. *a*, marked deviation of the septum along the crest, the vomer wedged firmly against the left inferior turbinate; *b*, abscess or hematoma of the septum obstructing both nasal chambers.

(*e*) Asthma of reflex nasal origin is sometimes due to intranasal pressure and irritation in the middle turbinate and ethmoidal regions. This is particularly true when polypi are present.

(*f*) The nasal secretions are changed in character and quantity. If a chronic catarrhal inflammation of the lower portion of the nasal mucous membrane is present the secretions are heavier than normal, and expulsion is only accomplished by blowing the nose. If the obstruction is in the middle turbinal and ethmoidal regions and a simple inflammation is present in the ethmoidal cells the secretion is sometimes watery in consistency, though it may be mucoid and quite acrid in character. Associated signs of this type of secretion are the reddened and irritated appearance of the mucosa and a fissure or eczematous eruption of the margins of the nostrils and the upper lip.

FIG. 25



A traumatic deformity of the external nose and of the septum. The straight dotted line indicates the median line of the nose while the curved one indicates the deviation of the septum.

(*g*) Postnasal or epipharyngeal "dropping" is usually present. The olfactory fissure may be obstructed, and, as the closure prevents drainage through the fissure, the secretions flow backward over the middle turbinal into the epipharynx.

(*h*) Intermittent stenosis is usually present in those cases in which there is an anterior deviation which does not completely block the nasal passage. The obstruction interferes with the intake of air, and the descent of the diaphragm acts as the piston valve of a syringe and produces rarefaction of the air in the nasal chamber posterior to the obstruction. This in turn develops turgescence of the erectile tissue and a temporary stenosis.

(*i*) Alternating stenosis is another sign of an obstructive lesion in the lower portion of the nasal chambers and is due to the same causes given

in the preceding paragraph. The associated disease is usually turgescent rhinitis.

The Objective Symptoms of Obstructive Deviations.—(*a*) The appearance of the septum and its relation to the various aspects of the outer walls of the nose constitute the most important objective symptom. For example, if the septum is characterized by a ridge on the left side opposite the inferior turbinate and by a convexity in the region of the middle turbinate on the right side, an examination shows the deviations and the impingement of the same against the inferior turbinate on the left side and the middle turbinate on the right side (Fig. 24, *B*, *b*). Each case should be carefully examined with reference to the equal distribution of space in the respiratory tract of the nose and with reference to its adequacy for physiological purposes. The various types of deviation, of course, present different pictures upon examination, each having its peculiar clinical significance in proportion to the degree of obstruction caused by it, and in particular to its proximity to the middle turbinated body.

(*b*) The presence of pus and dried secretions in the olfactory fissure between the deviation of the septum and the middle turbinate is suggestive of the causative relationship of the deviation to the diseased posterior ethmoidal sinuses, from which the secretions in all probability flow.

(*c*) Hemorrhage or epistaxis is often a sign of a deviated septum, more particularly in its lower and anterior portions. A prominent crest projecting into the breathway is subjected to an undue exposure to the air current and the secretions become dried and adherent to it. When the crust is detached, either by blowing or picking the nose, the epithelium is torn from the mucous membrane and hemorrhage results.

(*d*) External deformity of the nose is often indicative of a corresponding deviation of the septum (Fig. 25).

CHAPTER V

THE CHOICE OF SEPTUM OPERATIONS. THE SURGICAL CORRECTION OF OBSTRUCTIVE MALFORMATIONS OF THE SEPTUM

There is no one method of correcting obstructive deviations or malformations of the septum nasi. The submucous resection of the septum is the most nearly universally applicable, though there are some deviations in which it can be used with great difficulty, whereas another method of surgical procedure may be easily and successfully used. Under such conditions poor judgment would be shown in selecting the submucous operation. In choosing a surgical procedure a method should be adopted that will remove the obstructive lesion of the septum with the most simple technique and the least risk to the integrity of the nasal septum. The object of the operation should be to establish free drainage and ventilation of the nasal chambers and of the accessory nasal sinuses (see Etiology of the Inflammatory Diseases of the Nose and Accessory Sinuses), rather than to exploit one method of operating over another. It will be my endeavor, therefore, to give some general rules to guide the surgeon in the proper selection of an operation for the correction or removal of obstructive lesions of the nasal septum.

Cartilaginous Deviations.—When the deviation is limited to the septal cartilage other operations than the submucous resection may often be chosen to correct it; indeed, they may often be chosen in preference to the submucous resection. An extreme angular deviation of the septal cartilage (Fig. 36) is rather difficult to correct by the submucous method, and is easily corrected by the Sluder operation (Figs. 35, 36, and 37). The Sluder operation is practically limited to extreme angular deviations of the cartilaginous septum, as stated by its author.

A cup-shaped deviation may be corrected by the Asch, the Gleason, the Watson, the Price-Brown, or the submucous resection operation. The simpler of these procedures are the Watson, the Gleason, and the Price-Brown operations, and of these the Watson is, perhaps, the more simple. The choice of operation will largely depend upon the location of the cup-shaped deviation and the thickness of the cartilage surrounding it. If, for example, the cartilage anterior to the deviation is extremely thin, or has become fibrous from antecedent chondritis, the triangular flap of the Watson operation will not engage against the opposing incised cartilage. If, on the other hand, the cartilage anterior to the cup is of the usual thickness and texture the Watson operation may be used with excellent effect. The cup deviation may also be corrected by the Gleason operation if the cartilage below the cup is firm and of the usual thickness. The H-incision of Price-Brown is also well adapted to this type of deviation. The perpendicular incision should be made, one anterior and

the other posterior to the cup, and the intersecting horizontal incision through the centre of the cup.

Compound or S-shaped deviations or compound angular deviations of the septal cartilage are peculiarly well adapted to the Kyle operation, provided the convexities are thickened. The redundancy of cartilage may be removed with the V-shaped file saws at the crest of each convex surface, thus permitting the septum to be forced to an upright position in the median line. This type of deviation is also easily corrected in the submucous operation by the author's method with the swivel knife, and is perhaps more fully and surely thus corrected. In this type of deviation there is usually little difficulty in elevating the mucoperichondrium, after which the cartilage is readily encircled with the swivel knife and removal *en masse* with dressing forceps.

Simple angular (anteroposterior) deviations and L-shaped angular deviations of the septal cartilage are usually very successfully corrected by the Watson operation (Figs. 33 and 34), though they are equally well adapted to the submucous resection operation with the swivel knife.

The deviated portion of the cartilaginous septum may be readily removed by submucous resection in practically all types of deviations except the extreme angular type, and even this may be thus removed. It is often preferable, however, to use one of the other methods of operating, as they are simpler and almost, if not quite, as satisfactory in their results. When, however, the obstructive deviation also involves the bony portion of the septum, it is often expedient to adopt a method of operating that will be equally applicable to both the cartilaginous and bony deviations. Obstructive deviations usually involve both the cartilaginous and osseous framework of the septum, hence the indications given above are not unqualifiedly applicable, except in deviations limited to the cartilaginous portion of the septum. One of the chief objections to the operations other than the submucous resection is the necessity of wearing a dressing or splint in the nose for two or more weeks. This alone should often influence the surgeon to elect the submucous operation.

Osseous Deviations.—As osseous deviations of the septum are nearly always associated with one or the other of the types of cartilaginous deviations already referred to, a method of operating should be adopted that will successfully remove both the cartilaginous and the bony deviations. The operation most universally applicable is the submucous resection. There are, however, important exceptions to this rule, notably a simple spur or ridge, unattended by other deviations of the septum in which the obstructive lesions may be removed by Bosworth's method with a saw. When the deviation consists of a deflection of the vomer to one side, it may be corrected by grasping it with the Asch septum forceps and freely fracturing it at the floor of the nose and introducing a nasal splint for a few days to hold it in its new position. Another important exception is a deviation limited to the perpendicular plate of the ethmoid, which may be successfully reduced with Roe's forceps.

1. *A simple spur or ridge* may be successfully removed with a saw or spokeshave, with less risk to the integrity of the septum than it can

by submucous resection. If, however, the spur or ridge is accompanied by a deviation of the cartilage or the perpendicular plate of the ethmoid, it may be necessary to adopt some other method of procedure.

2. *Spurs or Ridges Associated with a Cartilaginous Deviation.*—These types of compound deviation may be effectively corrected by first removing the ridge with a saw or spokeshave, and subsequently correcting the cartilaginous deflection by one of the methods described under cartilaginous deviations; or both may be removed at one time by the submucous resection operation.

3. *Spurs and Ridges Associated with an Obstructive Deviation of the Perpendicular Plate of the Ethmoid.*—These types of compound osseous deviations may also be corrected by two operations, or by a single operation. The ridge or spur may be removed with a saw or spokeshave at one time and the deviation of the perpendicular plate of the ethmoid corrected at a subsequent time with Roe's crushing forceps. The submucous resection operation is usually preferable, as the operation is completed at one sitting, and the results obtained are usually much better than by the two operations.

4. *A Simple Deviation Limited to the Perpendicular Plate of the Ethmoid.*—Two operative procedures are applicable to this type of deviation, one the Roe operation and the other the submucous resection operation.

As generally practised, the submucous resection operation sacrifices more or less of the cartilage whether it is deviated or not. This is done to expose the bony parts to operative interference. I have, in a few cases, in which the deviation was limited to the perpendicular plate of the ethmoid, made the incision just anterior to the union of the cartilage and perpendicular plate of the ethmoid, elevating the mucoperiosteum over the ethmoid plate on the side of the incision, then extending the incision through the cartilage and elevating the mucoperiosteum on the opposite side of the plate, as is done when the Killian incision is made.

Principles.—The principles which should guide the operator in selecting an operation other than the submucous resection are the following:

(*a*) Never choose an operation which requires the prolonged (more than four days) use of an intranasal splint or tampon. The operations requiring the prolonged use of a nasal splint or tampon are the Rach and the Kyle operations, as the flaps are not self-supporting; that is, the principle of a bevelled edge, or extensive overlapping flaps with union by adhesions, cannot be utilized in these operations.

(*b*) Operations utilizing bevelled-edged flaps do not require prolonged use of splints or tampons; hence, such operations may be chosen in selected cartilaginous deviations. The operations utilizing bevelled-edged flaps are the Watson, the Gleason, and the Price-Brown operations.

(*c*) Operations utilizing overlapping flaps with subsequent adhesion along the overlapping surfaces may be chosen in extreme angular deviation of the cartilaginous portion of the septum. The Sluder operation is such an operation.

(*d*) Operations in which the bony portion of the septum may be fractured or comminuted and reset in any desired position may be performed in selected cases, in which only the bony portion of the septum is deviated. Roe's crushing operation may be selected when only the perpendicular plate of the ethmoid bone is deviated. The author's method of fracturing the vomer may be chosen when only the vomer is deviated. These operations do not require the prolonged use of intranasal splints, as bony tissue remains in position without support.

When the foregoing principles cannot be applied, the submucous operation should be used.

THE SURGICAL CORRECTION OF OBSTRUCTIVE LESIONS OF THE NASAL SEPTUM

Having first determined that the deviation is an obstructive one (see Indications), the surgeon should next elect the procedure that will afford the greatest amount of correction with the least shock and inconvenience to the patient. The type of deviation will have much to do with the choice of the operative procedure. No hard-and-fast rules can be laid down as to the choice of operation, each case being somewhat different from all others.

The following operative methods will, however, with slight variations in technique, meet nearly all the indications for the surgical correction of the various types of septal deviations.

1. **Soft Hypertrophies of the Septum.**—Soft hypertrophies of the mucous membrane of the septum occur at two points, namely: (*a*) At the anterior portion just opposite to or below the inferior margin of the middle turbinated body, and (*b*) at the posterior end of the vomer. In the first instance the enlargement closes the anterior end of the olfactory fissure and interferes with the proper ventilation of the superior meatus and the sinuses draining into it. Its reduction is best accomplished as follows:

First, induce local anesthesia with a 5 to 10 per cent. solution of cocaine applied to the parts with a thin pledget of cotton.

Second, make one or two linear incisions through the hypertrophied tissue with the actual cautery at a bright cherry red heat (Fig. 26).

This procedure may be repeated two weeks later if the first application was insufficient to reduce the mass.

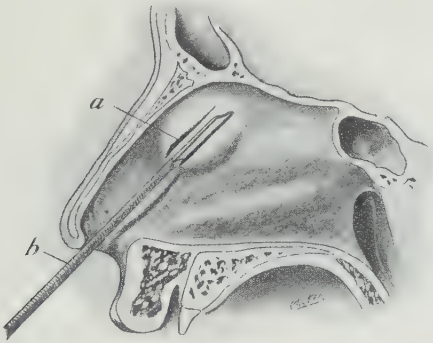
In posterior hypertrophy of the septum the same procedure may be followed, having first reduced the engorgement of the turbinated bodies with a spray of 1 to 2000 solution of adrenalin.

2. **Bosworth's Operation.**—When the septum is normally placed, with the exception of a spur or ridge, the obstructive lesion may be removed with a nasal saw (Fig. 27). If the deviation is a pronounced one, it may be preferable to resort to the submucous resection operation, as all other deflections can be removed by it at one time.

The technique of the saw operation is as follows:

(*a*) Induce local anesthesia over the spur or ridge by the application of pledgets of cotton saturated with a 5 per cent. solution of cocaine. After ten minutes remove the cotton, as anesthesia is usually complete in this time.

FIG. 26

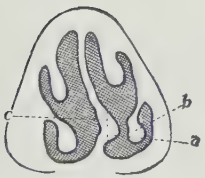


The reduction of an anterior hypertrophy of the mucous membrane of the septum in the region of the anterior end of the middle turbinate: *a*, linear cauterization; *b*, cautery electrode making a second linear incision.

FIG. 27

Bosworth's saw.

FIG. 28



a, ridge or deformity of the septum; *b*, the inferior turbinate encroached upon by the deviation; *c*, line of incision to be followed in removing the ridge with a saw.

FIG. 29

Showing the method of applying the saw to remove ridges from the septum.

(*b*) Introduce the nasal saw beneath the ridge or spur with its cutting edge turned inward and upward, as though it were the intention to saw obliquely through the septum (Figs. 28 and 29).

(*c*) After the saw is engaged in the bony tissue direct it upward (Fig. 29), parallel with the surface of the septum, until the ridge or spur is completely severed from it.

It is not necessary to make a preliminary incision along the crest of the ridge or spur for the purpose of elevating the mucoperiosteum, as experience has shown that healing takes place quite as quickly and satisfactorily when the mucoperiosteum is removed with the bone. Healing takes place by granulation and the periosteum is extended by the same process of repair over the sawn surface. In a number of cases thus operated on, and subsequently operated upon by the submucous method, I have had little difficulty in elevating the mucoperiosteum over the old field of operation.

The postoperative dressings should be omitted altogether unless the method described by Dr. Pischel is adopted. He first secures absolute dryness of the wound, and then applies a thin pledget of cotton over the surface and saturates it with an ethereal solution of collodion by means of a pipette, and allows it to dry in place. The wound is thus hermetically sealed with the collodion film, which protects it from the nasal secretions. The collodion dressing should be left in position until it is voluntarily thrown off, which usually occurs in three or four days. Subsequent dressings are not required.

FIG. 30

Chaleway's spokeshave.

3. **The Removal of Spurs and Ridges with the Spokeshave.**—The spokeshave may be used instead of the saw, though it is attended by more risk to the integrity of the septum and shock to the patient.

The technique is as follows:

(*a*) Local anesthesia.

(*b*) Make an elliptical incision around the base of the spur or ridge so as to prevent tearing the mucous membrane with the spokeshave (Fig. 31).

(*c*) Introduce the spokeshave (Fig. 30) into the nostril until its blade engages the posterior end of the ridge, and then pull it forward with considerable force, again and again if necessary, until it splinters the ridge from the septum (Fig. 32). The elliptical incision previously made saves the mucous membrane from mutilation.

(*d*) The dressing may be omitted or the collodion dressing may be used.

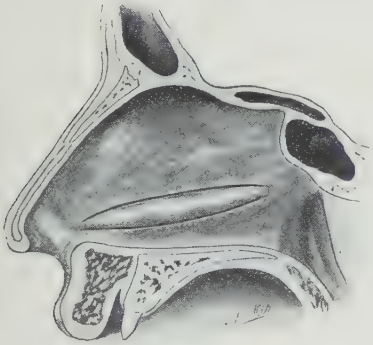
Caution.—So much force is usually required to engage the spokeshave that there is danger of fracturing the cribriform plate and causing meningitis.

Another accident which should be taken into consideration is perforation of the septum. It is not possible to exercise full control over the course of the spokeshave, as it does not cut through the tissue (bony) but acts as a wedge. I have sometimes resorted to a procedure which in a measure controls the direction of splintering, as follows:

After making the elliptical incision, grooves are made with a saw at the

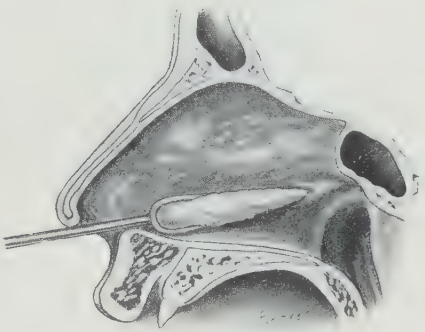
base of the ridge on its upper and lower aspects. The grooves guide the spokeshave as it comes forward through the bone, and thus prevents cutting too deeply into the tissue. The grooves weaken the attachment of the ridge and render its removal possible with less force.

FIG. 31



Incisions above and below the ridge.

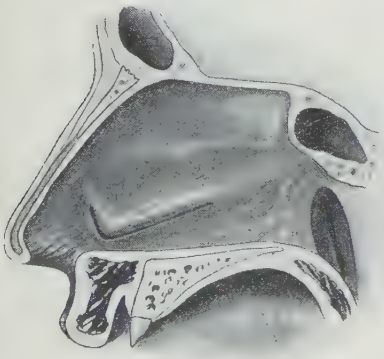
FIG. 32



Removal of ridge with the spokeshave.

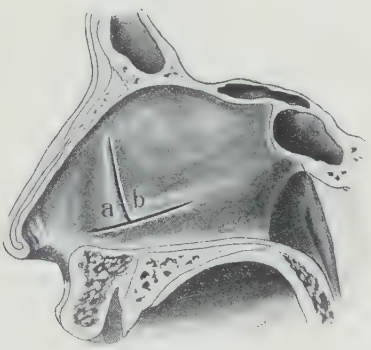
The Watson Operation.—The Watson operation consists in making one or more incisions through the septum and then pushing the projecting or deviated bevelled portion toward the concave side, the bevelled edges formed by the incision retaining the septal flap in its new position.

FIG. 33



The Watson operation for correcting a simple angular deviation of the cartilaginous septum.

FIG. 34



The Watson operation for a combined horizontal and perpendicular bowing of the nasal septum.

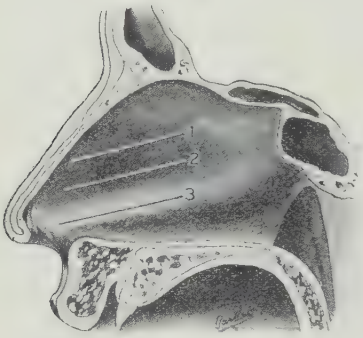
Technique.—(*a*) Local anesthesia.

(*b*) Make the incision or incisions with a short-bladed bistoury.

(*c*) Introduce the index finger or a broad, blunt instrument into the nose on the side of the septal convexity and force the deviated flap to the

opposite side. If the single incision is made (Fig. 33), force the angular flap to the opposite side along the entire line of incision. If the double incision (Fig. 34) is made, first force the anterior triangular flap (*a*) to the concave side and then force the posterior triangular flap (*b*) to the concave side. The bevelled edges formed in making the incision help to hold the flaps in the new position.

FIG. 35



Sluder's septum operation: 1. 2, and 3, the lines of incision.

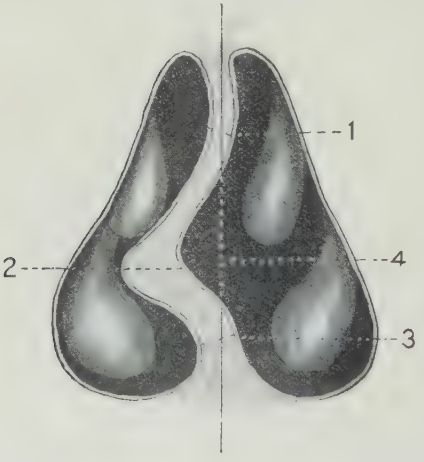
(*d*) Additional support should be given to the flaps by a tampon on the side of the convexity or by a septum tube splint for a period of three or four days.

Sluder's Operation.—Dr. Greenfield Sluder has used a modification of the Watson operation, with excellent results, and he especially recommends it in children with extreme angular cartilaginous deflections.

Technique.—(*a*) Cocaine anesthesia.

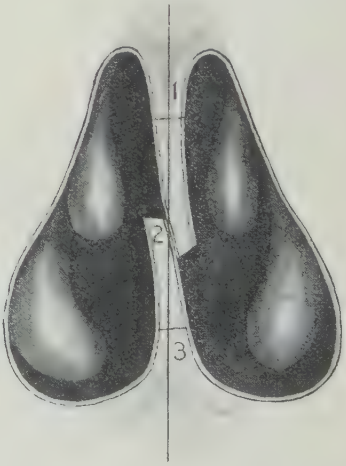
(*b*) Make three parallel incisions through the entire thickness of the septum parallel with the crest (Figs. 35 and 36). The middle incision

FIG. 36



Sectional view of the nose before the Sluder operation: 1, 2, 3, the lines of incision shown in Fig. 35; 4, the median line of the nose.

FIG. 37



Sectional view of the nose after the Sluder operation: 1, 2, 3, the lines of incision as shown in Fig. 35. The bands of cartilage overlap, and should be held in position with a nasal tube.

should extend the whole length of the crest. The other incisions are made at the apices of the less acute angles 1 and 2. Two strips of

cartilage are thus formed, their only attachments being at the anterior and posterior extremities.

(*c*) Either the upper or lower strip is then forced to the concave side with the index finger or a blunt instrument.

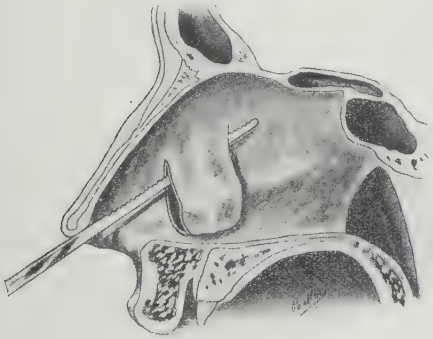
(*d*) The other strip is likewise displaced to the concave side, thus causing them to overlap, as shown in Fig. 37.

(*e*) A Mayer nasal tube is then introduced on the side of convexity to hold the strips in position while union takes place, a period of three or four days.

If the opposed surfaces are curetted before coaptation, union will take place more rapidly. Dr. Sluder reports 24 cases, 5 in adults and 19 in children, without perforation of the septum, all of which were cases of extreme deflections.

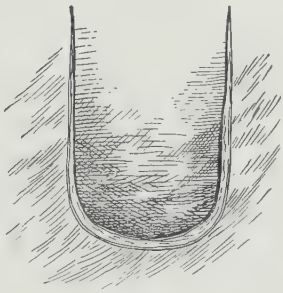
4. **The Gleason Operation.**—The election of this operation may be made when the septum is bowed or cup-shaped, and without a heavy ridge along the crest of the vomer. It consists essentially of a U-shaped incision extending either entirely through the septum and both its mucous coverings, or only through the mucous membrane of one side and the bone and cartilage. The incision may be made with a saw (Fig. 38) or with a knife.

FIG. 38



The Gleason operation. A tongue-flap of the deviated portion of the septum.

FIG. 39



Gleason's tongue-flap pushed through the window.

The Technique.—(*a*) Local anesthesia is induced with a 5 to 10 per cent. solution of cocaine applied to the mucous membrane on both sides of the septum.

(*b*) The nasal saw is applied on the convex side of the septum at its inferior portion, and the incision is carried through the septum in an upward direction, the ends of the saw remaining upon the side of convexity while its middle portion passes through to the concave or opposite side. A U-shaped incision is thus made with a bevelled tongue-flap suspended between the limbs of the U (Figs 38 and 39).

On account of the low position of the nasal orifice the anterior limb of the incision is usually too short. This is obviated by removing the saw and reinserting it through the anterior limb alone and continuing

the incision upward, or it may be extended with a knife, as the framework of the septum is cartilaginous in this region.

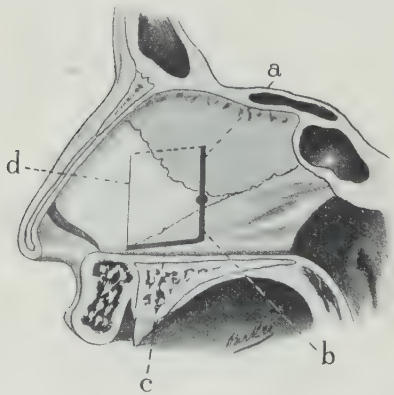
If it is not desired to extend the incision through the mucous membrane on the concave side the saw should be directed upward parallel with the septal surface on the concave side just beneath the mucous membrane. This is not at all difficult, as the mucoperichondrium and periosteum usually separate very readily from the cartilage and bone. Or the membrane may first be elevated on the concave side by the injection of normal salt solution beneath the mucoperichondrium and periosteum, thus lifting it away from the cartilage and bone.

Fig. 40

a, sectional view of the septum after the Gleason operation.

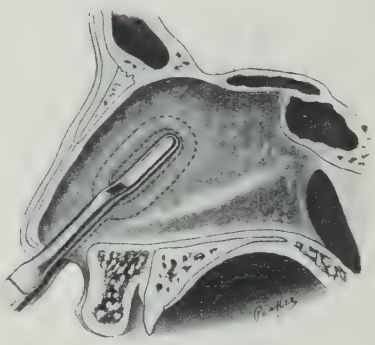
(*c*) Having made the U-shaped incision, the tongue-flap should be forced from the convex side through to the concave side with the finger inserted into the nostril. The bevelled edges of the flap and those of the fixed portion of the septum engage so as to hold it in its new position on the concave side (Fig. 40). The tongue-flap has a tendency to spring back into its former position, owing to the elasticity of the cartilaginous and bony tissue contained in it, hence it is necessary to overcome its resiliency by forcing it as far to the concave side as possible, the flap being thus fractured at its upper extremity.

Fig. 41



The Gleason operation, including the quadrilateral cartilage, the perpendicular plate of the ethmoid, and the vomer. The incisions *a*, *b*, *c* are made with a nasal saw, and incision *d* with a knife. The saw is introduced at the junction of the vomer and perpendicular plate, as indicated by the swelling of the line *a*, *b*.

Fig. 42



The Roe operation.

By the foregoing procedure the convex portion of the septum is displaced toward the side of the greatest nasal space, and the obstructed side is opened for freer drainage and ventilation.

The Gleason tongue-flap may also be used when the deviation embraces both cartilage and bony tissues, as shown in Fig. 41, which illustrates a case operated by me with entire success. There was a prominent ridge on the left side of the septum corresponding with the crests of the crestanasalis and the vomer. The quadrilateral cartilage was also deviated to the left. The septum was perforated at the junction of the cartilage, perpendicular plate and vomer (dark spot). A nasal saw was inserted through this opening and the perpendicular plate and membranes cut upward. The vomer was then sawn to the floor of the nose. The saw was then directed anteriorly and the vomer severed at the floor. (The heavy line shows the area of the incision made with the saw, the light line that made with a knife.) A small bistoury was used to make the anterior limb of the U-shaped incision. The saw and knife should be inserted from the side which will permit the bevelled edges to hold the flap in position when pushed through the opening. As a large portion of the thickened crest is cartilaginous, it will atrophy after being pushed through the window to the opposite side. If this fact is not borne in mind, it may appear to the operator that the opposite nostril will be occluded, and the patient be left in as bad a condition as before the operation.

Dressings.—It may be necessary to insert a nasal tube (Fig. 47) on the side of convexity for a day or two to insure the fixation of the tongue-flap in its new position. Dressings are not otherwise needed.

5. **The Roe Operation.**—The Roe operation (Fig. 42) may be used to correct deviations of the perpendicular plate of the ethmoid bone, and it may also be used to correct the bowing of the septum in the region of the middle turbinal, where there is also a ridge on the lower portion of the septum, though it is not applicable for the correction of an obstruction due to a heavy ridge. Roe has devised special forceps, with a male and a female blade, for this operation.

Technique.—(*a*) Local anesthesia upon both sides of the septum, indeed of the whole nasal mucous membrane, is necessary; or the operation may be done under general anesthesia.

(*b*) The Roe forceps should be introduced, the male blade into the side of convexity and the female blade into the opposite side. By closing the forceps blades the convex portion of the septum is forced toward the opposite side through the opening of the female blade. The entire area of obstruction may be thus fractured and forced toward the concave side.

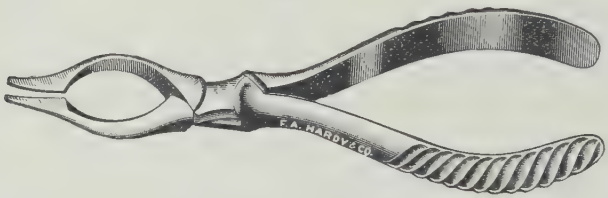
(*c*) The fractured portion of the septum should be held in its new position, with nasal splints, or with strips of bismuth gauze, for a few days, or until it becomes fixed in its new position. This operation is especially adapted to deviations of the perpendicular plate, which, being composed of bony tissue, remains in position after being fractured.

6. **The Asch-Mayer Operation.** This operation consists of a crucial incision through the cartilaginous portion of the septum, the four triangular flaps thus created being pushed toward the side of concavity and held in their new position with a Mayer nasal tube (Fig. 47). The opera-

tion may be used in curved or cup-shaped deviations of the cartilaginous septum. In other words, the Gleason, Watson, Sluder, Roe, and Asch-Mayer operations are suitable for much the same type of deviated septa. I have often included the deviated portion of the perpendicular plate of the ethmoid in the field of operation with good results, and see no objection to it, though the operation as originally devised by Dr. Asch was limited to the cartilaginous portion of the septum.

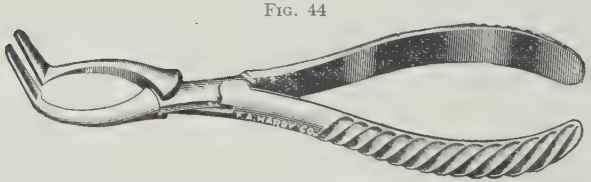
Technique.—(*a*) The operation may be performed under local anesthesia, though it is generally preferable to do it under general anesthesia, as the shock and pain are otherwise considerable.

FIG. 43



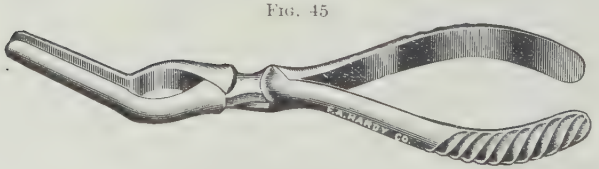
Asch's straight scissors.

FIG. 44



Asch's curved scissors.

FIG. 45



Asch's septum forceps.

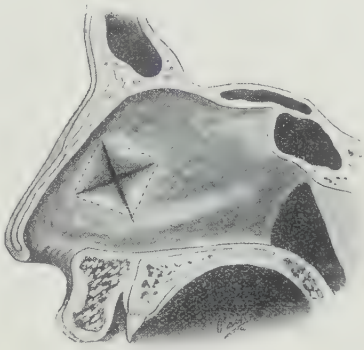
(*b*) After cleansing the nasal chambers and the face, the straight Asch scissors (button-hole) (Figs. 43, 44 and 45) should be introduced into the nostrils, the narrower blade into the side of convexity and the wider into the opposite, from three-eighths to one-half of an inch above the floor of the nose, and the septum cut through. The Asch angular scissors (Fig. 44) is then introduced and the perpendicular incision made, bisecting the middle of the horizontal one. Four triangular flaps are thus made (Fig. 46).

(*c*) The septum should next be seized with forceps (Fig. 45) and fractured by rotating it from side to side. It has been my practice to include the perpendicular plate of the ethmoidal bone in the grasp of the septum forceps, as it is nearly always deviated with the cartilaginous

portion. I have also included the remnants of the ridge left after the sawing operation, thus fracturing it (the vomer) from its attachment to the maxilla.

(*d*) The index finger is then inserted into the nostril on the side of septal convexity and the four triangular flaps pushed as far as possible to the opposite side (Fig. 46), care being exercised to fracture the flaps at their uncut bases. If this is not done the resiliency of the cartilage gradually brings them back to their original position.

Fig. 46

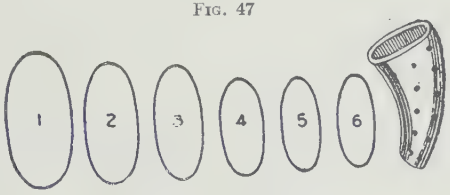


The Asch operation. The crucial incision is made through the deviated portion of the quadrilateral cartilage of the septum, thus forming four non-bevelled triangular flaps. The flaps are then pushed forcibly to the convex side of the septum and fractured at their bases, as shown by the dotted lines. This is done to overcome the resiliency of the cartilage.

(*e*) Severe hemorrhage usually occurs, but it may be quickly checked by the introduction of the Mayer nasal tubes. The tubes are primarily used, however, for the purpose of holding the incised and fractured septum toward the concave side (Fig. 47). The tube selected for the convex side should be large enough to force the septum beyond the point it is desired to fix it, as it will swing back a little toward its old position in spite of all precautions. A smaller tube should be introduced into the opposite nostril to exert counter-pressure against the septum to check the hemorrhage.

After-treatment.—Both tubes should be left in position for two or three days and then removed. A tube one size smaller should then be introduced into the side of convexity, but none into the opposite side. The tubes should be worn for about six weeks, being removed and cleansed every alternate day during this period. Experience has shown that the septum gradually swings back to its former position if the tube is not worn for about this length of time.

Fig. 47



Mayer's nasal tube splints.

Objections.—(*a*) Perforation of the septum sometimes follows the operation. (*b*) The shock attending the operation is often great. (*c*) The inflammatory reaction is sometimes severe. (*d*) The presence of the tube in the nose for six weeks is a source of considerable annoyance. (*e*) The hemorrhage is occasionally severe and difficult to control.

7. **The Kyle Operation.** The Kyle operation may be used in simple and compound curvatures of the septum in which there is a redundancy of tissue along the lines of convexity. It consists in making V-shaped grooves in the septum along the lines of greatest convexity, the object being to remove tissue where it is redundant, so that the septum may be made straight without overlapping along the lines of incision.

Fig. 48

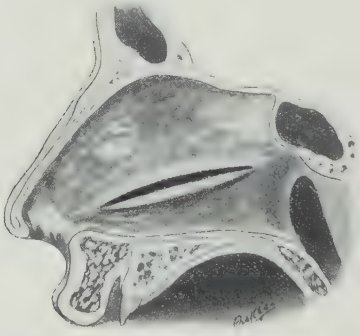
Fetterolf's file saw.

Technique. —(*a*) Local anesthesia of both sides of the septum should be induced.

(*b*) A linear incision with a small bistoury should be made along the lines of convexity.

(*c*) The Fetterolf V-shaped file saw (Fig. 48) should be used along the lines of incision until the thickness of the cartilage and bone are penetrated (Fig. 49).

Fig. 49



Kyle's operation. Side view of the septum after groove is made.

Fig. 50

a, sectional view of the septum after the V-shaped incision; *b*, Kyle's malleable tube holding the septum in position.

Fig. 51

Kyle's malleable tubes.

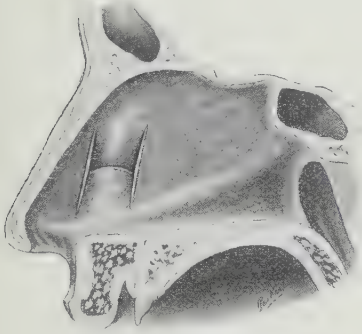
(*d*) The incised septum should then be forced into the median line by the introduction of Kyle's malleable tubes into either nasal chamber (Figs. 50 and 51). The tube being malleable, may be spread with

forceps introduced into its lumen until the septum is adjusted in the median line.

(*e*) The after-treatment consists in removing and reintroducing the tubes until all tendency of the tissues to return to their former position is overcome.

The Price-Brown Operation.—This operation consists of two parallel incisions united by an intersecting incision as shown in Fig. 52. The two rectangular flaps thus formed are pushed through to the side of the concavity and held in position for a few days with a nasal splint or dressing upon the side of the convexity. The operation is extremely simple, and is especially applicable to cup-shaped deviations of the cartilaginous portion of the septum. This operation is also applicable to simple perpendicular or horizontal angular deviations of the cartilaginous septum, the intersecting incision being made across the crest of the angular deviation, as shown in Fig. 52. The incision should be so made that the bevelled edges hold the flap in their new position as in the Watson operation.

FIG. 52



The Price-Brown operation. Two parallel incisions are made, one on either side of the long axis of the deviation. An intersecting incision is then made across the long axis of the deviation. All incisions are made with bevelled edges, so that when the two quadrilateral flaps are pushed to the concave side they will engage in the opening as in the Watson and the Gleason operations.

FIG. 53

The removal of the bony ridge of the septum, the preliminary step in Moure's operation, for the correction of deviations of the septum.

Moure's Operation.—Moure's method of straightening the septum is especially applicable to those cases in which there is a concavity on one side of the septum and a marked thickening or ridge of bone upon the opposite side (Fig. 54). This type of deviation is also well suited for the submucous operation.

Technique.—(*a*) Cocaine anesthesia.

(*b*) Remove the ridge with a spokeshave or saw as indicated by 2 in Fig. 54. The removal of this ridge of bone materially relieves the pres-

sure upon the middle (5) and inferior turbinated bodies (4). The septum may still crowd too much to the convex side, hence Moure advises the following procedure to force the remaining portion of the septum (3) to the opposite side:

(*c*) Having removed the ridge, two incisions are made, as shown in Fig. 55. One is made below the ridge (Fig. 56), and the other above and in front of it, parallel with the ridge of the nose. The incisions are made with specially devised scissors resembling those of Asch.

(*d*) A malleable metal splint is then inserted on the side of convexity and spread with forceps until the septum is sufficiently forced to the opposite side, as shown in Fig. 57. The two incisions permit the septum to be forced to the opposite side, where it should be held with the malleable splint until it becomes fixed in its new position.

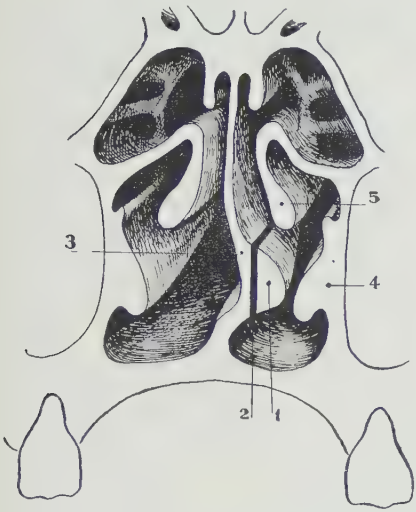
After-treatment.—The splint should be removed in three or four days, cleansed, and reinserted and moulded to the parts. This procedure should be repeated every two or three days for one week, or until firm union takes place. Should excessive granulations form they should be reduced with fused chromic acid crystals. The open skeleton tube used by Moure permits free respiration and nasal irrigation while it is in position.

THE SUBMUCOUS RESECTION OF THE SEPTUM

1. **Position of the Patient.**—The patient may be placed in either the sitting or the reclining posture. Most operators will probably prefer the sitting posture in an ordinary office chair (Figs. 4 and 5), though the reclining posture may become necessary if the patient faints either from psychical or cocaine depression. I have found, when the patient is thus overcome, that the reclining position gives immediate relief, and allows the operator to proceed with but slight loss of time. The back of the chair should be tipped almost to the horizontal position, and the head of the patient supported by a head-rest or by an assistant. When the patient is thus reclining the operator should sit by his right side, facing the patient. If the operator prefers to stand the patient may be placed upon an operating table.

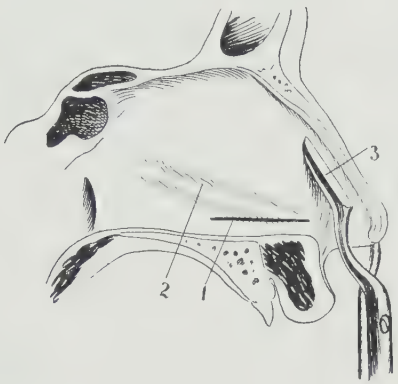
2. **Anesthesia.**—Cocaine anesthesia is preferable, though a general anesthetic may be administered. The method of applying the cocaine is important. Freer has called attention to this fact, and, in general, I follow his suggestion in reference to it. Pulverized cocaine is used instead of a solution. A delicate silver cotton-wound applicator is moistened in adrenalin solution, the excess squeezed from it, and then dipped into the powdered cocaine. The loose granules are then gently knocked off, and the mucous membrane of the entire septum on both sides is thoroughly massaged or rubbed with it. The membranes should be massaged for about three minutes. After an interval of five minutes they should be massaged again with a fresh preparation. Three applications usually induce complete anesthesia, though in rare instances numerous applications are required.

FIG. 54



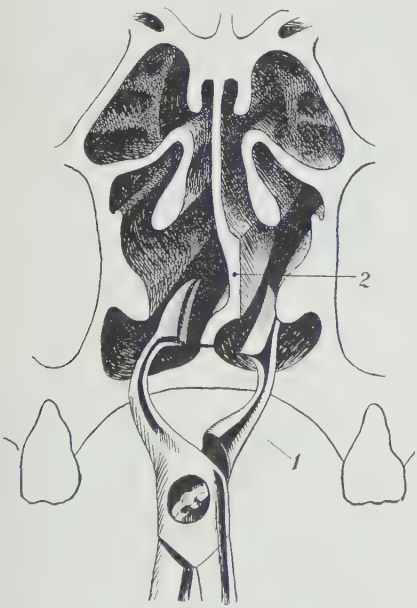
Cross-section of the nose, illustrating certain details of Moure's septum operation: 1, the ridge severed with the spokeshave; 2, the incision with the spokeshave; 3, the septum; 4, the inferior turbinate crowded upon by the ridge of the septum; 5, the middle turbinate also crowded upon by the deviated septum.

FIG. 55



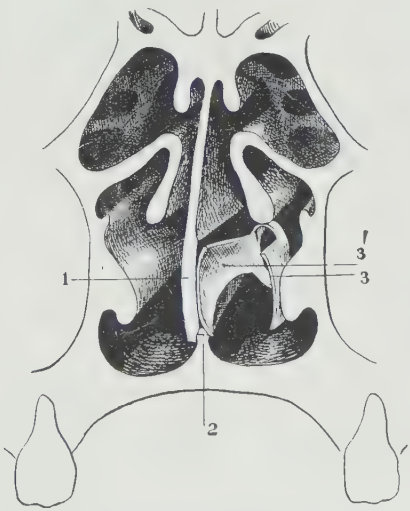
The incisions of the septum in Moure's operation: 1, the incision along the floor of the nose below the septal ridge; 2, the thickened septal ridge; 3, the upper incision through the septum being made with Moure's scissors.

FIG. 56



Making the incisions through the septum with Moure's scissors: 1, Moore's scissors; 2, the septum.

FIG. 57

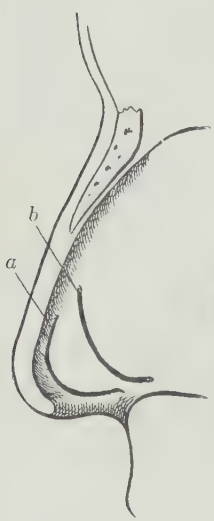


Moure's malleable splint in operation: 1, the septum displaced to the right side of the nose; 2, the incision made with Moure's septum scissors; 3, the outer wall of the nasal splint resting against the inferior turbinated body; 3′, the inner wall of Moure's nasal splint crowding the septum to the right side of the nose.

The advantages of this method of applying cocaine over the use of solutions are the speed with which anesthesia is induced and the comparative infrequency of cocaine toxemia. By this method little or no cocaine is swallowed, whereas when a solution is used a considerable amount may be swallowed and produce toxic symptoms.

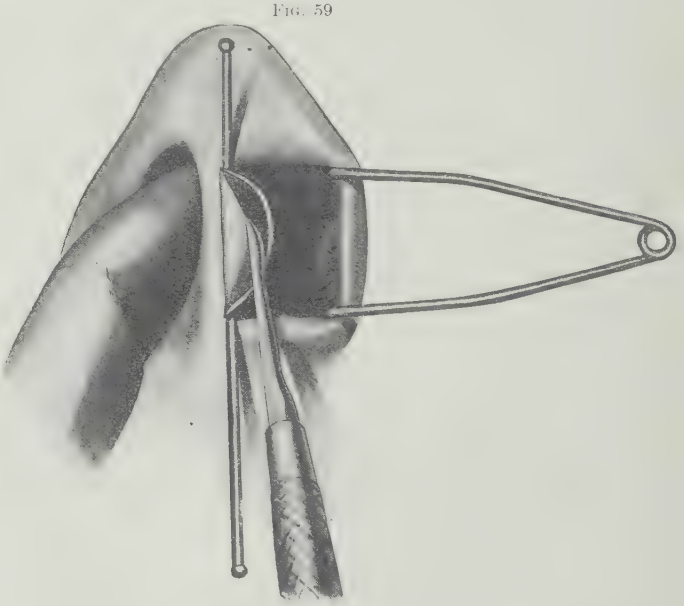
When Hajek's incision is made at the anterior end of the columnar cartilage (Fig. 58), Schleich's solution should be injected beneath the membrane, as the application of cocaine will not produce anesthesia. Furthermore, the membrane is very adherent in this region (vestibular portion of the septum) and is elevated with difficulty. The subcutaneous injection of the solution partially elevates the membrane and renders the completion of the elevation comparatively easy.

FIG. 58



Incisions for the submucous resection of the septum: *a*, the Hajek incision; *b*, the Killian incision.

FIG. 59



The elevation of the mucoperichondrium upon the side of the primary incision in the mucous membrane. The elevation is begun with a sharp or semisharp elevator and is completed with the blunt elevator.

3. **The Incision.**—The choice of the location of the incisions should depend upon the character and location of the septal deviation. If it extends into the vestibule of the nose, Hajek's incision should be made at the extreme anterior margin of the cartilage of the septum, as shown in Fig. 58, *a*. As the membrane of the vestibular portion of the septum is firmly attached to the fibrocartilage beneath it, this incision should only be made when the deflection is far enough forward to render it necessary to remove the anterior portion of the deflected cartilage.

When the deviation does not extend forward into the vestibule, Killian's

incision (Fig. 58, *b*) should be made at the junction of the vestibular membrane with the mucous membrane of the septum, as the mucoperichondrium elevates with comparative ease posterior to this point.

The Killian incision is usually preferable and should be made with a sharp-pointed knife upon the left side of the septum. All other writers have recommended that it be made upon the side of the convexity of the septum, as they believe this allows greater freedom of access in elevating the membrane over the region of convexity. I believe this to be ill advised, as most operators are more dextrous with their right hands. Furthermore, it is unnecessary, as the tip of the nose is flexible and may be turned to one side out of the way. Hence I recommend that the incision be made upon the left side of the septum except for left-handed or ambidextrous surgeons.

The tip of the index finger of the left hand should be introduced into the right nasal chamber to exert counterpressure while the incision is being made. The incision should only extend through the mucous membrane and perichondrium. If it is carried deeper it interferes with the elevation of these tissues.

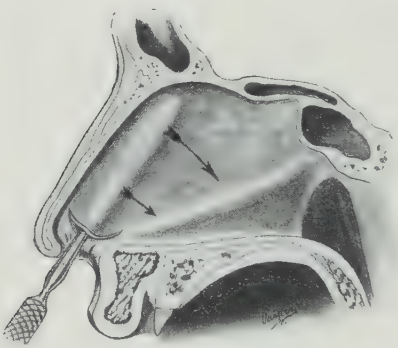
4. **The Elevation of the Mucoperichondrium and Periosteum.**- This step of the operation is often the beginning of either success or failure in the operation. If the elevation is properly done over the entire area of the deviation on both sides of the septum, the subsequent steps are comparatively easy to carry out. If, however, the elevation is not properly executed and extended over the entire field of the deviation, it may interfere with the remaining steps of the operation to such an extent as to defeat its purpose. Many of the younger rhinologists have told me of the difficulties they have encountered, and almost without exception they have failed to elevate over a large enough field. In the average case in which the cartilage, perpendicular plate of the ethmoid, and the vomer are involved in the deviation, the membrane should be elevated over almost the entire area of both sides of the septum. If, however, only the cartilage of the septum is affected, the elevation should be extended about one-half inch beyond the junction of the cartilage and the perpendicular plate, and down to the floor of the nose. Always elevate at least one-half inch beyond the area of the tissue to be removed, as otherwise the membrane may be injured in the process of removing the deviated portion of the framework of the septum.

The technique elevation of the mucoperichondrium may be accomplished in various ways. Some operators, notably Freer, prefer small, thin, sharp elevators with which the mucoperichondrium and periosteum are dissected from the framework of the septum. Curved elevators are also used to work around curved portions of the septum. Personally, I prefer heavy, broad and dull elevators, and I have never found it necessary to use curved elevators to get around a curved or an angular deviation. A study of the following descriptive technique will show how the heavy blunt elevators may be successfully used to encompass curved and angular deviations of the cartilage and the perpendicular plate of the ethmoid. The chief reason for using the blunt heavy elevators is

the greater speed and the lessened liability of tearing the membrane in the process of elevation.

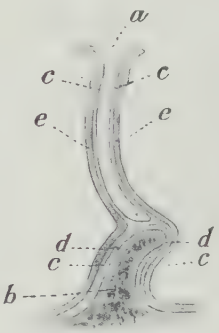
To start the elevation a sharp or semisharp elevator should be used (Fig. 78), care being exercised to get beneath the perichondrium. If the elevator penetrates between the mucous membrane and perichondrium, the surface of the cartilage will present a velvety red appearance as the perichondrium is still covering it. If, however, the elevator penetrates beneath the perichondrium the exposed cartilage presents a glistening white surface. Great patience is often required to start the elevation properly; this being done, the remaining elevation is comparatively easy. The point of least resistance is usually at the upper portion of the Killian incision, whereas at the lower portion the perichondrium is often so adherent as to require a knife to separate it from the cartilage.

Fig. 60



The Hajek elevator introduced beneath the mucoperichondrium along the line of least resistance. When thus introduced the elevation should be made in the whole shank of the instrument in a downward and backward direction to the crest of the vomer. The periosteum along the crest of the vomer should then be incised, as shown in Figs. 61, 62, 63, and 64.

Fig. 61



Section through the nasal septum: *a*, quadrilateral cartilage; *b*, vomer; *c*, *c*, agglutination of the perichondrium to the periosteum; *d*, *d*, periosteum reflected over the crest of the vomer (it is not continuous within the perichondrium); *e*, *e*, mucoperichondrium.

Having succeeded in starting the elevation (Fig. 59) abandon the sharp elevator and insert the blunt one (Fig. 78) into the small pocket already made. Direct the elevator parallel with the ridge of the nose, as this is the direction of least resistance (Fig. 60). Having introduced the elevator almost to the cribriform plate the elevation should be continued backward and downward with the whole length of the shank of the elevator within the pocket of the membrane. The mistake is usually made of attempting to elevate with the tip of the elevator, whereas it should be done with the shank. With the former it is easy to tear the mucoperichondrium, while with the latter the elevation may be rapidly accomplished with but little danger of tearing it. The principle involved is obvious, namely, a small tip will perforate more readily than a long shank. As a matter of fact, the mucoperichondrium and periosteum elevate readily under moderate tension with a broad dull

instrument, whereas if a small sharp elevator is used extreme care must be constantly exerted to avoid making a perforation.

After introducing the heavy blunt elevator as high as the cribriform plate (Fig. 60), exert pressure downward and backward with a twisting motion, and, as a rule, the membrane will strip down to the crest of the vomer in a few seconds, or at most in a minute or two. Five minutes or more may be required to start the elevation, whereas to complete it will require but a comparatively short time.

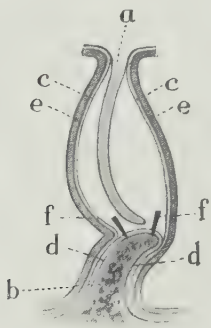
The question naturally arises, *How can the elevation be accomplished with the shank of the elevator when the cartilaginous or perpendicular plate portion of the septum is convex?* The operator should remember that these portions of the septum are thin and flexible. Being so, they may be forced with the elevator to the median line and thus temporarily rendered straight. While held in this straightened position the shank of the instrument is passed downward and backward, elevating the membrane as it proceeds. I have rarely observed a septum in more than 400 submucous operations that did not yield to this method of procedure. It may also be asked, *How can the elevation be accomplished with the tip of the straight, blunt elevator when there is a perpendicular deviation of the cartilage?*

The procedure is very simple. The tip of the nose is flexible, and the instrument should be held parallel with the anterior portion of the cartilage until it reaches the crest of the perpendicular deviation. The instrument should then be shifted until it is parallel with the cartilage posterior to the crest. The flexibility of the tip of the nose makes this possible, hence a curved elevator is not necessary for the purpose; or the crest may be forced to the concave side, thus rendering it straight and the elevation continued. My contention in favor of the use of blunt elevators (after the elevation is started) is based upon the conviction that the average operator will do less damage and will operate with greater speed than he will with small sharp elevators, or with small blunt ones. Otherwise, I have no objection to Freer's elevators, with which he, with infinite pains, accomplishes good results.

According to Neumann (J. R. Fletcher), the development of the periosteum of the septum nasi throws interesting light upon the technique of the submucous resection of the septum. He has found upon histological examinations of sections of the septum that the periosteum is not uniformly reflected over the bony portion of the septum. That only where bone unites with bone, as where the perpendicular plate of the ethmoid unites with the vomer, is the periosteum continuously spread over the septum; and that where the vomer unites with the cartilage of the septum, the periosteum is not continuous with the perichondrium of the cartilage. In the latter region the periosteum arises from the floor of the nose and passes upward over the lateral surface of the vomer to its crest, over which it is reflected, and then passes downward over the opposite lateral wall of the vomer to the floor of the nose. He also claims that the perichondrium is reflected over the periosteum in this region and that it is closely adherent to it (Figs. 61 and 62).

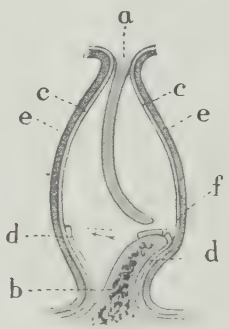
This arrangement of the periosteum and perichondrium explains the well-recognized difficulty experienced in elevating the periosteum below the crest of the anterior portion of the vomer when the elevation

FIG. 62



Elevation of the membranes of the cartilage and vomer: *a*, quadrilateral cartilage; *b*, vomer; *c*, *c*, perichondrium; *d*, *d*, periosteum of vomer with two incisions (*f*, *f*) at the crest; *e*, mucous membrane; *f*, *f*, two incisions through the periosteum along the crest of the vomer, to facilitate the elevation of the membranes anterior to the junction of the perpendicular plate of the ethmoid with the vomer.

FIG. 63



a, cartilage; *b*, vomer; *c*, *c*, perichondrium; *d*, *d*, periosteum of the vomer; *e*, *e*, mucous membrane; *f*, two incisions through the periosteum along the crest of the vomer. On the concave side the periosteum over the vomer is elevated.

is begun above it. I have long recognized this difficulty and attributed it to fibrous prolongations from the periosteum to the vomer, which, according to Carter, were due to the presence of the tuberculum or gland in this region. It appears, however, from the investigations of Neumann that this is not the true explanation.

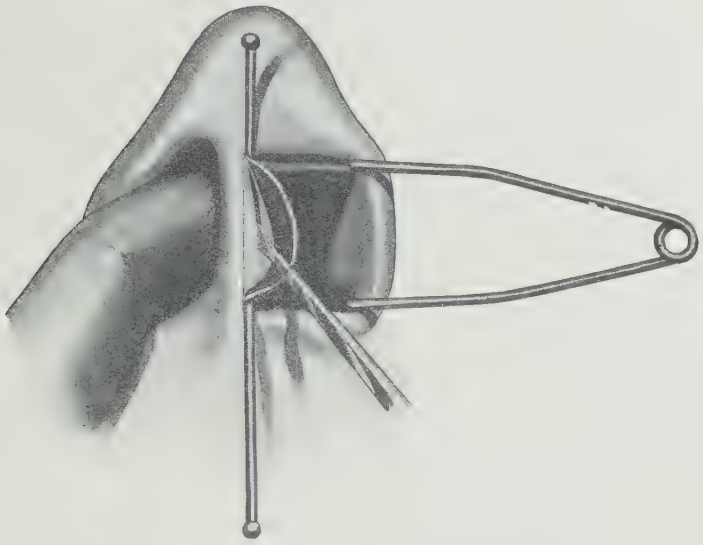
FIG. 64

Showing the line of incision (*a*, *a*) through the periosteum along the crest of the vomer to facilitate the elevation of the membranes. A similar incision should be made on the opposite side of the crest.

The elevation should be begun along the ridge of the nose, as shown in Fig. 60, and carried down to the upper border of the vomer with the whole length of the elevator. The elevator should then be removed and a short-bladed scalpel introduced and an incision made with it along the crest anterior to the perpendicular plate of the ethmoid (Figs. 62, 63, and 64). The elevator should then be reintroduced and the elevation (on the side of concavity of the septum) continued to the floor of the nose. Posterior to the cartilage the elevation is easily made to the floor of the nose as the periosteum is continuous from the roof to the floor of the nose.

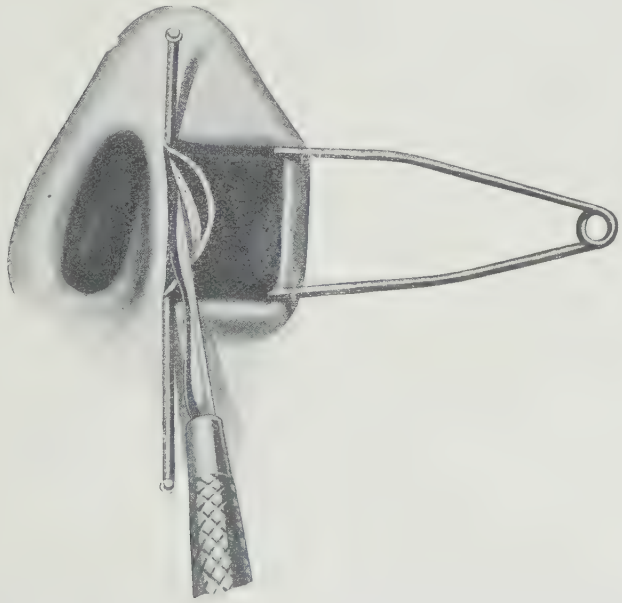
I have often noted the liability of the mucoperiosteum to tear at the junction of the vomer with the cartilage. Neumann's findings,

FIG. 65



The mucoperichondrium being elevated, the cartilage is incised, care being exercised to avoid perforating the mucoperichondrium upon the opposite side of the septum.

FIG. 66



The cartilage being incised, the mucoperichondrium of the opposite side of the septum is being elevated. The elevation is begun with a sharp or semisharp elevator, and is completed with a blunt elevator.

namely, the close adherence of the mucoperichondrium to the underlying periosteum and the reflection of the periosteum over the crest, adequately explain it. This knowledge, and the periosteal incisions I have recommended, greatly facilitates the elevation and reduces the liability of perforations.

5. **The Incision through the Cartilage.**—The incision through the cartilage (after Killian's incision) may be made with a small short-bladed sharp knife, though it may be done with the tip of a curette or other semisharp instrument. Some operators prefer the latter method, believing there is less danger of perforating the opposite mucous membrane. If a knife is used the tip of the finger should be placed in the opposite nostril to exert counterpressure while the incision is being made (Fig. 65). The cartilage should be incised very cautiously, almost cell by cell, with very delicate pressure, until the tip of the instrument is felt through the thickness of the opposing mucoperichondrium. Under no circumstance should the opposite mucoperichondrium be incised, as this would cause a permanent perforation of the septum unless the incision were immediately closed with sutures. I wish to emphasize the fact that if both mucous membranes are perforated, at points exactly opposite, a permanent perforation will follow unless one is sutured by Yankauer's method. If the perforations are not opposite, a permanent perforation will not result, though the process of repair will be prolonged.

If the incision through the cartilage is made with a curette or other semisharp instrument, the finger should be placed in the opposite nostril to exert counterpressure while the instrument is being ground through the cartilage. The tip of the finger enables the operator to detect when the entire thickness of the cartilage is penetrated. The cartilage should be incised in a line corresponding with the Killian incision. If, however, the Hajek incision is made the cartilage is not incised, as the incision is anterior to its forward extension. When this incision is made the mucocutaneous membrane is dissected from both sides of the fibrocartilage of the septum with a small, sharp knife.

Fig. 67

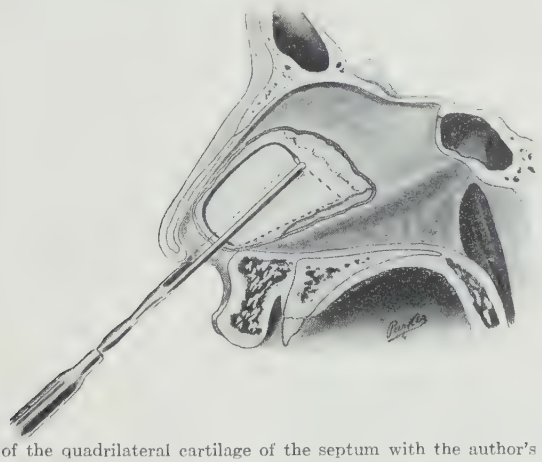
Showing the Foster septum speculum in position after the membranes are elevated.

6. **The Elevation of the Opposite Mucoperichondrium and Periosteum.**—When the cartilage is completely incised, the semisharp elevator (Fig. 66), with its flat surface in apposition with the cartilage, is inserted into the cartilaginous incision. The sharp edge of the tip of the elevator should be moved up and down between the edge of the cartilage and the adherent mucoperichondrium, especially at the upper limit of the incision, as the membrane is less adherent at this point (Fig. 66). Having started the elevation the blunt elevator should be introduced and passed upward parallel with the ridge of the nose (direction

of least resistance) until its tip is near the cribriform plate of the ethmoid bone. The elevation should then be continued downward and backward, with the shank of the instrument as previously described, and extending over an area considerably larger than the area of cartilage and bone to be removed. Never attempt to elevate below the crest of the vomer when it forms a dense bony ridge, as to do so would only result in an extensive laceration of the membrane. (See Removal of the Vomer.)

7. **The Removal of the Cartilaginous Portion of the Septum.** In nearly all cases this is most easily accomplished with my swivel knife (Figs. 69 and 76), though it may be done with Killian's double-edged spokeshave, a biting forceps, or angular knives. The advantage of the swivel knife is the ease, precision, and rapidity with which it encircles the cartilage, and the further fact that it removes it in one piece, thus allowing the operator to study the specimen as a whole.

FIG. 68

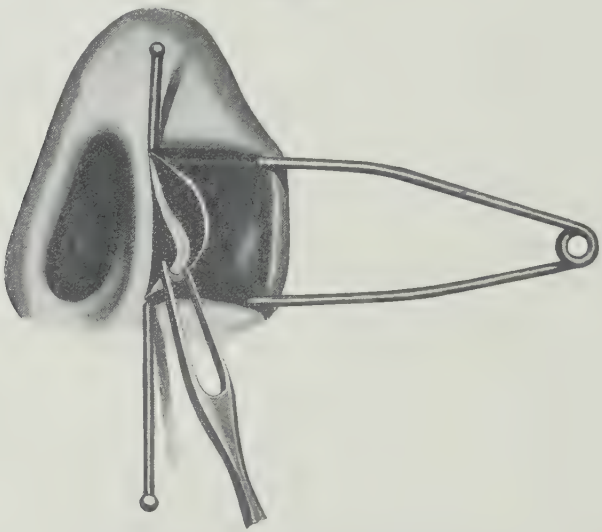


The removal of the quadrilateral cartilage of the septum with the author's swivel knife. The membrane is shown removed to expose the knife to view. In the actual operation the membrane is not removed.

Before using the swivel knife the mucoperichondria should be distended with a septum speculum (Figs. 67 and 84) to lift them from the cartilage and to provide room for the swivel knife. This exposes the cartilage to full view. The swivel knife may be applied to the cartilage at either the upper or lower portion of the incision. If to the upper portion, the incision will be made upward, backward, downward, and finally forward, along the floor of the nose, thus completely encircling the portion of the cartilage to be removed (Fig. 68). If applied at the lower portion of the incision, the cut will extend backward, along the crest of the vomer to the junction of the vomer and perpendicular plate of the ethmoid, thence upward and forward, along the antero-inferior margin of the perpendicular plate, and then downward, parallel with the ridge of the nose to the upper limit of the primary incision of the cartilage, thus encircling the portion of the cartilage to be removed (Fig. 69). If the incision is

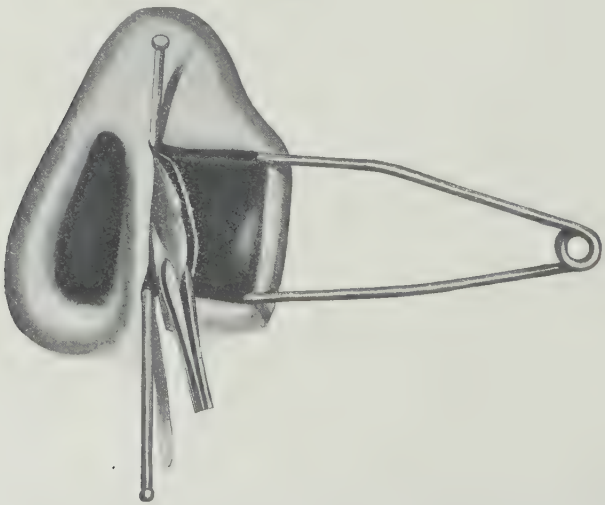
begun at the lower limit of the primary incision it may be necessary first to make a slight cut with a knife or scissors, as the cartilage is often fibrous at this point.

FIG. 69



The author's swivel knife in position at the lower portion of the incision of the cartilage.

FIG. 70

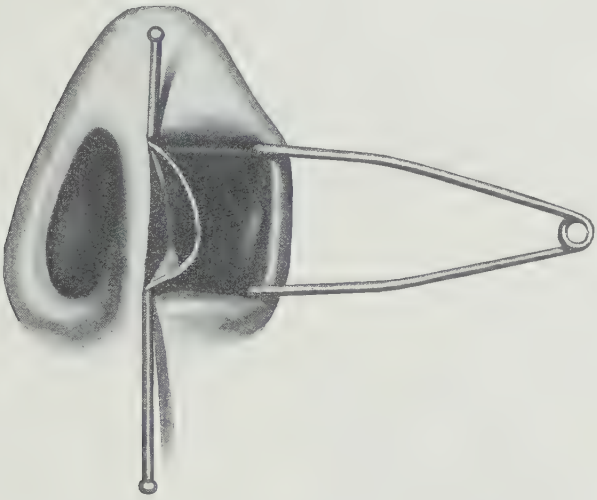


The cartilage, having been excised submucously with the swivel knife, is removed from the mucoperichondrial pouch with dressing forceps.

The swivel knife is easily controlled and is an instrument of great precision. The swivel blade follows the direction toward which the tips of the prong are directed. The resistance of the tissues controls the position of the swivel blade so that it always follows the prong-tips.

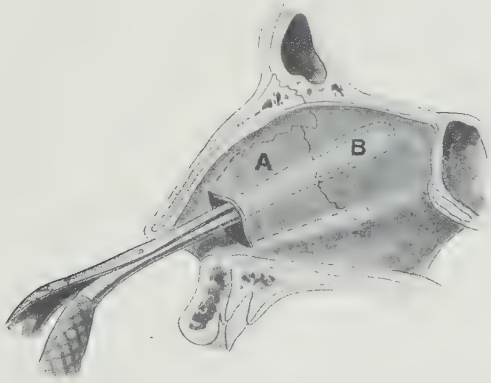
This instrument was suggested by Killian's fixed double-edged septum cartilage spokeshave, though the swivel blade is an entirely new principle in surgical instruments. While the general appearance of the

FIG. 71



Showing the mucoperichondrial pouch after the removal of the cartilage. The bony crest of the vomer is shown in the bottom of the pouch, while deep in the pouch is shown the perpendicular plate of the ethmoid extending upward from the crest of the vomer. This should be removed with the Ballenger-Foster forceps, as shown in Fig. 72.

FIG. 72



The removal of the perpendicular plate of the ethmoid bone with the Foster-Ballenger forceps: *a*, the area of cartilage previously removed with the swivel knife; *b*, the area of perpendicular plate removed with the Foster-Ballenger forceps.

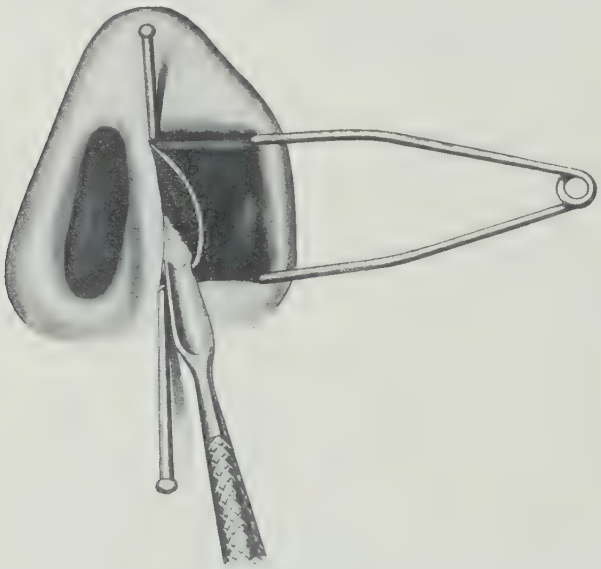
instruments are much alike, the swivel principle in my knife makes it quite different. They are alike only in the fact that the handle and prongs are similar.

Having encircled the cartilage, it is removed *en masse*, with dressing forceps, as shown in Fig. 70. Fig. 71 shows the perpendicular plate

in the depth of the mucoperichondrial pouch after the cartilage is removed.

8. **The Removal of the Perpendicular Plate of the Ethmoid.**—This is accomplished with the Foster-Ballenger bone forceps (Fig. 77). They remove a comparatively large piece at each bite, and two or three bites remove all that is necessary. The bites may be made without removing the forceps from the mucoperichondrial pouch (Figs. 72 and 77), a point of considerable importance, as each introduction of an instrument into the perichondrial pouch increases the chance of injurying the membranes. The perpendicular plate may also be removed by seizing it with heavy dressing forceps and twisting it from its attachments, though this is a crude and dangerous method, as it may fracture the cribriform plate.

FIG. 73

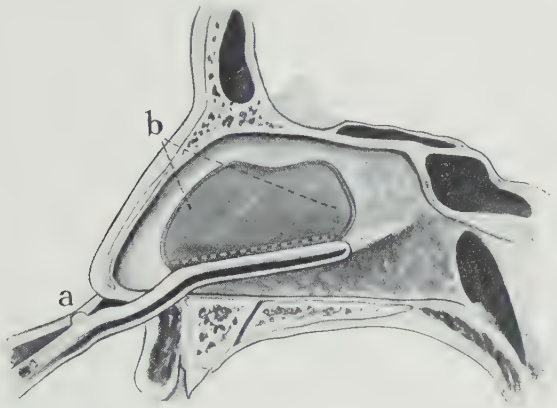


The removal of the thickened crest of the vomer with the author's V-shaped gouge.

9. **The Removal of the Vomer.**—Various methods are in vogue for the removal of the deviated vomer, which often forms the so-called ridge of the septum. It is obviously almost impossible to elevate the mucoperiosteum beneath the crest of the ridge (vomer), as its anterior portion is near the floor of the nose, and to attempt to pass the elevator over the margin of the crest would almost certainly tear the tense mucous membrane along this line. Fortunately it is not necessary to elevate below the crest, as the deviated or thickened bone can be removed without previously elevating the membrane beneath the crest.

An old and approved method of removing the vomer is with Hajek's gouge or some modification of it (Figs. 73, 80 and 81). The V-shaped end of the gouge is engaged at the anterior end of the ridge of bone and driven with a mallet into its substance for a short distance, and then the handle

FIG. 74



The author's method of removing the ridge of bone in the submucous resection of the septum: *a*, the septum forceps grasping the ridge, the blades being external to the mucous membranes. The forceps is rotated on its longitudinal axis, as in the Asch operation, thus fracturing the vomer from its lower attachment; *b*, the area of cartilage and perpendicular plate of the ethmoid previously removed; the mucous membrane is shown removed, though this is not actually done in the operation.

of the gouge is depressed, and thus partially splinters the bone from its attachment. The gouge is then driven farther into the ridge until it is finally removed in its entirety. As the vomer is loosened it separates from the mucoperiosteum without tearing it, provided, of course, the gouge is always directed parallel with the anteroposterior direction of the crest of the vomer.

Another method of removing the deviated vomer is with a specially devised bone-cutting forceps. Of these, L. M. Hurd's is probably the best (Fig. 82). It is powerful, has downward cutting blades, and with it the bone may be bitten away with considerable ease.

R. H. Brown has devised a guarded drill, to be used with an electric motor for the submucous removal of the deviated vomer.

Personally, I prefer to first fracture the vomer from the premaxillary bone at the floor of the nose, and then to remove it with heavy dressing forceps, introduced into the mucoperiosteal pouch. During the process of fracture the mucoperiosteum separates from beneath

FIG. 75

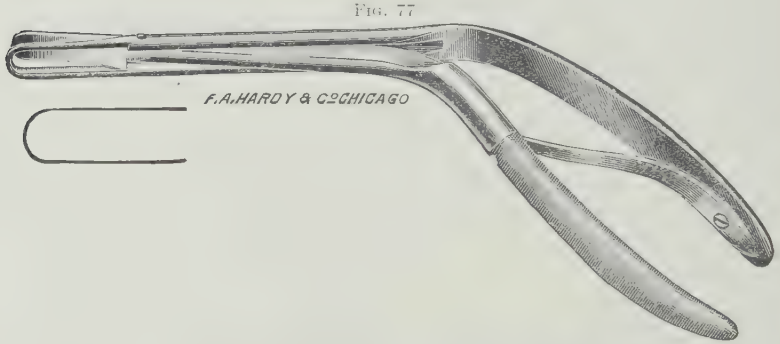
The removal of the vomer after it is fractured is shown in Fig. 74.

the crest of the vomer and thus allows the long ridge of bone to be removed from the pouch (Fig. 75). In young adults and children my method is not applicable, as the vomer is not yet fully ossified. In

Fig. 76

The author's swivel cartilage knife.

Fig. 77

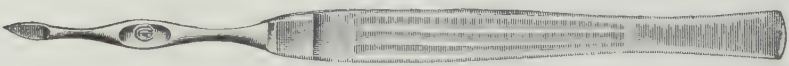


Foster-Ballenger perpendicular plate bone forceps.

Fig. 78

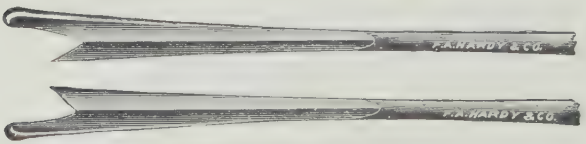
Hajek-Ballenger mucoperichondria elevators.

Fig. 79



The author's mucosa knife.

Fig. 80



Hajek's septum gouge.

Fig. 81

The author's septum gouge.

adults it is a speedy and an almost painless procedure, and results in but little or no shock, as the cartilage and perpendicular plate of the septum have been previously removed. There is, therefore, no solid

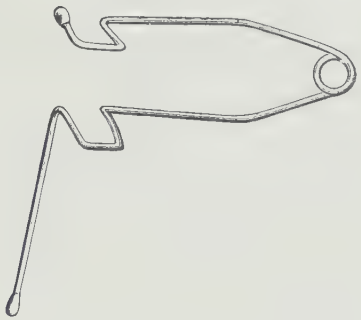
tissue above to communicate the shock to the cranial contents (Fig. 74). The technique of the procedure is as follows:

Introduce the blades of the Asch septum forceps into the nasal chambers *outside of the mucoperichondria*, and grasp the deviated vomer firmly, twisting the forceps in its longitudinal axis and fracturing the vomer from its attachment at the floor of the nose. The blades of the Asch forceps should be placed a little above the floor of the nose, as they may otherwise tear the mucous membrane at the junction of the vomer

FIG. 82

Hurd's bone septum forceps.

FIG. 83

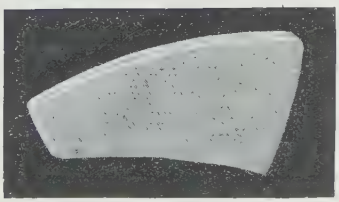


Allen's nasal speculum.

FIG. 84

Ballenger-Foster septum speculum.

FIG. 85



Simpson's nasal sponge splint.

with the floor of the nose. The fracture should be thorough, in order to permit the detachment of the fragments from the floor of the nose. Remove the Asch forceps and introduce the tips of heavy dressing forceps into the mucoperichondrial pouch, grasp the vomer, and with a tugging, teasing motion lift it from its fractured base. The mucoperiosteum remaining attached below the crest will readily separate and allow the bone to be removed (Fig. 75).

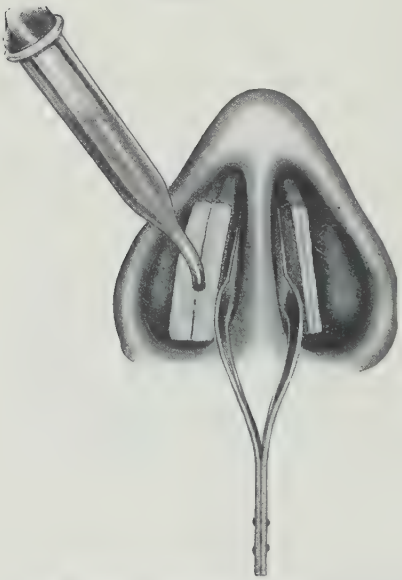
10. **Inspection of the Field Operated Upon.**—After the completion of the various steps of the operation, the field operated upon should be

subjected to the closest scrutiny. If a portion of the deviated cartilage or bone is left in place it may be found, when healing is complete, that it will still cause obstruction of the nasal chambers. Every vestige of the deviated framework of the septum should be removed (Dundas Grant). Bone-cutting forceps of one type or another are usually used for this purpose in the cartilaginous and perpendicular plate portions of the septum, though the gouge may be more useful for cutting along the floor of the nose. I have found it a very helpful practice to insert a finger an inch or two into the nasal chambers, as it enables me to detect the presence of bony prominences which might otherwise have escaped my notice.

11. **The Dressing.**—A dressing should be placed in the nasal chambers for two purposes, namely: (*a*) Coaptation of the membranes, and (*b*) prevention of the formation of a blood clot in the mucoperichondrial pouch.

The dressing most frequently used is composed of compressed cotton or Berney's sponge tents. They have been placed on the market under the name of the Simpson-Berney nasal splints (Fig. 85). The mucoperichondria are first clamped together with the septum speculum, then one or two of the splints are introduced into each nasal chamber. The patient's head is then inclined backward and a few drops of distilled water, or of the peroxide of hydrogen, are instilled into the ends of the splints (Fig. 86). This causes them to swell and compress the membranes together.

Fig. 86



The Simpson sponge-tent dressing in position at the close of the submucous operation. The left side shows the tents dry, the right moist and swollen. The Foster speculum holds the membranes in apposition while the tents are being introduced.

12. **The After-treatment.**—The nasal dressing should be removed in from twenty-four to forty-eight hours after the operation. The use of bismuth paste on the splints has a chemotactic effect (reaction of inflammation) upon the mucous membranes (Emil Beck) and thus reduces the chance of infection. The splints interfere with the ventilation and drainage of the nose, and are therefore usually removed at the expiration of twenty-four to forty-eight hours. Subsequently the nasal chambers are irrigated with a mild solution of the permanganate of potash three or four times daily. The temperature of the solution should be about 104° F., or as hot as the patient will tolerate. If crusts form over the incision the patient should be provided with a tube of sterile vaseline and instructed to squeeze some of it into the vestibules of the nose, twice a day, and to compress the

alæ of the nose and thus smear it over the mucous membranes. Healing should be completed in from three to ten days, unless one of the membranes has been lacerated, in which event it may be somewhat prolonged.

Accidents.—This operation is peculiarly liable to certain accidental complications, some of which are inherent in the difficult technique, while others are the results of the inexperience or temperamental weaknesses of the operator.

Incision through Both Mucous Membranes.—The novice is likely to extend the incision through both mucous membranes, as the cartilage is easily incised and the most delicate manipulation of the knife is necessary in making the incision through it. Before the operator realizes it the incision has extended through the mucous membrane upon the opposite side. To avoid this accident the cartilage should be incised, cell by cell, as it were, until the point of the knife is perceived by the tip of the index finger, which is in the opposite nostril. Should both mucous membranes be incised along the line of the Killian incision it will be necessary to close one of the incisions with Yankauer's needles and methods of suture. The sutures should be removed at the expiration of the third day.

Tears through Both Mucous Membranes.—Sometimes during the process of elevating them, the mucous membranes are lacerated at points exactly opposite. Should this accident occur an endeavor should be made to close one of the apertures by Yankauer's method of suturing, or to reintroduce the cartilage removed from the septum as suggested and practised by Dr. P. G. Goldsmith, of Toronto. (See Perforation of the Septum, at the end of this chapter.)

Destruction of the Mucous Membrane upon One Side of the Septum.—This accident may occur during the elevation of the membrane or during the removal of the cartilaginous and bony portions of the septum with cutting forceps. This is especially true if the elevation of the mucoperiosteum has not been extended over a sufficiently large area. It may also occur while the cutting forceps are in use, the mucous membrane being accidentally engaged in the forceps. This can be avoided by exercising great care through observation before closing the forceps.

Sinking in of the Ridge of the Nose.—This accident has been reported only a few times and need not be feared except under a few conditions. When it occurs it is due to one of three conditions: (*a*) The removal of the cartilage too near the ridge of the nose, (*b*) chondritis following or preceding the operation, and (*c*) traumatism.

(*a*) A cartilaginous ridge at least one-fourth of an inch in depth should be left to support the external nose. A greater width is desirable, especially if the deviation is traumatic in origin, as in this case chondritis may have weakened the cartilage.

(*b*) Chondritis or inflammation of the cartilage following the operation may soften the cartilage of the ridge of the nose and cause it to drop or sink in and thus produce external deformity. The nose should be carefully observed for several days after the operation for inflamma-

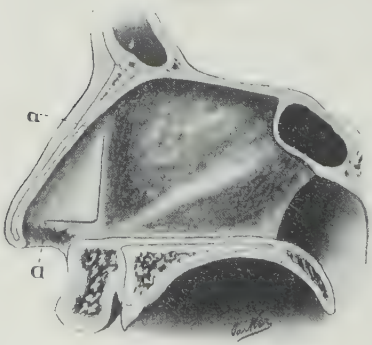
tory symptoms, and if they occur strenuous efforts should be made to combat them. Perhaps the best procedure is to employ heat over the nose. The application of hot fomentations every fifteen minutes is the best mode of procedure. In addition to this the nasal chambers should be irrigated with normal salt solution (one teaspoonful of table salt to each pint of water) every three hours. The head should be inclined well forward, almost between the knees, and the mouth kept open during the irrigations. These precautions prevent the patient swallowing and carrying the solution to the tympanic cavities, in which case it might produce otitis media or mastoiditis.

When the ridge of the nose sinks in after submucous resection of the septum, it is sometimes possible to correct the deformity by the subcutaneous injection of cold paraffin.

(*c*) A blow upon the nose after the submucous resection operation might cause a sinking-in of the ridge below the nasal bones. I have never known of such an accident, though I presume it will occur in a few cases in due course of time, as the cartilaginous support of the nose is weakened by the submucous operation.

The Freer or Open Method.—According to O. T. Freer, this procedure is especially adapted to cases in which unusual difficulties necessitate an operative field as open as possible for inspection, as those in which the mucous coverings are very adherent, or in which the operation is performed in the small nostrils of children, for deviations with extreme angles or for extensive deep-seated deflections. The open operative field is obtained by means of Freer's reversed L mucous membrane incision (Fig. 87), consisting of a vertical limb, made well back in the nose, joined by a horizontal one conducted forward from it along the base of the septum, in most cases to the front of the nasal vestibule. These incisions outline a flap which is dissected upward and backward with a suitable blade from its basal line until the vertical incision is reached (Fig. 87). The flap is then uplifted by means of the dulled elevator and held forward out of the way by the use of a retractor, by means of which the nose is held open by an assistant, these retractors taking the place of a speculum. A large field of cartilage is thus uncovered in front so that the first incision through it can be made in plain view. It outlines a tongue-shaped flap of cartilage with its base backward, and which, when uplifted from the mucous coverings of the concave side of the deviation gives a broad entrance into the concavity of the deflection, making all of its recesses readily accessible to sight as the denudation progresses, so that sharp dissection can be safely accomplished without risk of per-

Fig. 87



a, *a*, Freer's incision.

foration. After the posterior portion of the mucous coverings have then been uplifted on the side of the convexity of the deviation, the cartilage, now entirely denuded, is excised with a little keen, hoe-shaped blade and by sharp elevators. The remains of the cartilage are then detached posteriorly from their usual attachments to the side of the vomer by means of long elevators; and the bony resection is begun by an incision upon the upper border of the ridge (often hidden) and anterior border of the vomer, splitting the periosteal envelope of these structures. The periosteum is then pushed off from their convex and concave sides by means of suitable chisel-edged raspatories and blades and the entire bony deviation bared by them and by the elevators. It is then cut away by the Freer reinforced punch forceps, including the ridge of the nasal floor, and as much of the vomer and perpendicular plate as is needed. The chisel should only be used in cases in which the ridge is unusually broad.

Freer operates with the patient in a semirecumbent position on a dental chair, which can be raised and lowered. He employes the Kirstein head lamp, and stands beside the patient.

He has devised a special instrumentarium for the operation. It includes a number of keen-edged blades for dissection, which he uses whenever, in his opinion, the coverings of the deviation cannot be uplifted readily with dull-edged instruments.

After the operation, the nostril of the side on which the incisions have been made, should be packed with narrow strips of lint saturated with bismuth subnitrate and soaked in oil vaseline; the strips should be introduced in layers, in order to avoid injurious bunching, and also to hold the flaps in place.

Hematoma of the septum does not occur when coaptation of the mucoperiosteal membranes has resulted from the use of suitable dressings in either method of operating, and perforations are rare if the technique is carefully carried out, even in extensive bony resections.

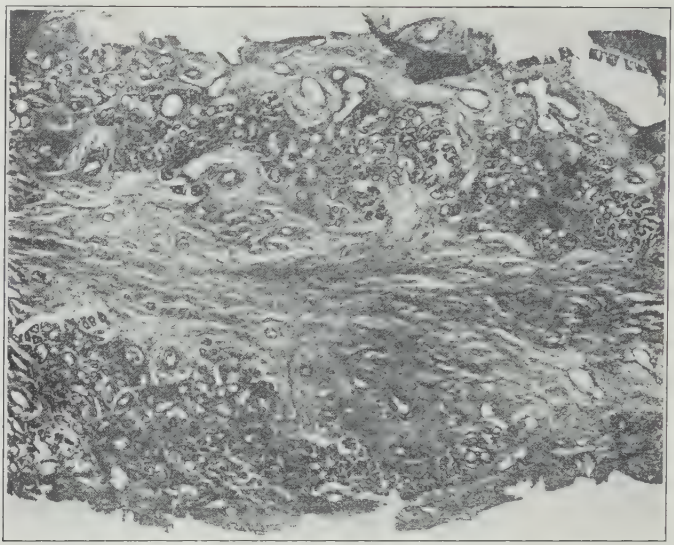
Remarks.—Some writers have stated that the swivel knife is objectionable because it is likely to tear the mucous membrane. Such a statement can mean but one of two things, namely: (*a*) That the operator is extremely awkward, or (*b*) that he fails to elevate the membrane sufficiently. Any operator with but a moderate experience with the submucous resection of the septum knows that it is almost impossible to tear the mucous membrane with the swivel knife if the mucoperichondrium is previously elevated over the entire operative field.

One writer makes the claim that the swivel knife is not an exact instrument—is not under the exact control of the operator. This is a mistaken idea, and is not based upon personal experience, but is a theoretical deduction. As a matter of fact, it is one of the most exact and easily controlled instruments used in this operation. It cuts cartilage with but slight resistance, and may be directed with the greatest precision, so as to encircle the amount of cartilage it is necessary to remove.

Authors differ as to the reformation of the cartilage of the septum after its removal. According to J. C. Beck (Figs. 88 and 89), no cartilage cells were found in the tissue after a lapse of two and one-half years.

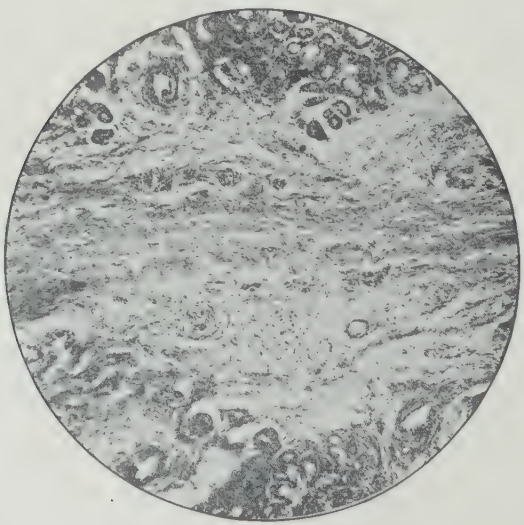
The removed cartilage was replaced by dense fibrous tissue. Freer, on the other hand, claims that the cartilage reforms, especially in the younger subjects.

FIG. 88



Section of septum two and one-half years after a submucous resection of bone and cartilage shows no regeneration of either bone or cartilate, but is replaced by a dense fibrous tissue. Age, forty-seven years. (Specimen kindly loaned by Dr. J. C. Beck.)

FIG. 89



Same as Fig. 88, with higher power.

PERFORATION OF THE SEPTUM

Etiology.—The causes of perforation of the septum may be divided into (*a*) congenital, (*b*) chronic granuloma, (*c*) traumatic, (*d*) acute infection, and (*e*) atrophic or perforating ulcer.

(*a*) Congenital perforation is extremely rare, Zuckerkandl having reported a few cases.

(*b*) Chronic granulomata—as syphilis, tubercle, and lupus—have caused a considerable percentage of the cases, some authors attributing as high as 50 to 60 per cent. to syphilis alone. In my experience the percentage due to syphilis is much less than this; syphilis is not, however, as common in this as in some other countries. Syphilitic perforations almost always include the bony portion of the septum, whereas, tubercle and lupus are limited to the cartilaginous portion. The tuberculous and lupous origin of the perforating ulcer may be determined by finding the tubercle bacilli, or tuberculous histological changes in the tissues. A slow but reliable method of demonstrating the tuberculous process is to inject a guinea-pig with some of the tissue from the ulcer. Six weeks later hold a postmortem on the pig and note the presence or absence of a tuberculous process.

(*c*) Traumatic perforations may include any portion of the septum, as they are usually due to surgical procedures, though they may be due to accidental violence and to picking the nose with the finger nail.

(*d*) Acute infectious diseases, as diphtheria, scarlet fever, typhoid fever, phlegmonous abscess, etc., may result in perforations.

(*e*) Atrophic or perforating ulcer of the septum is probably the most common type of perforation. Several conditions contribute to the etiology of this type of perforating ulcer. An anterior spur or deviation of the cartilaginous portion of the septum is usually present, and on account of its projection into the field of the inspiratory current of air, it is subjected to constant mechanical irritation and to the desiccation of the secretions which constantly accumulate upon it. The ciliated columnar epithelium undergoes retrograde changes to a less specialized type of epithelium (pavement epithelium). The dust and other foreign substances in the air also irritate the epithelium and mucous membrane.

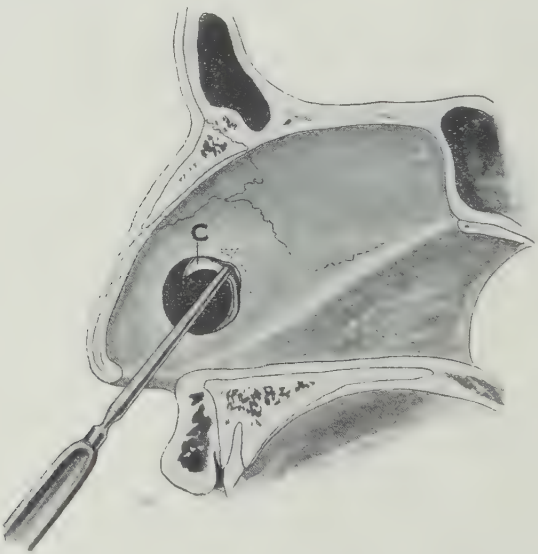
The crusts thus formed in this area become adherent, and are forcibly blown or picked off with the finger nail, the epithelium coming away with them. Hemorrhagic deposits in the mucous membrane occur, and epistaxis is of frequent occurrence. The retrograde process continues until the entire thickness of the septum is destroyed. Infection plays a part in the foregoing process.

Symptoms.—The symptoms of perforation of the septum vary with the size, cause, and location of the perforation. A small anterior perforation, sometimes gives rise to a musical, whistling sound, whereas, a large one does not. If the perforation is associated with a prominent bony spur, there may be a sense of stuffiness in the nose. Crusts, if of large size, may give rise to the feeling of a foreign body in the nose, and,

if forcibly blown or picked off, may cause nasal hemorrhage. Repeated epistaxis should arouse suspicion of a perforating ulcer. Syphilitic ulceration is usually accompanied by an offensive necrotic odor. Many cases will progress to complete perforation without the patient's knowledge of the fact.

Treatment.—If seen in the ulcerative stage, before perforation, the progress of the local retrograde changes may be checked by appropriate local cleansing and antiseptic washes and ointments, or, if due to syphilis, by the administration of the proper remedies for this disease. When the perforation is complete, little can be done except in a surgical way. Large perforations are not, however, amenable to surgical closure. Small ones may often be closed by proper plastic surgical procedures.

FIG. 90



The edge of the cartilage around the perforation (*c*) being removed with the author's single-tined swivel knife in Goldstein's plastic septum operation.

Goldstein's Plastic Flap Operation.—Dr. M. A. Goldstein has suggested and successfully used the following operation. A plastic flap of mucous membrane is turned into the opening and inserted and sutured between the elevated membranes of the two sides of the septum.

Technique.—(*a*) Cocaine anesthesia.

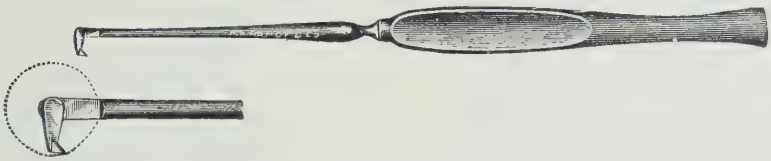
(*b*) The rim or edge of the perforation is freshened by paring off the epithelium and mucous membrane.

(*c*) The mucoperichondrium is then elevated for a distance of one-half inch around the edge of the perforation.

(*d*) A ring of cartilage is then resected for one-eighth to one-fourth inch from the edge of the perforation, the author's single-tined swivel knife being used for the purpose (Fig. 90).

(*e*) A mucous membrane flap, the area of which is considerably larger than the perforation, is then dissected from the most convenient surface of the septum and turned into the perforation and tucked between the elevated membranes around the perforation. I have devised a trailing swivel knife (Fig. 91) for outlining this flap. The method of using it is shown in Fig. 92.

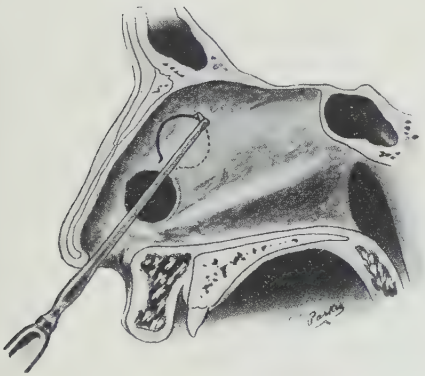
FIG. 91



The author's mucosa swivel knife.

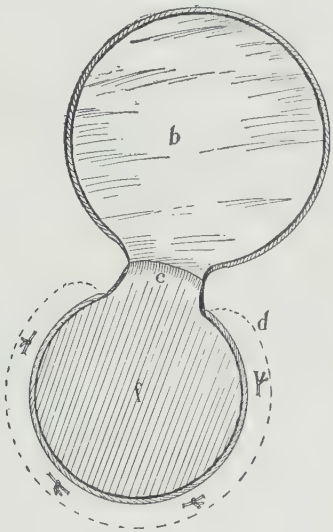
(*f*) When the pedicled flap is in position (Fig. 93), three or four Yankauer stitches hold it in position. One surface is covered by epithelium, while the other is left to heal by granulation from the edges of the closed perforation.

FIG. 92



Showing the method of outlining the flap with the author's swivel mucosa knife for the closure of a perforation of the septum.

FIG. 93



f, the plastic flap sutured in the perforation; *c*, the pedicle of the plastic flap; *b*, the denuded area from which the plastic flap is removed heals by granulation; *d*, the edge of the plastic flap between the mucoperichondria of the septum.

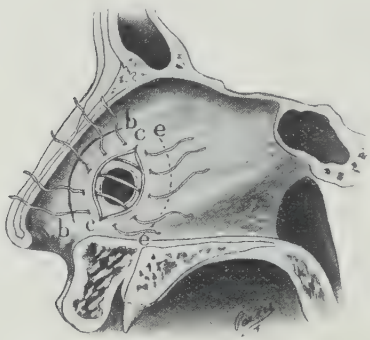
Hazletine's Plastic Operation.—This operation is also only suited to small perforations. It is more simple than the pedicled flap operation, and appears to be a more satisfactory procedure.

Technique.—(*a*) Cocaine anesthesia.

(*b*) Freshen the edges of the perforation and elevate the mucoperichondrium, as in the submucous resection operation.

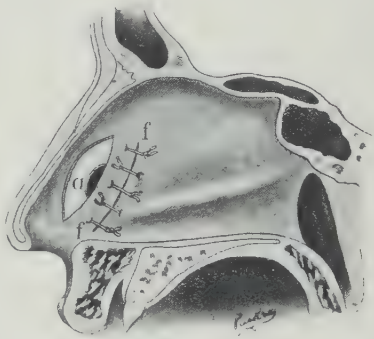
(*c*) Make a long curved incision (Fig. 94, *b*, *b*) through the mucoperichondrium, one-fourth to one-half inch anterior to the perforation, and elevate the ribbon-flap thus made.

Fig. 94



Schema of Hazlette's plastic operation for the closure of perforations of the septum: *b*, *b*, incision in front of the perforation; *e*, *e*, the incision posterior to the perforation on the opposite side of the septum; *c*, *c*, the freshened edges of the perforation.

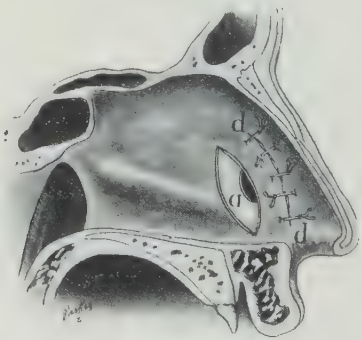
Fig. 95



Detail of Fig. 94, showing the opposite side of the septum, the flap formerly covering area *a* is sutured to the posterior margin of the perforation.

(*d*) Make a long curved incision (*e*, *e*) through the mucoperichondrium of the opposite side of the septum, one-fourth to one-half inch posterior to the perforation, and elevate the flap.

Fig. 96



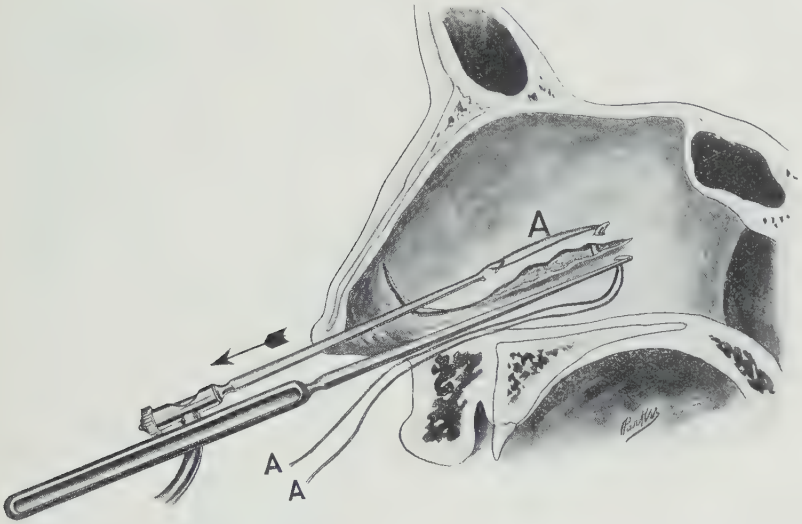
Detail of Fig. 94. *a*, the denuded cartilage after the plastic flap (*d*, *d*) is sutured.

(*e*) Suture the anterior flap to the freshened posterior edge of the mucous membrane of the perforation (Fig. 95), and the posterior flap on the opposite side of the septum to the freshened anterior edge of the membrane of the perforation, as shown in Fig. 96. The areas *a* and *a* heal by granulation.

(*f*) Remove the sutures in twenty-four to thirty-six hours. By this procedure the perforation is covered by two mucous membranes, and, the lines of suture not being opposite, closure of the perforation follows.

Yankauer's Intranasal Suture.—Sydney Yankauer has devised instruments for intranasal suturing which may be applied in repairing rents in the mucous membrane of the septum following the submucous resection operation, in closing the mucous membrane wound of the inferior turbinate after resecting the hypertrophied membrane and bone, and in the plastic operations upon the septum for the closure of chronic perforations. The technique is as follows:

FIG. 97



Yankauer's intranasal suture: *A*, *A*, *A*, the suture thread, being drawn forward with the hook. The needle is then reversed and withdrawn from the nose, rethreaded, and another stitch taken in the torn mucous membrane.

The Introduction of the Suture.—Catgut suture eighteen inches in length should be used. It should be placed in a carbolic solution for a few moments to soften it. The suture may be passed through either flap, preferably through the more movable one. It should then be passed through the other flap after first coapting the two flaps. If necessary, the crotch forceps may be used to facilitate the penetration of the flaps with the needle.

Grasping the Thread.—The eye of the needle should project only one-eighth of an inch through the membranes. One of the threads should then be seized with the hook, which may be rotated with the pilot wheel at the end of the instrument until it is in position to seize the thread.

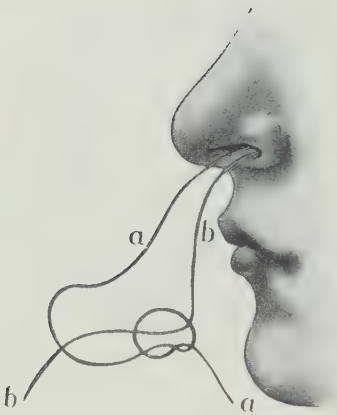
Withdrawing the Needle.—When the thread is in the grasp of the hook, the needle should be removed from the flaps by rotating it back-

ward until it is free from the membranes. It should then be withdrawn from the nose. The hook should in the meantime be kept close to the needle puncture to prevent the thread from tearing out.

Withdrawing the Hook.—The hook is then withdrawn from the nose with the loop of thread. One side of the loop is then drawn from the nose ready for making the slip-knot.

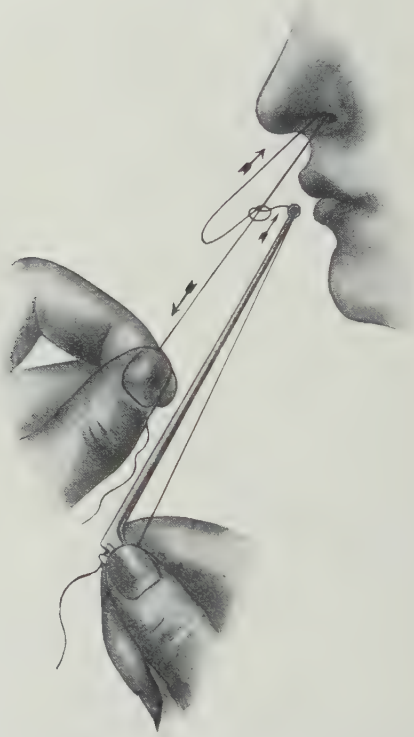
Making the Slip-knot.—First see that both ends of the thread are outside of the nose, and that they are not entangled. To make the slip-knot, have one end include half of the thread (nine inches) outside

Fig. 98



The slip-knot.

Fig. 99



Yankauer's intranasal suture method of conveying the knot into position in the nasal chamber.

of the nose, the other end being correspondingly shorter. Then make a simple overhand knot near the middle of the long ends, and pass the shorter end through the bight of the knot, as shown in Fig. 98. Tighten the slip-knot until it binds the through thread. Two threads now come through the knot, one the knot end, the other the slip end.

Closing the Slip-knot.—The slip-knot being drawn tight over the thread, it is brought near the nostril. The knot end of the thread is passed through the ring of the suture closer until the ring is near the knot. The end of the thread is then held with the thumb against the

handle of the instrument, as shown in Fig. 99. The left hand holds the slip end, and the ring suture closer is advanced into the nose and the knot closed where the suture passes through the mucous membrane. The ring passes beyond the point where the suture passes through the membranes, and thus makes as firm a knot as may be desired.

The remaining portion of the wound may be closed by making a continuous suture with the longer end of the thread, only using the slip-knot for the last stitch to fix it in place. If preferred for any reason, each stitch may be made separately as above described, cutting off the ends as in external suturing.

The sutures should be removed in from two to three days.

The Safety Knots.—In order to prevent the slip-knot from becoming loose, it is advisable to make a true surgical knot, consisting of two overhand knots, above the slip-knot.

Goldsmith's Operation.—When both mucoperichondria are torn during a submucous operation, thus making it probable that a permanent perforation will follow, the cartilage removed may be reintroduced between the membranes, and thus afford a bridge over which the granulating edges of the mucous membranes may extend and close the perforation.

Technique.—(*a*) When the cartilage is removed with the swivel knife during the submucous resection operation, it should be placed in normal salt solution to preserve it for use in case the mucous membranes are torn. The cartilage may be thus preserved for about six weeks in cold weather.

(*b*) If the cartilage is misshapen, it may be straightened or trimmed to adapt it to the requirements of the case.

(*c*) It should then be introduced between the membranes, care being exercised to bring the torn and ragged edges of the membranes well over the cartilage on both sides.

(*d*) A Simpson-Berney sponge-tent splint should then be introduced into each nasal chamber to hold the cartilage and membranes in position. The sponge-tents should then be moistened with sterile water to swell them. (See Fig. 86.)

(*e*) The tampons should be removed on the third day.

By the end of this time the granulations will have extended well over the cartilage and in a few more days will have covered it. In this way the perforation is bridged with new mucous membrane. The cartilage is gradually absorbed, leaving a membranous septum at this point.

Cartilage may also be used to close old perforations. The edges of the perforation should first be pared, the membranes separated around the circumference of the perforation, some cartilage removed, and the foreign cartilage introduced and retained in position, as in Goldsmith's procedure for rents and tears during the submucous operation.

CHAPTER VI

THE ETIOLOGY OF INFLAMMATORY DISEASES OF THE NOSE AND ACCESSORY SINUSES

INFLAMMATION

Acute Inflammation.—Acute inflammation is a threefold reaction excited by the presence of certain noxa, or irritant material, in the tissues. The noxa or irritant is usually a pathogenic microörganism and its toxin, or it may be of chemical or traumatic origin. When of chemical or traumatic origin the irritant primarily consists of the dead or broken-down cells of the tissues.

Dead or broken-down cells, when present in the tissues in excess, become foreign bodies, and, as such, a reaction of the living cells is excited for the purpose of eliminating them from the body. Furthermore, the dead cells in the process of disintegration give off a ferment or chemical substance which also excites a reaction, the purpose of which is to free the tissues of its presence. The reaction thus far excited is directly traceable to the presence of dead and disintegrating tissue cells. Ordinarily, after a short time, a secondary irritant gains entrance to the injured tissues and becomes the more important factor in the reactionary process. That is, pathogenic bacteria infect the impaired tissues so that in nearly every acute inflammatory process, whether it is due to primary infection or to chemical or mechanical trauma, pathogenic microörganisms must be regarded as the paramount exciting or noxious agent causing the reaction of inflammation.

The reaction of inflammation is, therefore, an increased physiological activity of the living tissues of the body for the purpose of disposing of a noxious or irritant substance or organism that has invaded them in excess of the normal quantities.

The reaction of acute inflammation is a threefold process, namely:

1. Increased hyperemia.
2. Increased nutrition (increased resistance).
3. Increased leukocytosis.

1. Increased hyperemia is a constant and important reaction, as through it the cells are provided with the extra nutrition they need under conditions of stress. The increased blood supply also stimulates and facilitates the increased migration of leukocytes, and it flushes the poisoned area and dilutes the noxious substance, and thus reduces the intensity of the irritation. The hyperemia is nearly always passive in type.

2. Increased nutrition of the cells is promoted by the hyperemia,

for obvious reasons. They are under stress because of the presence of noxious substances, and need extra nutritional facilities. Their vital force, or resistance, is not equal to the emergency placed upon them, and upon their resistance depends the issue of the warfare. Their means of defense may be characterized as twofold, namely: (*a*) their ability to envelop and digest microörganisms, and (*b*) their ability to produce and emit a biochemical substance or ferment, the purpose of which is to weaken or destroy their foe. All this requires increased nutrition (blood), which begets increased powers of resistance. If the nutrition is not adequate for these purposes, the microörganisms and their toxin, or biochemical irritant, may cause destructive and what we are accustomed to call pathological changes in the tissues.

3. Increased leukocytosis is also an important reaction of inflammation. While the function and modes of activity of the keukocytes is not fully understood, it has been fairly well demonstrated that the polymorphonuclear leukocytes envelop and destroy bacteria, while the lymphocytes envelop and destroy broken-down cells. Other cells, as the fibroblasts, also participate in these functions under certain conditions.

Quality of Reaction.—Parenthetically, I wish to add one additional statement concerning the adequacy of the reaction of inflammation. According to Adami, the reaction of inflammation may be of three types:

1. Adequate reaction.
2. Inadequate reaction.
3. Excessive reaction

The reaction is usually inadequate; that is, the increased hyperemia, cell nutrition, and migration of leukocytes is insufficient to dispose of the pathogenic microörganisms and their toxins before they have caused considerable damage to the tissues. It follows therefore, that in the treatment of inflammatory diseases the reaction of inflammation should be promoted rather than diminished. By so aiding the defensive and offensive activities of the tissues, the bacteria, their toxins, and the broken-down tissue cells may be speedily removed and a cure effected.

Inflammation Affecting Mucous Surfaces.—According to Adami, the main distinguishing feature of the mucous surface is the presence of a layer of mucous cells of a glandular type, capable, when stimulated, of forming and discharging relatively large amounts of mucin. The hyperemia, the exudation of serum, the migration of leukocytes, occur in the submucous layer just as in the subserous layers. The changes in the reaction are due solely to the interposition of this layer of mucous cells. There is, in the first place, a more definite basement substance interposing a certain amount of resistance to surface exudation. The layer of mucous cells is more complicated, and although the fully developed cells may be discharged, they are apt to remain relatively undifferentiated "mother cells" at the base; or otherwise the same intensity of irritation does not lead to as extensive a denudation. And, thirdly, by the combined action, it may be, of the irritant and of the hyperemia, the fully formed mucous cells are stimulated to produce increased amounts of mucin, so that an inflammation of moderate grade is char-

acterized by an abundant amount of mucinous discharge rather than of fibrinous deposit.

Adami speaks of such a moderate case, with exudation of serum containing abundant mucin, cast-off mucous cells, and relatively few leukocytes, as a "catarrhal inflammation;" if sufficient leukocytes are extruded the character is altered to that of a "mucopurulent inflammation;" if more severe, with complete destruction of the mucous membrane proper, then, as in serous surfaces, there is the same tendency for the leukocytic exudation to favor a deposit of fibrin upon the surface, and then we obtain a "membranous inflammation."

He says that despite the fact familiar to all that diphtheria is a disease set up by a specific bacillus, and the equally well-known fact that a like membranous inflammation may be induced by several forms of microbes, we still commonly speak of such a membrane as being diphtheritic. It would be better to confine this term purely to cases in which we know that the bacillus diphtheria is the causative factor; failing this, we may accept the term diphtheritic as covering all such membranous inflammation, and employ the term diphtherial for such cases as are of pure diphtherial origin.

If there is a more severe destruction of the superficial cells, ulceration may occur. When pyogenic organisms are present, there is a dissolution and breaking down of any fibrin that is formed and a consequent absence of a membrane. In such cases there is a distinct tendency for the process to extend in the submucosa beneath the still intact mucous membrane, the part becoming infiltrated with pus, forming what is known as phlegmonous inflammation.

Chronic Inflammation.—The reaction of chronic inflammation consists of the following phenomena:

(*a*) Slightly increased hyperemia.
(*b*) Slightly increased cell nutrition.
(*c*) Slightly increased migration of leukocytes.

It is needless to add that the reaction is inadequate to remove the noxa or irritants, which, according to pathologists, are usually bacteria of low virulence.

A product of chronic inflammation that is always present is the proliferation of fixed cells, usually of the least differentiated type, namely, connective-tissue cells. (See Hyperplastic Rhinitis.)

Etiology.—Having thus briefly defined inflammation, we are prepared to discuss its causes.

The causes of inflammatory diseases of the nose and accessory sinuses are divided into two groups, namely:

1. Exciting causes.
2. Predisposing causes.

1. **Exciting Causes.**—The exciting causes are bacteria and chemical and traumatic destruction of tissue cells. This phase of the subject has already been discussed under Inflammation, and will not be dwelt upon in this connection further than to say that pathogenic bacteria cannot irritate the tissues of the body so long as the resistance of the cells

is normal; that is, so long as they are healthy. There may be an exception to this rule when the germs are exceptionally virulent, though this is rare. Virulent pathogenic bacteria are constantly present in the upper respiratory tract, though they are harmless until the resistance of the cells is lowered by some intracorporeal or extracorporeal influence.

2. **Predisposing Causes.**—There are many predisposing causes of inflammatory diseases of the nose, some of which are best explained by grouping them around a well-recognized physiopathological law, namely: *When the drainage and ventilation of a mucous membrane-lined cavity is impaired or blocked, the conditions are favorable for the growth of pathogenic bacteria.*

If this is true, each case of inflammatory disease of the nose and accessory sinuses should be examined to ascertain if the drainage and ventilation of these spaces are impaired or blocked. If they are, the obvious therapeutic duty is to remove the obstruction by such remedial measures as will best accomplish the purpose. These measures may be either medicinal, hygienic, or surgical.

If, on the contrary, no obstructive lesion is found, other causes for the lowered resistance of the tissue should be sought for. If the inflammation is a primary acute one and the lowered resistance is due to shock from exposure, it may be useless to attempt to remove the cause, as it is transient. The immediate duty in such a case is to promote the reaction of inflammation and thus check the inflammatory process. As Adami so aptly says, the way to cure inflammation is to increase it.

In order to logically approach the consideration of the causes of the lowered resistance of the mucous membrane of the nose and accessory sinuses, they should be divided into two groups, namely:

(*a*) Extranasal.

(*b*) Intranasal.

Extranasal Predisposing Causes.—*Age* seems to exert some influence upon the resistance of the nasal mucous membrane. Young children and young adults are more frequently subject to inflammatory diseases of the nose and accessory sinuses than those of more advanced years. This is, no doubt, due in part to indiscretion, as improper habits, and insufficient protection of the body from the inclemencies of the weather. Persons of more mature years have more mature minds and better judgment and do not expose themselves needlessly, as in youth and childhood. Then, too, the tissues acquire resistance, or immunity to the noxious irritations.

Sex, perhaps, exerts some influence on the occurrence of inflammatory processes. Males are more exposed and more reckless than females, hence they are more often affected by inflammatory diseases. They are more pugilistic, and often have broken noses and consequent nasal obstruction.

Climate undoubtedly influences the occurrence of inflammatory processes. In regions where there is much cold, wet weather with sudden changes of temperature and of hygroscopic conditions of the atmosphere, it is more difficult to protect the body, particularly the feet, from the

shock incident to such exposures. The shock thus sustained by the vasomotor nervous system leads to a lowered resistance of the mucous membranes, especially of the nose and accessory sinuses, hence the growth of bacteria in these regions is favored.

Exposure, especially unusual or unequal exposure of the body to damp, cold, or other atmospheric and metallurgic conditions, weakens the resistance of the tissues. The exposure of the feet to damp and cold is a most fruitful source of rhinitis and inflammations elsewhere in the body. Draughts striking a single portion of the body are detrimental to the resistance of the tissues much more than when the whole body is thus exposed. Within certain limitations the exposure of the whole body often has a tonic effect, as all the animal mechanisms of the body are equally and simultaneously stimulated. When partial exposure is experienced, only a portion of the mechanism is stimulated, and an imbalance of the functional processes results; that is, there is confusion and havoc in the cellular activities, the nasal expression of which is often some form of inflammation.

Clothing is an important factor in maintaining or lowering the resistance of the mucous membranes of the upper respiratory tract. Too much is as productive of evil as too little clothing. If too much is worn, the skin is rendered sensitive to slight exposure, and if too little, the body is subjected to continual stress, and exhaustion of the vital forces results. Either condition prepares the soil for the growth of pathogenic bacteria in the respiratory passages. Perhaps the most vulnerable part of the body is the feet, through the soles of which course large bloodvessels. Cold or wet feet is a common cause of acute rhinitis and sinuitis.

The proper selection of underwear is a much mooted question. Wool is advocated by some, while linen or linen mesh is strenuously recommended by others. At the present time, most persons buy cotton for summer and cotton and woollen mixtures for winter wear; not because they believe they are the best, but because they are cheaper. My ideas on the subject are as follows:

Linen absorbs moisture better than either cotton or wool, and is, therefore, better for summer wear. Wool is warmer than either cotton or cotton and wool, and is better for winter wear. Those who perspire easily in winter should wear linen next to the skin. If this does not retain enough body heat, light wool should be worn over the linen underwear. Cotton or cotton and woollen mixtures are perhaps never preferable to wool and linen, and woollen underwear during the winter months. Silk is warm, absorbs perspiration, and is non-irritating to the skin. For persons with sensitive skins, it is, therefore, the ideal material for winter undergarments.

Undergarments should be of medium weight for the winter months, the overgarments being depended upon for extra protection for outdoor wear. If the indoor clothing is too heavy the skin becomes tender and subjects the wearer to shock upon undue exposure when out of doors.

The underclothing and outergarments should, therefore, be selected

for their absorptive, non-irritating, and heat-retaining properties. Hard-and-fast rules cannot be laid down with reference to clothing, as every individual is a law unto himself. The aim should be to so regulate the clothing as to avoid either extreme, since to do otherwise subjects the system to shock, and thus lowers the cellular resistance, and prepares the soil for the growth of microörganisms and inflammation.

The *digestive tract* is regarded by Woakes and Stucky as contributory to inflammatory processes of the upper respiratory tract. In this they are correct. If the processes of digestion and nutrition are imperfectly performed, noxious material enters the vascular lymphatic circulation and thus places unusual stress upon all the fixed and migratory cells of the body. Lowered resistance, therefore, naturally follows.

Certain *constitutional diseases* likewise produce a lowered resistance of the tissues, including the mucous membranes of the nose, accessory sinuses, and ears. Diabetes, syphilis, and all diseases due to faulty metabolism especially affect the tissues of the respiratory tract, and predispose them to infection and inflammation.

Heredity probably has no direct influence in the predisposition to infectious and inflammatory diseases of the nose. Indirectly it may have such an influence. That is, certain anatomical conformations of the nasal chambers may be transmitted from parents to the child and thus establish a predisposition to infection and inflammation.

Adenoids may interfere with the drainage and ventilation of the nose and accessory sinuses, or inflammation focalized in them may lower the resistance of the mucous membranes of the nasal and accessory sinuses, and thus predispose to infection and inflammation. These and other extranasal influences may prepare the soil for the growth of pathogenic bacteria in the nose and accessory sinuses and result in inflammation of the sinuses without obstructive lesions in the nose. Whatever the cause of the lowered resistance of the mucous membrane, the result is the same.

I do not wish to be understood as saying that infection and inflammation always follow a lowered resistance of the nasal mucous membrane. I only claim that a lowered resistance predisposes to such a process. The virulence of the microörganisms and other conditions enter into the equation.

Intranasal Predisposing Causes.—I wish to repeat the physiopathological law which largely explains the occurrence of infection and inflammation of the nose and accessory sinuses, namely: *Cavities lined with mucous membrane are predisposed to inflammation when their drainage and ventilation are obstructed.*

We know that when such obstructions have been present and are removed, either by local applications or by surgical interference, relief often promptly follows.

Let us direct our attention, therefore, to some of the obstructive lesions of the nose which predispose the mucous membrane to infection and inflammation.

Obstruction of the Lower Portion of the Nose.—I desire to first call attention to a fact that has long impressed me as very important, namely,

that obstructions in the lower portion of the nasal cavity have a different clinical significance than those located higher in the nasal passages. I also wish to call attention to the clinical significance of anterior obstructions as contrasted with obstructions otherwise located.

Obstruction of the inferior portion of the nasal passage causes an approximation or an impingement of the inferior turbinal against the septum at certain points. The pressure may be either intermittent or constant. The question of greatest importance is, How does such an obstruction affect the drainage and ventilation of the nose and sinuses? As most of the mucous membrane of the nose and sinuses is located above the inferior turbinal, it is obvious that ventilation is but little affected by such an obstruction. The pathway of the inspiratory current is largely limited to the middle and superior meatuses of the nose, and, inasmuch as an obstruction located inferiorly does not materially occlude the inspiratory tract, there is comparatively little disturbance of function. Furthermore, the drainage of the secretions is not materially blocked. The usual obstructive lesion in this region is a spur or ridge on the septum. The ridge is rarely equally prominent along its entire length. On the contrary, it presents one or two prominent spines or knuckles which approximate or impinge against the inferior turbinated body, thus leaving wide gaps through which the secretions may drain to the floor of the nose without marked impediment.

The practical deduction to be drawn from these facts is, that an obstruction in the lower portion of the nose does not markedly reduce the resistance of the mucous membrane, especially in the upper portion of the nasal chambers and in the accessory sinuses. It does, however, have some influence in this direction, and in a degree predisposes to infection and inflammation. The crests of the spines or knuckles may accumulate secretions, which become desiccated in the form of moist or dry crusts. The tissue cells beneath the crusts are injured and their resistance lowered, and to this extent there is a predisposition to infection and inflammation. Furthermore, the impingement of the spur or ridge against the outer wall of the nose causes traumatic injury and results in some degree of lowered resistance, which may lead to bacterial infection and inflammation.

Obstructive lesions in the lower portion of the nose, therefore, may cause a turgescence of the mucous membrane, which is richly supplied with erectile tissue (the "swell bodies"), which after a more or less prolonged period may result in hypertrophy. In the early or turgescent stage the condition is called turgescent rhinitis; in the later stage it is called hypertrophic rhinitis. If, however, repeated infection occurs, the irritation is of a different type and causes hyperplastic changes.

Unfortunately, however, a deviation of the lower portion of the septum is usually accompanied by a deviation of the upper portion in the region of the middle turbinal. When this is the case the type of inflammation is radically different from that present in an uncomplicated lower deviation. That is, a deviation in the region of the middle turbinate often obstructs the drainage and ventilation of the superior meatus and

of all, or nearly all, of the nasal accessory sinuses. The secretions are retained, undergo decomposition, liberate a ferment, and irritate the mucous membranes. In brief, the inflammation is attended by the proliferation of the least differentiated of the fixed cells or connective-tissue cells. In other words, hyperplasia of the mucous membrane occurs. This is known as hyperplastic rhinitis. The irritation in the middle turbinal region may extend by continuity of tissue to the inferior turbinate and cause hyperplasia of this structure as well. Hence, hyperplastic rhinitis often involves both turbinated bodies. In simple deviations, however, limited to the lower portion of the nasal chambers, the inflammation is usually of the hypertrophic type.

Obstruction of the Anterior Portion of the Nose.—Deviation of the anterior portion of the septum from traumatism is a common cause of obstruction of the anterior portion of the nasal chamber. The relationship it bears to inflammatory processes of the nose and accessory sinuses is interesting and instructive. An anterior deviation does not interfere with the drainage of the secretions except in so far as it may interfere with the mechanical force of the respiratory currents of air. The mechanical force of the inspired air is especially manifested in the region of the infundibulum and posterior ethmoidal cells where the inspiratory current sweeps over the hiatus semilunaris and the ostei of the posterior ethmoidal cells and causes slight rarefaction of the air within the sinuses drained by these openings. The mechanical impact facilitates the flow of secretions from the ostei and hiatus semilunaris, and thus prevents desiccation and stoppage of these openings. To this extent obstructive anterior deviations of the septum interfere with drainage.

The ventilation upon the obstructed side, however, is very materially affected. The slight interference with the flow of the secretions caused by the absence of the mechanical impact of air results in a moderate retention of secretions. Decomposition of the secretions may, therefore, take place and cause a lowered resistance of the mucous membrane, and thus establish a predisposition to infection and inflammation.

When the ridge or spur in the lower portion of the nose extends well forward into the vestibule, it also interferes with the ventilation and drainage, as described in the preceding paragraph.

When either type of anterior obstructive deviation is present, another and more important etiological factor must be taken into consideration, namely, the rarefaction of air posterior to the obstruction. Air being unable to enter the nostrils rapidly enough during the descent of the diaphragm is rarefied, or a state of negative air pressure is established. This, according to Bier's theory, should prevent serious inflammatory processes, as the negative air pressure thus produced promotes the reaction of inflammation and should prevent serious inflammatory disease. Doubtless the negative pressure thus automatically produced does exert a favorable influence upon the inflammatory process excited by the lack of ventilation and the slight retention of the secretions. Thus, strange as it may seem, the anterior obstructive lesion predisposes to infection and inflammation, and at the same time tends to cure it.

Clinically, I have often noted the comparatively slight inflammatory disease of the nasal mucous membranes which is present in cases of simple anterior deviations.

The chief departure from the normal is a turgescence or an hypertrophy of the inferior turbinates. Little pathological change is present in the middle turbinate region unless there is an associated obstruction in that location. The negative air pressure easily accounts for the turgescence of the erectile tissue of the inferior turbinates. After a prolonged duration of the turgescence, whether intermittent, alternating, or constant, hypertrophy occurs as a result of the increased nutrition.

Obstruction in the Middle Turbinal Region.—Obstruction in this portion of the nasal chambers is productive of more serious inflammatory disease of the nose and accessory sinuses than obstruction in any other portion of the nose. The reason is obvious when we recall the fact that the ostei of the posterior ethmoidal and sphenoidal sinuses drains into the superior meatus above the middle turbinate, while the frontal, anterior ethmoidal, and maxillary sinuses drain into the middle meatus beneath the middle turbinate.

If the septum is deviated so as to press against or approximate near to the middle turbinate, the olfactory fissure is blocked and the drainage of the posterior ethmoidal, and possibly of the sphenoidal, cells is interfered with.

Clinically, I have noted the presence of two types of deviations of the septum that close, or nearly close, the olfactory fissure. One is a bowing of the perpendicular plate of the ethmoid bone and quadrilateral cartilage, and the other is a thickening of the septum in the region of the middle turbinated body. The bowed septum is thin and easily corrected by the submucous resection of the septum, whereas the thickened septum often involves only the mucous membrane and is more difficult to correct.

In some subjects there are large ethmoidal cells in the middle turbinate, which may either close a part or all of the olfactory fissure or they may encroach upon the hiatus semilunaris beneath it. In the first instance the drainage and ventilation of the superior meatus of the nose, and in the second instance the drainage and ventilation of the frontal, anterior ethmoidal, and maxillary sinuses is impaired.

A large bulla ethmoidalis projecting medianward and downward may obstruct the hiatus semilunaris, and thus obstruct the drainage and ventilation of the cells draining into the infundibulum, namely, the frontal, anterior ethmoidal, and maxillary sinuses.

Likewise, the occasional presence of cells in the inner wall of the infundibulum, or uncinate process of the ethmoid bone, may block the infundibulum and cause serious inflammatory disease of the frontal and anterior ethmoidal cells and the maxillary antrum ("vicious circle").

In about 50 per cent. of the cases the frontonasal canal does not communicate with the infundibulum, but opens directly into the middle meatus more anteriorly (Logan Turner). In these subjects an enlarged projecting bulla ethmoidalis and cells in the uncinate process would not

block the drainage and ventilation of the cells draining through the frontonasal canal, namely, the frontal and anterior ethmoidal cells. The ostium of the antrum, however, may be obstructed, as it always opens into the infundibulum.

Results of High Obstructions in the Nose.—When the olfactory fissure is obstructed by either septal or turbinal deformity, drainage of the secretions and ventilation of the posterior ethmoidal and sphenoidal sinuses are impaired. The secretions are retained and undergo retrograde changes. The mucous membrane bathed in the secretions is injured and its functional activity and resistance are lowered. The biochemical substances liberated in the process of decomposition constantly irritate the mucous membrane, especially of the middle turbinated body. Acute infection occasionally occurs. During the intervals between the acute inflammatory processes a mild staphylococcal or other infectious inflammation persists. Under these conditions there is a proliferation of fixed cells in the tissues, usually the least differentiated or connective-tissue cells.

The result is known as hyperplastic rhinitis, which chiefly involves the middle turbinated body and ethmoidal cells, though it may extend to the inferior turbinal.

Obstruction of the Olfactory Fissure.—The partial or complete closure of the olfactory fissure and the consequent retention of the secretions of the superior meatus, and the ethmoidal and sphenoidal sinuses draining into it, cause hyperplastic changes in the mucous membrane, not alone of the middle turbinate, but of the superior meatus and of the ethmoidal and sphenoidal sinuses opening into it. The conditions thus produced favor infection and inflammation. The inflammatory process may be either catarrhal, purulent, fibrinous, or phlegmonous in type, and in each instance the active causes are pathogenic microörganisms.

The sinuitis thus excited may continue for years without engaging the attention of either the patient or physician. Headache and slight dizziness, aggravated upon stooping, may be the only symptoms complained of, except, possibly, recurrent attacks of acute coryza. Or the sinuitis may be distinctly and frankly purulent, with copious discharge into the epipharynx, and possibly to some extent through the olfactory fissure into the middle meatus.

Atrophic rhinitis with ozena in adults is, in my opinion, often a result of suppurative sinuitis. Space does not permit of a full discussion of this phase of the subject. Personally, I have repeatedly overcome the ozenic secretion by treating the case as though it were a suppurative sinuitis. I have made skiagraphs of several cases of atrophic rhinitis with ozena, and without exception they have shown the existence of sinus disease. This does not, of course, determine which was primary, the atrophic rhinitis or the sinuitis. My opinion is largely based upon the results following the treatment for the sinuitis.

Obstruction Due to the Bulla Ethmoidalis, the Middle Turbinate, and Uncinate Cells.—As previously stated, a large bulla ethmoidalis may occlude the infundibulum and thus block the drainage and ventilation

of the maxillary sinus, the frontal and anterior ethmoidal cells. This, as heretofore explained, causes the retention of the secretions and lowered resistance of the tissue, thus establishing a predisposition to infection and inflammation. (See "Vicious Circle" of the Nose.)

Cells in the middle turbinated body and uncinate process may likewise block the infundibulum and cause similar results. The exception has been referred to wherein the frontonasal canal opens directly into the middle meatus anterior to the infundibulum.

It appears, therefore, that there are several factors entering into the causation of inflammatory diseases of the nose and accessory sinuses. The exciting causes are nearly always pathogenic microörganisms, while the predisposing causes are numerous extranasal influences which are often combined with obstructive lesions in the nose. The latter should always be studied with reference to whether they interfere with the drainage and ventilation of the nose and accessory sinuses. If only extranasal causes of lowered resistance are found, the treatment should be addressed to their removal; and if in addition to the extranasal influences obstructive lesions are discovered, they should be corrected by probing or by surgical interference.

Conclusions.—1. Acute inflammation is usually a threefold reaction excited by pathogenic bacteria and their toxins, namely:

(*a*) Increased hyperemia.

(*b*) Increased nutrition of the tissues.

(*c*) Increased migration of leukocytes.

The reaction of acute inflammation is the response of Nature's forces for the purpose of destroying the bacteria and their toxins.

2. The reaction of inflammation is usually incapable of removing quickly the infective bacteria and their toxins, hence the inflammation continues for several days, or it may be indefinitely prolonged.

3. Chronic inflammation consists of the same reactions in much less degree, and is still further characterized by the proliferation of fixed cells into the tissues, notably connective-tissue cells.

4. The exciting causes of inflammation are pathogenic microörganisms.

5. Pathogenic bacteria do not *per se* cause inflammation. There must be a lowered resistance of the tissues before they will rapidly multiply and produce inflammation.

6. Anything that lowers the vitality or resistance of the mucous membrane of the nose and accessory sinuses predisposes it to infection and inflammation.

7. The extranasal influences that lower the vitality of the mucous membrane are sex, climate, exposure, improper clothing, digestive disorders, constitutional diseases and dyscrasias, hereditary anatomical peculiarities of the framework of the nose, adenoids, etc.

8. The intranasal predisposing causes of inflammation of the mucous membrane of the nose and accessory sinuses are, perhaps, best explained by the well-recognized law: *Obstruction of the drainage and ventilation of mucous membrane-lined cavities predispose them to infection and inflammation.* The character of the inflammation and the final result are

partially determined by the location of the obstruction in reference to the various structures of the nose and to the accessory sinuses.

9. Anterior and inferior obstructions are the usual cause of turgescent and hypertrophic rhinitis. Obstructions in these lesions do not cause hyperplastic rhinitis, because they do not materially interfere with the drainage of the secretions, and therefore cause little or no irritation.

10. Obstruction higher in the nose, in the region of the middle turbinate and the infundibulum, causes the retention of the secretions and interferes with the ventilation of the superior meatus and the accessory sinuses, thus lowering the resistance of the tissues and establishing a marked predisposition to infection and inflammation of the nasal and accessory sinuses. The inflammation may be catarrhal or suppurative, acute or chronic in type.

11. The long-continued mild irritation excited by obstructive lesions in the middle turbinal region often results in hyperplastic rhinitis, which may be limited to the middle turbinate, though it may extend to the inferior turbinate.

12. Inflammation also extends to adjacent parts by the continuity of tissue, hence it may extend from one part of the nasal mucous membrane to another, or it may extend from the nasal mucous membrane to the sinuses, the Eustachian tube, and cavum tympani.

CHAPTER VII

THE METHODS FOR PROMOTING THE REACTION OF INFLAMMATION

In the preceding chapter I have shown that acute inflammation is a series of reactions excited by the presence of bacteria, their toxins, and the cellular debris. The object of the reactions is to rid the tissues of these substances. Experience has shown that in acute inflammation the reaction is not sufficient to do this as quickly as should be to prevent damage to the tissues. That is, necrosis, infiltration, and adhesive processes are likely to occur before the reaction removes these irritants from the tissues. It is rational therapy, therefore, to promote the inflammatory reaction rather than to repress it. As a concrete example, I will cite acute coryza, or "cold in the head." This is a reaction due to certain bacteria and their toxins. It is understood, of course, that certain predisposing causes have prepared the soil for the growth of the bacteria. Ordinarily, the reaction (increased hyperemia and leukocytosis) is inadequate to throw off quickly the bacteria and their toxins. The question naturally arises, How to promote and increase the reaction? Do not make the common mistake of assuming that the inflammatory reaction is already excessive; it may be, but it is usually inadequate. Those who assume the reaction to be excessive often apply adrenalin locally to reduce the reaction. This reduces the hyperemia, cell nutrition, and leukocytosis, whereas they should be increased. It does, however, establish better drainage, and to this extent acts favorably.

The same law applies to nearly all acute inflammations of the upper respiratory tract, including the ear. It is the purpose of this chapter to discuss the various procedures whereby the reaction of inflammation is promoted or increased, and to outline the indications and the methods for their therapeutic application.

Counterirritation.—Counterirritation has long been used to counteract inflammatory processes, the prevailing idea being that it diverted the blood to the surface and away from the seat of inflammation. We know now that while its use was rational, the explanation of its good effects was irrational. Counterirritation applied over the inflamed area not only increases the superficial hyperemia, but it increases it in the deeper tissues as well. It also increases the leukocytosis and cell nutrition. Thus, instead of diminishing the inflammation, it promotes the inflammatory reaction.

Counterirritation has but little place in otolaryngological practice for

two reasons: (1) Because the blistering and scarring which occasionally result are objectionable for cosmetic reasons, and should surgical interference become necessary the skin is in bad condition, and (2) because more efficacious methods may be employed.

Poulticing.—This is also an old method of treating inflammation. The moist poultice of bread and milk, or other ingredients, is usually applied hot, the whole being covered with cloths or oiled silk to retain the heat and moisture. While poulticing promotes inflammatory reaction, it has fallen into disuse, because better procedures have taken its place.

Scarification and Wet Cupping; Artificial Leeching.—Scarifiers were once a part of every family physician's outfit, whereas they are now rarely seen. Scarification was usually combined with cupping, and was designated "wet cupping." With a comb-like knife or with a series of concealed blades liberated by pressing a spring, the superficial layers of the skin were many times incised, and a cup in which a few drops of alcohol or a piece of paper was burned was quickly applied over the incised surface, and the negative air pressure created by the heat in the cup caused free oozing of blood. The idea prevailed that this diminished the excessive inflammatory reaction, whereas, as a matter of fact, it increased it. That is, it increased the hyperemia and leukocytosis, established adequate reaction, and hastened the elimination of the bacteria, toxins, and cellular detritus.

Wet cupping was formerly much practised in cases of acute mastoiditis, and doubtless with beneficial results, though leeching is a much better means for relieving the condition.

Leeching.—This is an old therapeutic measure of great value in promoting inflammatory reaction. I have seen children with bronchopneumonia quickly pass from a state of stupefaction, with a pulse of 200 per minute, to one of complete consciousness, with quiet respiration and a pulse of 100 per minute, after the application of a few leeches to the chest. Likewise, I have seen the pain and tenderness in acute mastoiditis subside under leeching. With the improved technique of mastoid surgery, and with the accumulated observations of aural surgeons to the effect that, while many of the cases of acute mastoiditis subsided, but few were cured; hence, leeching and kindred measures have been gradually abandoned. The keynote to the present-day mastoid therapy is the total eradication of the diseased process at the earliest possible moment by surgical intervention. Doubtless the pendulum has swung too far to the surgical side. An increased knowledge of the pathology of inflammation and of the processes of repair will enable the surgeon to differentiate more closely between the operative and non-operative cases.

From three to six leeches may be applied over the mastoid process and in front of the tragus in the early stages of acute mastoiditis with decidedly beneficial effect. This is good treatment while watching the development of a case, and in some instances it promotes the inflammatory reaction (increased hyperemia and leukocytosis) to such

a degree as to lead to a speedy recovery. It is doubtful if leeching is efficacious after the disease has continued several days. Even then, however, it will affect the inflammatory process favorably. The case must then be treated surgically (removal of adenoids in children, and possibly the exenteration of the ethmoidal sinuses in adults, or a mastoid operation) or it may assume a latent or chronic form.

Irrigation or Lavage. —This mode of treatment has long been applied to inflamed nasal chambers and accessory sinuses of the nose. The prevalent idea as to its mode of action is that the solution used mechanically removes the inflammatory secretion, and thus lessens the noxa or local irritant, all of which is doubtless true. It also increases the local hyperemia and migration of leukocytes, *i. e.*, promotes the inflammatory reaction. Its action, however, is usually slight and transient, and inadequate for the purpose. The inflammatory process passes into the chronic type with tissue deposit, thus causing permanent changes detrimental to the physiological integrity of the structures. There are circumstances, however, under which lavage must be used in the treatment of sinuitis. If for any reason operation is refused or is not advisable, lavage may be practised through the ostia or through artificial openings into the sinuses. In acute cases the reaction thus established quickly overcomes the noxa, and healing speedily results. In chronic cases the reaction thus promoted is inadequate, and, indeed, in the nature of things is not calculated to arrest the noxious process. Chronic inflammation consists of hyperemia, slight exudation, slight migration of leukocytes, and great tissue proliferation. The last-named process is probably not to be checked by any direct means we can employ.

From the foregoing it is plainly good treatment to employ such solutions by irrigation as will increase the hyperemia, the migration of leukocytes, and the nutrition of the chronically inflamed mucous membrane. To these ends normal salt, boric acid, mild iodine, and other solutions may be employed. It is to be expected, therefore, that while lavage will not remove the tissue proliferation, it will promote the inflammatory reaction, and in a measure remove the infective noxa still remaining. It also removes the irritating toxic secretions, and thus relieves the tissues of another source of irritation.

Massage. —Under this term are included three methods of treatment, namely: (*a*) Manual massage, (*b*) mechanical massage, and (*c*) alternate rarefaction and condensation of air in a cavity, the so-called pneumomassage as devised by Delstanche and as modified in the various mechanically driven machines so commonly used in America.

The effect of massage upon inflamed tissue is to increase the hyperemia and nutrition, and the diapedesis of leukocytes. The inflammatory reaction is thereby promoted and the tissues measurably relieved of the irritant noxa.

(*a*) Massage of the larynx in acute laryngitis and for the relief of singers' nodules has been used with decided benefit. It may be applied by hand manipulations or by a vibratory massage machine. The motion and physical force thus applied to the exterior of the larynx increases the

hyperemia, nutrition, and leukocytosis of the parts, and thus aids in the removal of bacterial infection.

(*b*) Mechanical or vibratory massage is of special value in acute adenitis of the cervical glands, and its application quickly reduces the swelling and tenderness. It is not good treatment, however, to limit the attention to this mode of procedure, for to do so is to ignore the primary source of the glandular disease, namely, the tonsils, adenoids, and pharyngeal glands. The massage is only an adjunct treatment.

(*c*) Pneumomassage by means of hand or mechanically driven devices has been used extensively and almost empirically for the relief of deafness and tinnitus, with but little result. The same procedure applied in cases of acute otitis media with an exudative secretion would promote the absorption of the exudate and prevent adhesive processes. That it has been used for this purpose I am unprepared to say. It is reasonable, however, to suppose that the movements thus imparted to the membrana tympani and the ossicular chain would increase the hyperemia, the cell nutrition, and the migration of the leukocytes in the inflamed mucous membrane, and thus hasten the reparative process.

Leukodescent Light.—During the past few years radiant energy in the form of light from a 500 candle-power incandescent globe has been used in the treatment of inflammatory processes (Fig. 100). The beneficial effects are, perhaps, best explained by saying that this treatment promotes inflammatory reaction (hyperemia, cell nutrition, and diapedesis of leukocytes), and thus hastens the removal of the bacteria and other noxious material. I have made use of the light for about five years, and have found it one of the most useful, if not the most useful, mechanical agency for promoting reaction in inflammatory diseases of the upper respiratory tract. Acute coryza is sometimes cured under its influence. I have repeatedly seen chronic suppurative sinuitis become painless and cease to discharge purulent secretions into the nose when this form of treatment was used. I have never cured such a case by its use, for the purulent discharge reappeared in a few days or weeks after discontinuing the treatment. Whether its prolonged use would have effected a cure I am not prepared to state. The rays of light relieve pain, tenderness, and swelling in an astonishingly short time, and superficial infections sometimes disappear rapidly. This is not surprising in view of our knowledge of radiant energy from the Finsen light, the Röntgen rays, and the high-frequency electrical currents. The 500 candle-power lamp is known to possess high chemical and penetrating properties. In addition to this the heat rays are, of themselves, of great usefulness in promoting inflammatory reactions. The combination of the chemical and heat rays is ideal in the treatment of inflammatory diseases, as the reaction is more profound than that which results from either the heat or the chemical rays alone. The range of application of the 500 candle-power lamp is as wide as inflammation itself. It will not cure all cases, but if the reaction is inadequate, it will be of benefit in so far as it promotes reaction. If the reaction is excessive its use is contraindicated, and cold applications should be made.

If the reaction is adequate, as in cases of incised wounds which heal naturally, its use is contraindicated. It should be remembered that the inflammatory reaction usually reaches its maximum of efficiency at the end of about twenty-four hours, and that to get the maximum results by any of the treatments referred to in this section, they should be applied within the first twenty-four hours, before tissue proliferation begins. Tissue

FIG. 100

The leukodescent light should be swung back and forth over the face at a distance of about twelve inches for from fifteen to thirty minutes at each treatment.

proliferation of a permanent type begins at about the fifth day of acute inflammation, and becomes more and more established as time goes on.

The failure of the leukodescent light to cure chronic inflammation is explained by the well-known fact that tissue proliferation is a manifestation of chronic inflammation, and that chronic inflammation is not readily checked by any direct mechanical means at our command,

except by the most thorough exenteration of all the diseased tissue and the establishment of free drainage and ventilation.

Bier's Treatment.—Bier's treatment has attracted a great deal of attention within the last few years. It is based upon the promotion of hyperemia in the treatment of acute suppurative, tuberculous, and other conditions. He promotes both active and passive hyperemia; active by the use of hot air, and passive by constriction of the parts and by negative air pressure in cavities. He finds active hyperemia of more value in chronic cases, where proliferative tissue is to be absorbed. He also finds it useful in acute cases, but not so useful as passive hyperemia induced by compression so applied as to obstruct temporarily the efferent veins of a part, without arresting the entry of blood through the afferent arteries. He also supplies suction by cupping over small inflamed areas, and by large glass chambers into which the affected part, as the hand or foot, may be introduced and the surrounding air rarefied.

Sondermann has devised an apparatus especially adapted for producing negative air pressure in the air cavities of the head. Brawley, Dabney, and Pynchon have also devised apparatuses for this purpose.

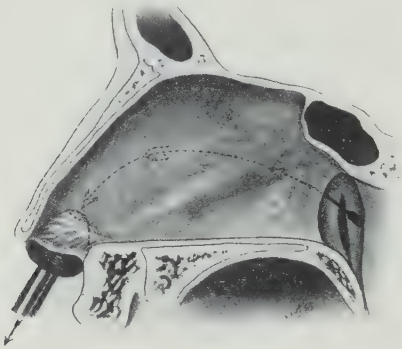
Bier's treatment is applicable to those cases of acute inflammation in which the inflammatory reaction is inadequate to cope with the irritant noxa causing the inflammation. The treatment should not be applied so as to produce excessive reaction (white edema) of the tissues. It should never cause pain. It must not produce paresthesia or false sensation. In the nasal chambers it should not be prolonged for more than one-half to one hour at a time. The mode of treatment requires great caution in its use, as much harm can be done with it. If white edema is induced, the bacteria spread through the tissues and the process becomes more generalized.

Inflammation is not yet fully understood, and until it is, cases cannot be individualized for treatment. Wright's demonstration of *antitropins*, *precipitins*, lysins, and opsonins in the blood, and that the opsonins are of greater importance than the leukocytes, as the latter are dependent upon the former for their efficiency, has disturbed existing ideas to such an extent that there is a "shuffling of dry bones" in the scientific world. It appears that the leukocytes cannot digest or neutralize the bacteria until the latter have been acted upon, weakened, or rendered vulnerable by the opsonins. These researches show that Bier's method of inducing hyperemia does not simply flush out the inflamed area, but that the supply of leukocytes and antitropins causes a rapid removal of the dead bacteria from the field of action through the energized leukocytes (Adami). It appears, therefore, that the opsonic index is of even greater importance than the leukocytic index. Should the leukocytosis be marked and the opsonins scanty, the bactericidal and scavengerial properties of the leukocytes would be greatly impaired, and the reaction, while apparently adequate according to the older standard, would be inadequate according to the newer standard of the opsinins. However this may be, further observations are necessary before the older standard is abandoned for clinical purposes.

Technique.—In acute inflammatory diseases of the nose and accessory sinuses negative air pressure produced by the Sondermann, the Brawley, or the Dabney-Pynchon devices may be obtained as follows:

(*a*) Introduce the nasal tip or tips into the anterior naris, turn on the exhaust power (hand bulb, water, or compressed air, according to the apparatus used), and instruct the patient to swallow. This brings the soft palate in contact with the posterior wall of the pharynx and closes the communication between the epipharynx and the mesopharynx. The air in the nose and accessory sinuses and the Eustachian tubes is rarefied, and hyperemia of the mucous membrane results. After a little practice the patient is able to maintain the state of negative pressure for several minutes at a time (Fig. 101).

Fig. 101



Showing the soft palate closed during suction through the nose.

(*b*) The negative pressure should be alternated every three to five minutes with periods of rest, the whole period of treatment extending over fifteen to forty-five minutes.

(*c*) If the treatment is attended by pain, bleeding, or white edematous swelling, the negative pressure is too great and should be reduced. Heat in the form of hot air is indicated to counteract the white edematous swelling should it occur.

(*d*) The nose-piece should be patterned after the Seigel otoscope, so that the mucous membrane may be inspected during the course of application of the negative air pressure, and if the membrane becomes pale and edematous, or bleeds, the treatment should be abandoned for twenty-four hours; that is, paralysis instead of dilatation of the vessels has occurred, and the nutrition of the cell structures and the local leukocytosis have been still further diminished. The method of treatment, therefore, requires the greatest care and intelligent application to be beneficial. Its careless and indiscriminate use can only produce harmful effects. The greatest objection to the mode of treatment is the ease of application and readiness with which great harm can be done with it.

Indications.—It should be used: (*a*) In the first five days of acute rhinitis. (*b*) In the first five days of acute sinuitis. (*c*) In the first five

days of acute inflammation of the pharyngeal tonsil. (*d*) In acute tubal catarrh. (*e*) In chronic purulent inflammation of the sinuses. In all cases the negative air pressure should be very moderate, as otherwise it will produce edema and white swelling and "add fuel to the flames." Its greatest efficiency will be found in acute inflammation. In chronic inflammation, either catarrhal or suppurative, heat in the form of hot air is a more rational mode of treatment, as it produces an active hyperemia and increases the cell nutrition. The negative pressure produces a passive hyperemia and leukocytic migration, processes much needed to promote speedy resolution of the inflammatory process.

(*e*) When purulent secretions are present, they are drawn into the bottle reservoir of the apparatus. In these cases the negative air pressure not only promotes the inflammatory reaction, but it removes the irritating secretions as well.

(*f*) The treatment should be repeated every day or every other day.

CHAPTER VIII

INFLAMMATORY DISEASES OF THE NOSE

ACUTE RHINITIS COMPLICATING SPECIFIC FEVERS AND CONSTITUTIONAL DYSCRASIAS

The initial stage of the various exanthematous or specific fevers is characterized by an attack of acute rhinitis. Certain constitutional dyscrasias also give rise to it. The infectious or exanthematous fevers, commonly characterized by an attack of acute rhinitis, are smallpox, typhoid fever, acute articular rheumatism, epidemic influenza (la grippe), erysipelas, measles, and diphtheria.

The symptoms of all the foregoing types of specific acute rhinitis are about the same, except in diphtheria, in which case a pseudomembrane may be present. The usual manifestations found in coryza with conjunctivitis and photophobia are present. An examination of the mucous membrane of the nose and fauces sometimes shows an eruption quite similar to that found on the skin.

The treatment should consist in the use of mild alkaline solutions with an atomizer or a nasal douche. The objection to the douche is the possibility of carrying the infection to the middle ear should the patient happen to swallow while the fluid is in the nose. The nose should be irrigated three or four times daily.

The constitutional dyscrasias which cause acute rhinitis are diabetes mellitus and scorbutus. In diabetic rhinitis the symptoms when present rise and fall with the percentage of sugar in the urine. Scorbutic rhinitis is associated with infantile scurvy, and is characterized by an excoriation about the nasal orifice.

The *treatment* should be addressed to the relief of the local nasal symptoms and to the improvement of the constitutional dyscrasias.

ACUTE RHINITIS

Synonyms.—Acute coryza; cold in the head.

Definition.—Acute rhinitis is an acute inflammation of the mucous membrane of the nose and accessory sinuses, characterized by chilly sensations, lassitude, nasal discharge, and a swelling of the mucous membrane of the nose. The patient also complains of a stuffiness of the nose and of sneezing.

Etiology.—The chief predisposing cause of acute rhinitis in adults is an obstructive lesion of the nasal septum, which predisposes to the local

growth of the pathogenic bacteria and the development of their toxins, hence the inflammatory reaction in the form of an acute rhinitis. The ridge or other deviation of the septum impinges upon, or is closely approximated to, the inferior nasal concha (inferior turbinated body), thus interfering with drainage and ventilation of the nose and accessory sinuses. When the anterior portion of the septum is thus deformed it obstructs the breathway, and each descent of the diaphragm acts like the piston valve of a syringe and rarefies the air in the nasal chamber posterior to the obstruction. The negative pressure thus created causes the blood to fill the vascular tissue of the "swell bodies" on the inferior and middle turbinals, hence the stuffiness of the nostrils. Furthermore, the mechanical irritation caused by the pressure of the ridge or other deviation against the turbinals still further aggravates the irritation and swelling of the mucous membrane. The secretions are thereby increased in quantity and changed in character.

Inquiry usually elicits the statement that the patient (if an adult) has been inclined to chronic rhinitis; indeed, a complete examination often shows the patient to have been subject to acute exacerbations of a chronic rhinitis, and that a septal deformity is present. Septal deformity is not, however, always present, hence each case should be studied for its peculiar etiological factors, so that the treatment for the ultimate cure and prevention of the acute exacerbations may be intelligently directed.

Another very common cause of acute rhinitis is a disturbance in the vasomotor nervous system. There is a paralysis of the vasoconstrictor muscle fibers of the capillaries, or an irritant in the blood which affects the dilator fibers.

The paresis and irritation may be due to the presence of uric acid and its kindred products or to other acquired dyscrasia. The lack of balance of the vasomotor nervous system may also be due to the inadequate ventilation of the living and sleeping rooms, offices, etc., or to the wearing of improper clothing. The removal from the country to the city is often followed by frequent attacks of acute rhinitis on account of the changed conditions of living. In the country the houses are less tightly constructed and but partially heated, whereas in the city the houses are more tightly constructed and either overheated or, as is often the case, are underheated in all rooms. In either case the conditions are less healthful in the city dwelling because fresh oxygen is a negligible quantity on account of the poor ventilation. Then, too, residents of the country spend much of the day in the open air, whereas those in the city spend much of the time in crowded and illy ventilated offices and shops. It is obvious, therefore, that rhinitis due to poor ventilation should be treated by changing the mode of living to one which keeps the patient in the open air or in a well-ventilated residence and business building.

The causative relationship of clothing to acute rhinitis is unquestioned, though it is difficult to describe the exact mode of clothing that predisposes to rhinitis. It may be said, however, that clothing which promotes perspiration is pernicious. There is normally some evaporation of

moisture from the body, hence the underwear should be of such material as to absorb it readily. The function of underwear is twofold, namely: (*a*) To retain the body heat between it and the skin; (*b*) to absorb the excess of perspiration. If, therefore, the clothing is of such density that it causes undue perspiration, and of such material that it does not absorb it, the conditions are favorable for the development of acute rhinitis, even though the septum is normal. Wool retains the body heat, but is a poor absorbent. Cotton is neither a good heat retainer nor an absorbent. Linen is a fair heat retainer and a good absorbent. In some cases wool retains too much heat and induces profuse perspiration. A garment of wool and cotton, or wool and linen, or of thin linen under a light woollen garment, seems to be suitable to the proper protection of the body. Linen mesh in some cases is insufficient protection during the winter months for some people, whereas it is worn with the greatest comfort and satisfaction by others throughout the year. It should be determined in each case whether the rhinitis is due, in part, at least, to excessive protection and perspiration, or to deficient absorption of the perspiration. Then, too, the question extends to the outer garments worn both indoors and outdoors. For the sake of convenience, the outer garments should be lessened or added to as the exposure to the temperature and weather demands, while the undergarments should be of moderate weight and capable of absorbing the visible and invisible perspiration.

A preëxisting chronic rhinitis is a common factor in the causation of acute rhinitis, especially in adults, whereas infants and young children are more susceptible, and often have colds in the head without a preexisting chronic rhinitis.

As stated in Chapter VI, inflammation is almost always of bacterial origin, the condition necessary for the growth of the bacteria being a lowered vitality of the cells of the tissues. I also stated that mucous membrane-lined cavities with blocked drainage and ventilation were especially subject to infection and inflammation. Trauma, chemical injury, and shock also lower the cell vitality and prepare the soil for infection and inflammation. Exposure to cold and draughts are common sources of shock that result in acute coryza or inflammation of the nasal mucous membrane; hence, obstructive lesions of the nasal septum are not always present in patients subject to acute coryza. Certain constitutional diseases, as diabetes, rheumatism, etc., reduce the vitality of the mucous membrane of the nose and accessory sinuses, and are, therefore, predisposing causes of this disease. All conditions, local and general, which lower the resistance of the mucous membrane of the nose act as predisposing causes to infection and inflammation of the nasal mucous membrane. I wish to emphasize again the fact that in many instances the chief predisposing cause of acute coryza (acute infectious inflammation of the nasal mucous membrane) is an obstructive lesion of the septum. The influence of exposure to cold, draughts, foul air, poor ventilation of houses, offices, etc., have heretofore been given undue prominence, to the neglect of nasal stenosis (partial and

complete), which so often bears an important relation to this disease. It follows that chronic rhinitis is often present in persons subject to recurrent attacks of coryza, a condition which still further lowers the vitality of the membrane and predisposes to the growth of bacteria and the development of their toxins, which excite the inflammatory reaction known as coryza, acute rhinitis, and "cold in the head."

In emphasizing these facts I do not wish to obscure or belittle the other factors that reduce the vitality of the tissues and which predispose to the acute inflammatory disease. I only wish to give a true perspective to the underlying causes of acute coryza, so that in the treatment a more rational basis of procedure may be adopted.

Acute rhinitis undoubtedly has an infectious origin, and the foregoing etiological factors predispose to the infection. Nasal polypi and other morbid processes within the nasal chambers also predispose to rhinitis.

Pathology.—The vasomotor constrictor muscle fibers of the capillaries are paralyzed and the dilator fibers irritated, and, as a consequence, there is a passive hyperemia of the venous capillaries and lymph vessels, and the nose becomes "stuffed." There is also an increased migration of leukocytes and a transudation of lymph and serum. The production of mucus is temporarily checked, but later is increased. The epithelium is exfoliated and admixed with the other inflammatory products and secretions.

During the first stage the secretions are greatly reduced in quantity or are entirely absent. In the second stage the secretions are at first serous, and later become thick and viscid from the excessive degeneration of the goblet and glandular epithelial cells. In the third stage the secretions are mucopurulent or purulent in character.

The duration and course of the inflammatory process varies. The course of the average case is completed in from eight to ten days, though under appropriate treatment it may be greatly shortened.

Symptoms.—The symptoms are, for clinical purposes, divided into three groups, as follows:

First Stage, or Onset.—The patient experiences a sense of dryness or prickling in the nose, with itching at the inner canthi of the eyes. Chilly sensations and a feeling of malaise are complained of. Examination shows the mucosa to be red and hyperemic, but not fully turgescent. The mucous membrane is abnormally dry and free from secretions. Headache is usually present, and there is a sense of fulness between the eyes. This stage lasts but a few hours. The temperature ranges from 100° to 103°.

Second Stage.—This stage is characterized by a profuse serous discharge and turgescence of the mucous membrane. In some cases the headache and the sense of fulness between the eyes are diminished, whereas in others they are increased, depending upon the patency or closure of the ostei of the accessory sinuses. In those cases in which there is a marked deviation of the nasal septum in the region of the middle turbinate the obstruction to drainage on one side may be great and the pain and sense of fulness correspondingly increased on that side.

Third Stage.—This stage is characterized by a mucopurulent or purulent discharge and by a marked decrease in the temperature. The headache and the sense of fulness between the eyes may be diminished to a dull heavy feeling across the forehead and between the eyes. If the nasal accessory sinuses are also markedly involved in the inflammatory process, the frontal headache and the sense of pressure are correspondingly pronounced. If the sinuses are not involved these symptoms may be entirely absent. Dizziness and vertigo also may be present if the sinuses are involved.

The use of the eyes in reading, sewing, or at the theatre often produces headache or other evidence of ocular irritation when the sinuses are involved in acute rhinitis.

Prognosis.—The natural duration of acute rhinitis is from eight to ten days. When the sinuses are extensively involved the duration is extended to two weeks, or even longer, unless the attack is aborted by appropriate treatment. Some writers claim that there is no curative treatment of acute rhinitis. I believe this to be an erroneous view, and hold that nearly all cases may be cured if taken sufficiently early and rational treatment is used.

Treatment.—The treatment of acute rhinitis should be undertaken with a knowledge of the nature of inflammation and the chief predisposing and active etiological factors in mind. These are (*a*) obstructive lesions; (*b*) lowered tonicity of the cellular structures of the nasal mucous membrane, and (*c*) the infectious microörganisms.

(*a*) If there is an obstructive lesion in the nose it should be located by rhinoscopic examination. When found, and demonstrated to be spongy or erectile tissue, local applications of cocaine, adrenalin, and antipyrine should be made to this region to reduce the swelling and to establish the patency of the nasal chambers. By so doing drainage and ventilation are reëstablished, points of immense value in promoting the reaction against bacteria and toxins which cause the disease. It is not advisable to attempt to remove by surgical means the obstructive lesion during the acute symptoms, though such a procedure may well be undertaken after they have subsided. The retention of the secretions and the lack of ventilation, together with the mechanical irritation from pressure, aggravate the existing irritation and tend to perpetuate the reaction of inflammation and prolong the disease. The reaction is often inadequate to throw off the bacteria and their toxins, hence measures should be used that will promote the reaction of inflammation, which is Nature's effort to cure the disease.

The question naturally arises, How may the reaction of inflammation be promoted? That is, what measures may be adopted that will aid in combating the bacteria and their toxins? As stated in the section on Inflammation, acute inflammation consists in three reactions, namely: (*a*) Increased hyperemia, (*b*) increased cell nutrition, and (*c*) increased migration of leukocytes. The purpose of these reactions is (1) to increase the vitality of the attacked tissues, (2) to remove the bacteria and toxins, and (3) to remove the dead and broken-down cells.

The increased hyperemia furnishes extra food for the cells which have been attacked and weakened, while the increased migration of leukocytes provides for the destruction and removal of the invading bacteria and the dead and broken-down cells. Adami has shown that in acute inflammation the inflammatory reaction is usually inadequate for these purposes, although it has generally been thought to be excessive. He advises, therefore, that acute inflammations be treated by such methods as will promote the reaction of inflammation, rather than check it. Formerly, remedies which acted favorably upon acute inflammations were said to lessen the inflammatory reaction, whereas a more correct and scientific statement is, that the remedies promoted the inflammatory reaction (Nature's effort to rid the tissues of bacteria and their toxins) and thereby hastened the cure of the disease. It is with this understanding that I advise the use of such remedial measures as will promote the reaction of inflammation.

The empirical use of drugs has long been practised, and must doubtless continue to be practised until their action is better understood. We know enough about a few of them to criticise their use in acute coryza. Adrenalin has been much used in this disease because it was thought that the progress of the disease would be affected favorably by reducing the inflammatory reaction. I believe that its use for this purpose is contraindicated except as a temporary measure to establish drainage and ventilation, because the inflammatory reaction is an effort to remove certain noxa or irritants from the tissues, and should not, therefore, be checked by the local use of adrenalin or any other substance. The physician should recognize the activities known as inflammation as forces directed against a noxious foe, and should aid or promote them rather than thwart or check them. The chief difficulty in arriving at a correct understanding of inflammation is that the results of inflammation are confused with the process itself. When I advise the promotion of inflammatory reaction, I do not mean that it should be made worse, that cell proliferation should be increased, that the pain and soreness should be increased, that adhesive processes should be encouraged, etc. These are the results of inflammation, and are not essential features of the reaction. What I mean by promoting the reaction of inflammation is to use such treatment as will increase the hyperemia, the cell nutrition, and the migration of leukocytes. By so doing the irritant noxa is removed, and the cell proliferation, pain, and adhesive processes are quickly relieved or altogether prevented.

While the methods of treatment to be given are somewhat hypothetical, and in some instances purely empirical, they have been rather extensively tried, and have proved to be of more or less value in promoting the inflammatory reaction of acute coryza; that is, they have hastened the destruction of the bacteria and noxa which cause the disease.

(*b*) The tonicity of the vasomotor nervous system should be maintained by the administration of strychnine and arsenous acid in the usual tonic doses. Furthermore, the patient should have plenty of fresh air in his room if it can be arranged without exposing him to a draught.

The administration of aconite or belladonna may be resorted to for the immediate effect upon the turgescence and the secretions, especially in the second stage. An alcohol rub over the entire body also acts as a tonic to the vasomotor nervous system and increases the hyperemia of the arterioles and capillaries, and thereby increases the nutrition of the mucous membrane.

(*c*) While it has not been shown that the disease is due to a specific microörganism, it is evident that bacteria are the exciting cause. An endeavor should be made, therefore, to establish conditions favorable for their destruction and elimination. This should be done by establishing and maintaining drainage and ventilation and promoting the reaction of inflammation. The use of antiseptics has no effect in destroying the bacteria, though they do promote reaction of inflammation. Surgical experience has shown that free drainage is of prime importance in the treatment of infected cavities, as, for instance, in septic peritonitis complicating a ruptured appendix. Irrigation of the abdominal cavity has been abandoned and simple drainage substituted, with brilliant results The same principle applied to acute infectious inflammations of the nasal and accessory sinuses brings equally good results. Hence, the mode of treatment described in paragraph (*a*) will, in most instances, meet the indications. If it does not, the obstructive lesions of the septum (or other lesion) should be removed by surgical means at the earliest possible time, so as to prevent such a complication during subsequent attacks of acute rhinitis.

In addition to the foregoing measures, the use of the leukodescent lamp over the nose and eyes is recommended, to promote the reaction of inflammation. The light from this lamp is rich in blue violet rays, in addition to the heat rays, and they exert a powerful and immediate salutary effect upon the inflammatory process; that is, they greatly increase the hyperemia and the leukocytosis, and thus dispose of the bacteria, their toxin, and the dead cells of the tissues. Having done this, the reaction often rapidly subsides and a cure results.

A treatment with the lamp should cover a period of from twenty to thirty minutes. It should be placed at a distance of about eighteen to twenty inches from the face. The light is more effective if applied over the closed eyes, as the tissues are soft and easily penetrated by the rays, and because the veins of the accessory sinuses empty into the ophthalmic vein. Hence, any increased flow through the ophthalmic vein promotes the flow from the veins of the sinuses and the nose. As acute rhinitis is essentially an acute sinuitis, the reaction affecting the sinuses effects speedy relief or a cure.

The above mode of treatment is based upon rational principles, which, for the sake of emphasis, are recapitulated here:

(*a*) Establishment of ventilation and free drainage of the nasal accessory sinuses.

(*b*) Establishment of tonicity of the vasomotor nervous system.

(*c*) Promotion of elimination of the bacteria by drainage and ventilation of the nasal and accessory sinuses.

(*d*) Promotion of reaction of inflammation by the leukodescent light.

Other Methods of Treatment.—1. The administration of full doses of quinine and a hot lemonade at bedtime will, in some instances, during the first stage, abort acute rhinitis by increasing the hyperemia and leukocytosis. If given during the second or third stages they are ineffective. This method is not as efficacious as the one given above, but is worth trying.

2. Ten grains of Dover's powder and a hot mustard foot bath at bedtime promote the reaction of inflammation to a considerable degree, and if given during the first stage may abort the disease. During the second and third stages it is more difficult to promote the reaction of inflammation, hence this mode is not sufficiently effective in these stages to be of much value.

3. The administration of rhinitis or coryza tablets, containing quinine, belladonna, and morphine, during the first stage will often abort acute rhinitis. One tablet should be given every twenty minutes until dryness of the nose is produced.

4. Aconite administered hourly in the first stage in 1 minim doses until dryness of the throat or tingling of the fingers is produced will sometimes abort the disease. During the second and third stages the remedy is of little use.

Cathartics should always be given early in the disease.

CHRONIC RHINITIS WITH TURGESCENCE

Synonyms.—Alternating stenosis; simple chronic rhinitis.

Definition.—Chronic rhinitis with turgescence is characterized by fugitive swelling or turgescence of the "swell bodies" of the inferior turbinates, and the patient complains of attacks of nasal obstruction and a thick mucous discharge.

Etiology.—The causes of chronic rhinitis are given under the etiology of acute rhinitis, and will not be repeated in detail. It should be stated, however, that in most cases there is a deviation of the septum in its lower and middle portion. The deviation may also be an anterior one near the vestibule of the nose in the cartilaginous portion of the septum, thereby producing anterior nasal stenosis. With each descent of the diaphragm the air is rarefied posterior to the obstruction, and a negative pressure in the nasal chambers results. The blood in the mucous membrane lining the nasal chambers is thus drawn to the venous plexuses (swell bodies) of the turbinates, and turgescence or engorgement results.

In the section on Deviations of the Septum it has been shown that obstructive lesions in the region of the inferior turbinal act in such a way as to produce engorgement of the tissues without much irritation. Hence the effect at first is simply one of turgescence, which in the course of years of increased nutrition results in hypertrophy or hypertrophic rhinitis. If in addition to the local turgescence there is an asso-

ciated obstruction in the region of the middle turbinal, the retention and decomposition of the secretions in the superior meatus and the posterior ethmoidal cells cause a prolonged low-grade irritation which may result in a hyperplasia of the mucous membrane, not only of the middle turbinal, but of the ethmoidal cells as well. As an obstructive lesion of the septum in the middle turbinal region often co-exists with the obstructive ridge or spur in the inferior turbinal region, hyperplasia or hyperplastic rhinitis affecting the inferior and middle turbinals is often present. When, however, the upper obstruction is absent, the rhinitis is usually of the turgescent or hypertrophic type.

Pathology.—In the early stage there is a distention of the venous or cavernous tissue of the conchæ (turbinates). If the inflammatory process continues a true hypertrophy of the tissues takes place on account of the increased nutrition from the large blood supply.

Symptoms.—The symptoms are chiefly caused by transient stenosis of the breathway of the nose. In addition, the secretions are heavier; that is, the mucoid element is increased, while the serous element may be decreased in quantity. The patient believes there is an actual increase, whereas, as a matter of fact, there is probably a decrease in the amount of secretion. The apparent increase is due to the greater consistency of the secretion, which renders it less absorbable by the ingoing current of air. In a normal nose the secretions are comparatively thin or serous, and are largely absorbed for physiological purposes and carried to the lower respiratory tract.

The transient stenosis is either intermittent or alternating; that is, both sides may be stenosed for a period and then open, or the stenosis shifts from one side to the other. These symptoms are quite characteristic of turgescent rhinitis.

The objective signs of turgescent rhinitis are chiefly found in the evidences of engorgement of the "swell bodies" of the inferior turbinates. Upon inspection by anterior rhinoscopy, the outline of the inferior turbinate is smooth and boggy-like, whereas, in true hypertrophic rhinitis it is firm and unyielding. The application of cocaine or adrenalin causes shrinkage of the mucous membrane which covers the inferior turbinate, whereas in hypertrophic rhinitis there is little or no shrinkage.

The secretions are mucoid in character, and when the "swell bodies" are contracted, strings of mucus extend from the septum to the inferior turbinate.

A spur or ridge is usually present upon the lower portion of the septum, causing obstruction in some degree in the region of the inferior turbinate. The cartilaginous portion of the septum may also be deflected, thereby causing anterior nasal stenosis and a consequent rarefaction of the air within the nasal chambers with each inspiratory current.

Epistaxis is also occasionally complained of. The ridge or crest of the septum projects into the inspiratory tract, and is thereby subjected to excessive evaporation of the secretions accumulated upon it. The dried crusts are blown or picked off, tearing the underlying epithelium and the capillary vessels; hence the epistaxis.

Cough when present is due to an associated bronchitis or laryngitis.

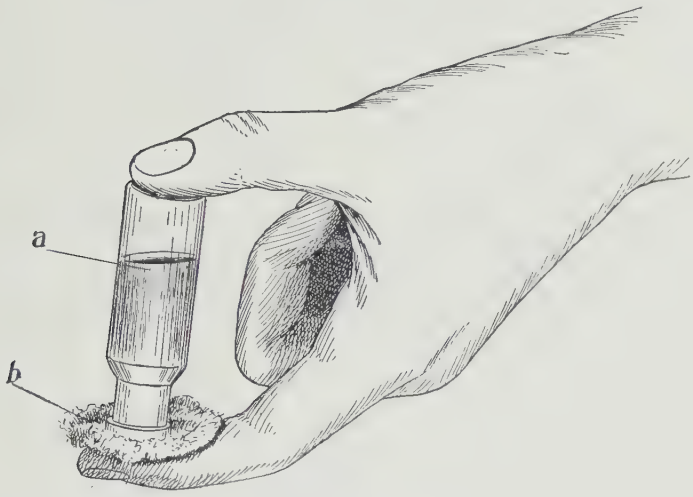
Posterior rhinoscopy reveals an enlargement of the "swell bodies" upon the posterior ends of the middle and inferior turbinates. The enlargement has often been likened to a mulberry. It is nodular in outline and of a grayish-blue color.

Prognosis.—If allowed to run its course, true hypertrophy and a lessened functional activity of the tissue occurs. Under appropriate treatment the disease is curable.

Treatment.—The treatment should be twofold in character: (*a*) The removal of the predisposing causes, and (*b*) the control of the immediate symptoms.

(*a*) The removal of the predisposing causes is usually accomplished by the correction of the deviated septum. (See Treatment of Deviations of the Septum.) When this is done the negative air pressure in the nasal

FIG. 102



Method of moistening a thin pledget of cotton with cocaine or adrenalin solution: *a*, the solution in an inverted bottle; *b*, the pledget of cotton.

chambers disappears and the blood ceases to be drawn to the mucous membrane, and the tendency to intermittent and alternating stenosis is greatly reduced. The choice of operation should be determined according to the type and location of the deviation of the septum.

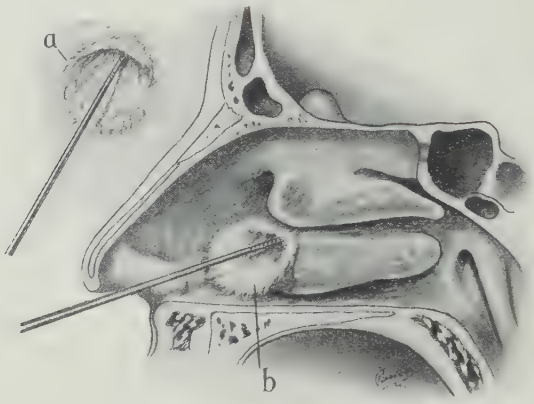
(*b*) The palliative treatment should be addressed to the immediate control of the distressing symptoms, namely, the stenosis and the heavy secretions. The transient stenosis may be controlled by the use of the electric or chemical cautery or by incising the turgescent "swell bodies."

Electrocauterization.—The technique of electrocauterization is as follows:

(*a*) Induce cocaine anesthesia by the application of a 4 per cent. solution of cocaine on a thin pledget of cotton to the swollen free border of the inferior turbinate for a period of ten minutes (Figs. 102 and 103).

(*b*) Turn on the electric current until the point of the cautery electrode is of a bright cherry-red color.

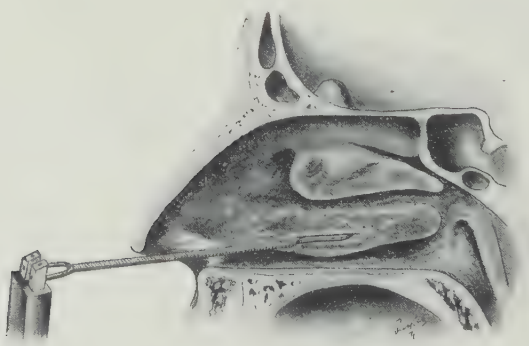
FIG. 103



Method of applying the pledget of cotton to the inferior turbinated body: *a*, the pledget of cotton after being moistened with the cocaine or adrenalin solution is engaged upon the tip of a delicate silver probe; *b*, the pledget of cotton being "pasted" or spread upon the inferior turbinated body.

(*c*) Introduce the electrode into the nasal chamber cold and place it on the free border of the inferior turbinate (Fig. 104). Then move it backward and forward, while still cold, until sure of its correct position. Maintain the to-and-fro motion and press the contact spring of the

FIG. 104



Lateral view, showing the cautery electrode in position for cauterizing the inferior turbinated body.

cautery handle for one or two seconds, when the contact should be broken. The to-and-fro motion should be continued until the electrode is cold, that is for two or three seconds after the spring contact is broken, and then it should be removed from the nose.

If these instructions are followed, the procedure is painless and does not tear the eschar from the turbinal. If the to-and-fro motion is not maintained before, during, and after the electrode is heated, the eschar will be torn off and the cautery effect lost.

The eschar must be left in place. If bleeding follows the removal of the electrode, the eschar is lost and the cauterization rendered useless.

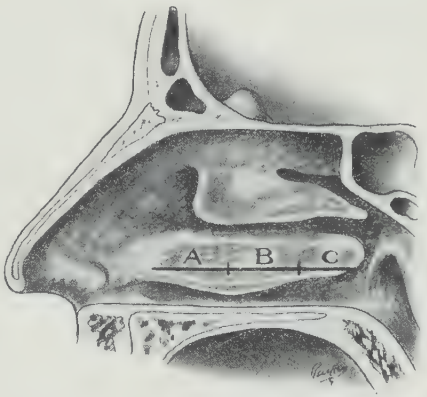
The cauterization should be linear, and should be about one inch in length. The whole length of the inferior turbinate may be cauterized in three sittings (Fig. 105), never in one, as too great a reaction and sloughing may follow.

The sittings should be from five to seven days apart. A week after the first cauterization the opposite side may be treated in like manner. At the end of another week the middle portion of the inferior turbinate first cauterized may be thus treated. And so continue to cauterize the turbinates alternately, at weekly intervals, until the whole length of both turbinates has been cauterized.

The after-treatment of a cauterized turbinate should consist in an immediate spray of an alkaline solution—Dobel's or Seiler's solution. An oily aromatic nebula should follow this. Prescribe Seiler's solution for daily use by the patient. The wash should be used with a glass nasal douche rather than an atomizer, as the force of the spray might injure the cauterized surface.

Should infection occur, gently pack the nose with small cotton pledgets saturated with a 10 per cent. aqueous solution of Merck's ichthyol. Remove the pledget in about fifteen minutes and insufflate bismuth powder into the nose. The clothing of the patient should be regulated according to indications. Heavy-soled shoes should be prescribed.

FIG. 105

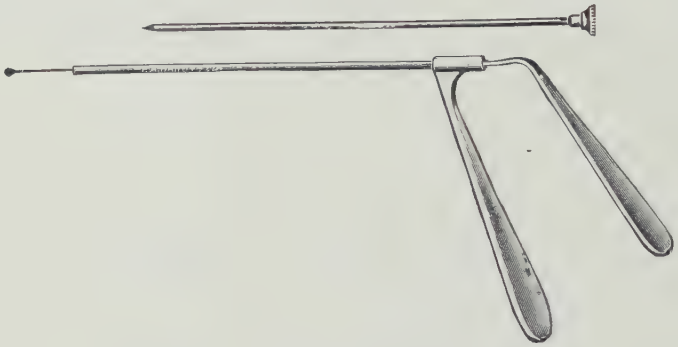


Showing the lines for linear cauterization in turgescent rhinitis: *A*, *B*, and *C*, representing respectively the first, second, and third cauterizations, which should be made one week apart.

Submucous Cauterization.—N. H. Pierce first introduced the submucous cauterization of the inferior turbinated body for the reduction of turgescent and hypertrophic rhinitis. The mucous membrane is punctured near the anterior end of the free border of the turbinate and a tunnel made with a blunt probe beneath the turgescent membrane. A fused bead of chromic acid is then introduced into the artificial tunnel or channel. M. A. Goldstein improved the instruments for this procedure, as shown in Fig. 106. By Goldstein's method the bead of chromic acid is concealed in the cannula while being introduced, the fused bead of acid then being thrust from the end of the cannula and withdrawn through the channel in the submucous tissue.

Sloughing sometimes follows this method of cauterization. Chromic acid is very irritating to the kidneys and may cause nephritis. It should never be used in a patient already subject to nephritis, for obvious reasons.

FIG. 106



Goldstein's chromic acid applicator for submucous cauterization.

HYPERTROPHIC RHINITIS.

Synonyms.—True hypertrophic rhinitis; obstructive rhinitis; hypertrophic nasal catarrh; hypertrophic ozena; hypertrophy of the turbinated bodies; hyperplastic rhinitis.

Definition.—Chronic hypertrophic rhinitis is characterized by a partial stenosis of the nasal chambers, due to hypertrophy of the mucous membrane of the inferior turbinated body.

Etiology.—The causes of hypertrophic rhinitis are essentially those given under turgescent rhinitis. When there is an anterior deviation of the septum there is a negative air pressure within the nasal chambers with each inspiratory effort. The hyperemia resulting therefrom leads to an overnutrition of the mucous membrane, especially of the "swell bodies." The contact of the deviated septum with the mucosa of the inferior turbinal irritates it and thus still further excites the hypertrophic process. The altered secretions add to the irritation, and still further increase the hypertrophy of the mucous membrane.

In cases which are complicated by a high deviation of the septum, and in which there is a complicating sinuitis (catarrhal or suppurative), the tissue changes are somewhat modified. Instead of hypertrophy, the irritating discharge from the sinuses often causes hyperplasia of the mucous membrane. There may be present, therefore, both hypertrophy and hyperplasia of the tissue. Either the hypertrophy or the hyperplasia may predominate. The so-called hypertrophic rhinitis may, therefore, be divided into two groups: (*a*) The hypertrophic variety, and (*b*) the combined hypertrophic and hyperplastic variety. This subdivision is still further justified by the clinical fact that the symp-

tomatology and treatment of the two conditions are often quite different. The hypertrophic variety presents symptoms which are due chiefly to the anterior and the inferior obstruction of the nose, whereas the combined variety presents symptoms due to obstruction in the middle turbinal region as well as to the obstruction in the anterior and inferior portions of the nasal chambers.

The causes of uncomplicated hypertrophic rhinitis are, therefore, those conditions which give rise to a chronic hyperemia of the mucosa and to a passive engorgement of the "swell bodies." These conditions are the anterior and inferior obstructive deviations of the nasal septum and the climatic and hygienic conditions which affect the vasomotor nervous system.

Pathology.—The morbid anatomy of hypertrophic rhinitis consists in an increased blood supply and an increase of tissue from nutritional rather than from irritative and inflammatory causes. The part most frequently hypertrophied is the mucous membrane containing the "swell bodies," as there is naturally a greater flow of blood through these vascular bodies.

Symptoms.—The symptoms are chiefly those of more or less nasal stenosis. The secretion is usually heavier than normal, and pasty in consistency, although it may be comparatively thin and watery, especially during an acute exacerbation.

The nasal stenosis may be limited to one side, the side of greater septal convexity. The inferior turbinate on the side of the concavity is often greatly hypertrophied, a so-called compensatory hypertrophy, although, as a matter of fact, it may be due to a negative air pressure within the nasal chamber on that side. The anterior opening of the nose on that side, while normal in size, is, on account of the diminished size of the opposite chamber, inadequate to admit air rapidly enough for physiological purposes; hence, engorgement and subsequent hypertrophy results. It follows that both nasal passages are often more or less constantly blocked in the region of the inferior turbinate. The patient complains of stuffiness, or sense of a foreign body in the nose, and makes frequent but ineffectual attempts to remove it by blowing the nose.

Upon anterior rhinoscopic examination the inferior turbinal is observed to be enlarged and to have an irregular nodular surface. Probe pressure does not cause pitting, as in turgescent rhinitis, but elicits a sense of resistance and of thick fleshy tissue. The application of cocaine or adrenalin is not followed by marked contraction of the tissue.

Epistaxis from the dislodgement of an adherent crust upon the crest of the deflection occasionally occurs.

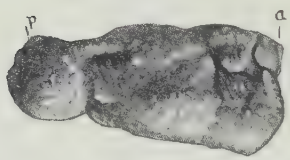
Prognosis.—If allowed to run its natural course, hypertrophic rhinitis tends to become worse rather than better. Indeed, in the course of time the secretions may become so heavy and so adhesive in quality as to be removed with great difficulty. In such subjects irritation results and hyperplasia of the tissue follows. If this is allowed to progress the vascular and glandular tissues become enmeshed in the contractile hyperplastic tissue, and atrophy of the mucous membrane begins.

If, on the contrary, appropriate treatment is instituted sufficiently early, the prognosis is fairly good.

Treatment.—The treatment consists mainly in overcoming the stenosis and removing a part or all of the hypertrophic tissue. Sprays and douches of alkaline antiseptic solutions do little more than temporarily increase the reaction of inflammation and relieve the symptoms by the removal of the altered secretions. The nasal stenosis is overcome by the surgical correction of the septal deformity and the removal of the excessively hypertrophied turbinal tissue (Fig. 107). (See Obstructive Deviations of the Septum and the Methods of Correcting Deviations of the Septum.) Be assured that in most instances hypertrophic rhinitis is a surgical rather than a medical disease. Be assured, also, that hypertrophic rhinitis cannot be cured by sprays and other local medicinal applications, although they may temporarily relieve some of the symptoms.

The actual cautery has been recommended for the reduction of hypertrophied mucous membrane. I can only condemn it as inadequate for this purpose. If it is used freely enough to accomplish anything, it produces excessive scar tissue, a result to be carefully avoided.

FIG. 107



Hypertrophy of the mucous membrane of the inferior turbinated body: *a*, anterior attachment; *p*, posterior attachment. Removed by the author with his turbinotome. (Dr. Henrietta Gould's case.)

Surgical Treatment.—If the hypertrophy is great enough to obstruct the nasal passages, it should be removed surgically with scissors, saw, or spokeshave.

The Scissors.—The scissors are generally used for the removal of the hypertrophied portion of the free border of the inferior turbinated body. The technique is as follows:

(*a*) Induce local anesthesia by the application of a 5 per cent. solution of cocaine by means of a thin pledget of cotton, which should be placed over the hypertrophied area for ten minutes.

(*b*) With nasal scissors (Fig. 108) cut off the necessary portion of the hypertrophied membrane.

(*c*) Use no dressing except an antiseptic dusting powder. An exception may be made, however, in favor of Pischel's collodion dressing if perfect dryness of the parts can be secured.

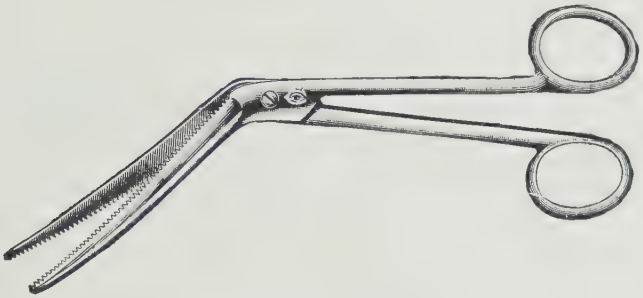
(*d*) If severe hemorrhage occurs, it becomes necessary to pack the nose in order to check it. This may be done by introducing a postnasal tampon with Bellocq's cannula (Fig. 109), or with a rubber urethral catheter. A long strip of gauze should then be packed against it through the anterior nares. When such a tampon is used it should be moistened with the compound tincture of benzoin or impregnated with bismuth powder to prevent decomposition of the secretions. When either of these precautions is taken the tampon may be left in place for three or four days without putrefaction.

The Saw.—The saw may be used instead of the scissors when it is necessary to remove a portion of the inferior turbinated bone with the hypertrophied membrane (Holmes, Vail).

Technique.—(*a*) Induce local anesthesia with cocaine.

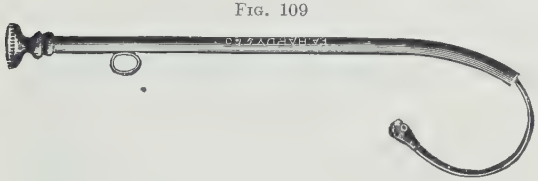
(*b*) Introduce a slender nasal saw beneath the inferior turbinated body and saw in an inward and upward direction through it. If it is

FIG. 108



Beckmann's serrated scissors.

FIG. 109



Bellocq's postnasal tampon cannula.

impossible to insert the saw beneath the turbinated body it may be introduced above it and the incision carried downward and outward through the tissue.

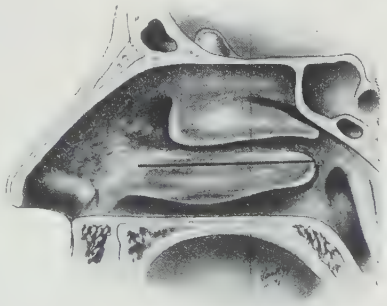
(*c*) Either use no dressing or use the Pischel collodion dressing when conditions are favorable, that is, when all hemorrhage ceases.

The Spokeshave.—The spokeshave may be used if it can be engaged posteriorly in such a position as to enable the operator to control its direction in cutting forward. This operation is rarely justifiable, as too much of the turbinate is removed by it.

The Technique.—(*a*) Induce local cocaine anesthesia.

(*b*) Make a linear incision along the mediosuperior surface of the inferior turbinate just at the upper margin of the hypertrophied tissue (Fig. 110). The incision is for the purpose of preventing laceration of

FIG. 110



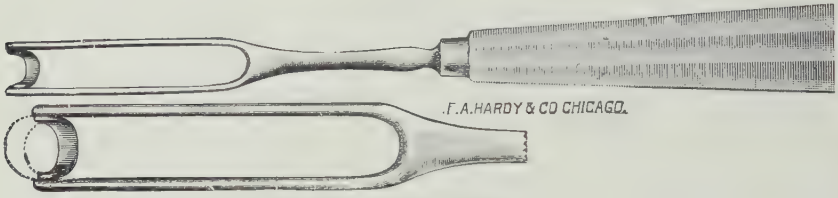
Showing the incision preliminary to the removal of the inferior turbinated body with the spokeshave or swivel knife.

the mucous membrane as the spokeshave is drawn through it. Healing is promoted by making a clean cut.

FIG. 111

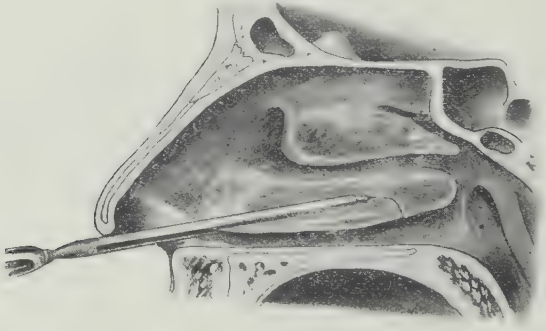
Spokeshave.

FIG. 112



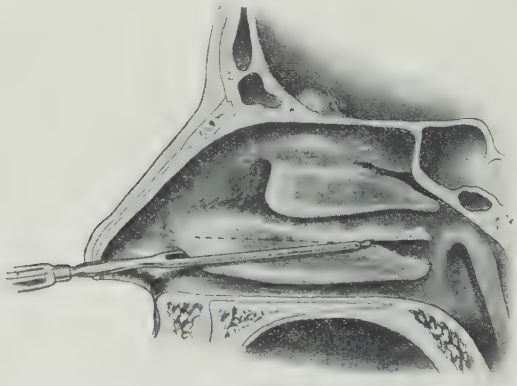
The author's swivel turbinotome.

FIG. 113



The removal of the anterior two-thirds of the inferior turbinate with the author's wide swivel knife (Fig. 112).

FIG. 114



Showing the removal of the inferior turbinate with the author's large swivel knife.

(*c*) Introduce the spokeshave (Fig. 111) at the posterior extremity of the turbinate if there is a mulberry hypertrophy there, or along the free border of it if only that portion is involved. Engage the turbinated body and pull forward in such a direction as to include only the hypertrophic tissue. The spokeshave should not be used unless it is desired to remove some bone as well as soft tissue.

(*d*) Follow the same method of after-treatment given in the previous operations.

The Swivel Knife.—The author's large swivel knife (Fig. 112) may be used with even greater advantage than the spokeshave, as it can be made to engage or leave the tissue at any desired point along the free border of the turbinate. The knife used for this purpose is especially designed with a view to its width and strength. Otherwise it is similar to the one used in the submucous resection of the nasal septum.

The Technique.—(*a*) Induce local cocaine anesthesia.

(*b*) Insert the swivel knife as though it were a spokeshave and force the blade into the turbinate posterior to the hypertrophied tissue (Figs. 113 and 114). When it is sufficiently engaged in the tissue pull it forward, as in the spokeshave operation, and disengage it by directing it downward toward the floor of the nose when the anterior limit of the hypertrophy has been reached. The preliminary incision of the membrane is unnecessary, as the cutting edge of the blade is concave and prevents laceration of the mucosa. Bone, as well as soft tissue, may be removed with it.

(*c*) The after-treatment should be the same as in the other operations.

HYPERPLASTIC RHINITIS

Synonyms.—The same as given under hypertrophic rhinitis, as the two conditions are often confounded.

Definition.—Hyperplastic rhinitis is characterized by an increase in the thickness of the mucous membrane as a result of prolonged mild irritation by the secretions from the sinuses. It differs from hypertrophic rhinitis in its causation and in its morbid anatomy. In hypertrophy there is an increase in the size of the cells from overnutrition, whereas in hyperplasia there is an increase in the number of cells, and especially of the connective-tissue cells, from the slight but prolonged irritation. Polypi are a later development of this condition (see Chapter XIII).

Etiology.—The chief causes are pressure, or the close approximation of the septum to the middle turbinate, the resultant retention of the secretions, and the inflammation of the obstructed sinuses. The septum does not, in all cases, impinge upon the middle turbinate, and is not, therefore, a constant etiological factor in producing the hyperplasia. The sinuses may be diseased independently of the septal deviation, and may thus be the primary cause of the hyperplasia. In either event the irritation resulting from the secretions constantly flowing over the mucous membrane of the middle and inferior turbinates causes the morbid changes in these structures. The secretion is not necessarily

purulent, but, on the contrary, is often serous or mucous in character; that is, the inflammation in the sinuses may not be suppurative, but may be catarrhal in character.

Symptoms.—The symptoms of hyperplastic rhinitis are often complex, as the disease is often associated with a catarrhal or a suppurative inflammation of the ethmoidal, sphenoidal, and possibly the frontal sinuses.

The symptoms arising from the hyperplasia are those of nasal obstruction, especially in the region of the middle turbinate; that is, there is more or less nasal obstruction and a sense of stuffiness or of pressure in this portion of the nose. The handkerchief is frequently used in efforts to dislodge the secretions and to overcome the sense of stuffiness. While the secretions may be thus removed, the stuffy feeling often remains, as it is due to the contact of the turbinate with the septum.

The secretions may be serous, mucopurulent, or purulent, depending largely upon the complicating disease of the sinuses. According to Uffenorde and Skilleren suppuration is rarely present, indeed, they claim that the microscopic appearances of the tissues in suppurative ethmoiditis and hyperplastic ethmoiditis or rhinitis are different. According to my clinical observations hyperplastic ethmoiditis or rhinitis is nearly always attended by suppuration, though in less degree than in simple empyema without marked hyperplastic changes.

Anterior rhinoscopy shows the inferior turbinate to be enlarged, paler than normal, or it may be red and boggy, and somewhat nodular in outline, and polypi may be seen growing from its lower border. The enlargement present is due to the hyperplastic change in the mucous membrane. If this tissue becomes edematous and pendulous, it constitutes true polyp. I have often examined specimens of the ethmoid bone removed from patients in which the cells were more or less filled with small polypi, while other larger polypi protruded from the cells through the ostia and were hanging free in the nasal chamber. In view of these findings, it is obvious that the removal of the visible polypi would fail to relieve the patient, as the small, budding polypi within the cells would sooner or later extend through the ostia into the nasal chambers. If the septum is deviated, and it usually is, a ridge corresponding to the crista nasalis and the crest of the vomer may be present on one side, while there is a bowing of the septum toward the opposite side in the region of the middle turbinate. The mucous membrane covering the septum is also often thickened just below the inferior border of both the middle turbinate bodies, thereby obstructing both olfactory fissures.

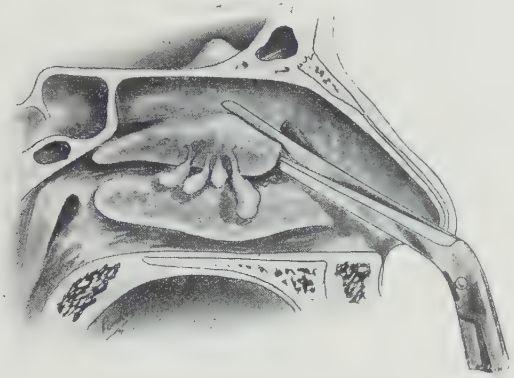
If empyema of the posterior ethmoidal cells (cellulæ ethmoidales) is present, pus may be seen in the olfactory fissure as well as in the lower portion of the nose. If there is catarrhal ethmoiditis, the anterior end of the middle turbinate may be red and boggy in texture. Patients with this type of ethmoidal inflammation sometimes complain of soreness or of fissures at the margins of the vestibules.

The subjective symptoms are due to obstructive lesions and to the disease in the accessory sinuses of the nose.

The obstruction in the upper part of the nose gives rise to a sense of stuffiness and of pressure across the bridge of the nose. These symptoms are rather constant, as the tissue enlargement is permanent.

The obstructive lesion in the upper portion of the nose gives rise to the additional symptoms of headache and vertigo peculiar to inflammation of the sinuses; that is, there is headache in the frontal region limited

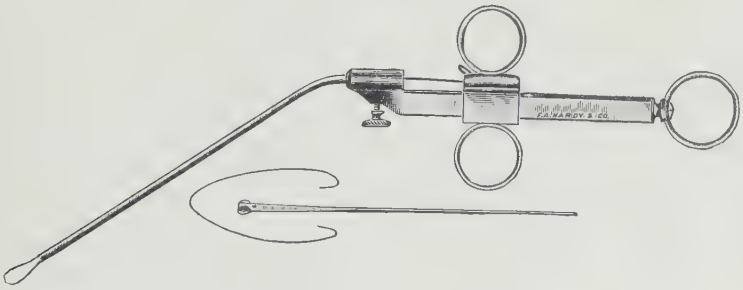
FIG. 115



The removal of the anterior end of the middle turbinated body with Casselberry's scissors.

to, or more pronounced on, one side, and to a feeling of soreness or tenderness of the eyeball upon ocular movements. The stooping posture increases the headache, and temporary vertigo is often thereby produced. The headache is also sometimes in the temporal, vertexial, and occipital regions, especially if the posterior ethmoidal and sphenoidal sinuses are involved.

FIG. 116



Krause's nasal snare.

The symptoms given in the above paragraph are due to the sinuitis, and are not always present in hyperplastic rhinitis.

Prognosis.—The prognosis of hyperplastic rhinitis is not as favorable as that of hypertrophic rhinitis. The etiology is more complex and the disease more serious, and it necessitates more extensive surgical procedures for its eradication.

Treatment.—The treatment of hyperplastic rhinitis should have two chief objects, namely: (*a*) The removal of the obstructive lesion, whether it be deviation of the septum or hypertrophy of the middle nasal concha (middle turbinate), and (*b*) the cure of the sinuitis, if present, whether it be in the ethmoidal and sphenoidal or the frontal and maxillary sinuses.

The Author's Turbinotome.—With the author's turbinal knife (Fig. 120) all or any portion of the middle turbinate may be removed under cocaine anesthesia. The technique for the removal of the anterior portion is as follows:

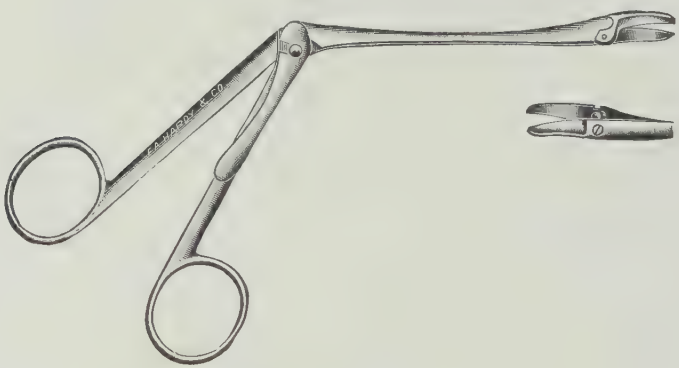
(*a*) Introduce the knife through the olfactory fissure as far posteriorly as it is desired to begin the incision.

(*b*) Turn the cutting edge of the blade outward and forward and force it into the turbinate as far as it will go.

(*c*) Then cut forward to the anterior attachment of the turbinated body as shown in Fig. 120.

(*d*) Remove the severed portion with dressing forceps.

FIG. 117



Holmes' middle turbinal scissors.

The Scissors and Snare.—The technique is as follows:

(*a*) Induce local anesthesia with a 10 per cent. solution of cocaine. A weaker solution is often inadequate in hyperplastic tissue.

(*b*) Grasp the anterior attachment of the middle nasal concha (middle turbinate) with the scissors and make an incision about one inch in length, thus severing the attachment of the anterior one-third or one-half of the middle turbinated body (Fig. 115).

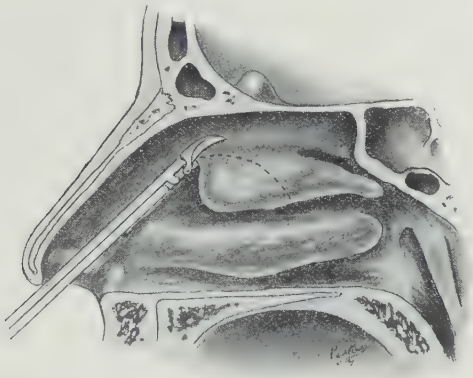
(*c*) Introduce a cold wire loop over the detached portion of the turbinate and cut it off at the posterior limit of the incision, or sever the detached portion of the turbinate with Grünwald's forceps. Still more tissue may be removed if necessary.

Holmes' Scissors.—With Holmes' scissors (Fig. 117) the snare is not necessary, as the blades are so curved that the cut made with them extends backward and downward until it emerges from the tissue (Figs. 118 and 119).

The Swivel Knife.—The technique of the removal of the middle turbinate with the swivel knife differs from that employed with a larger instrument in the removal of the inferior turbinate.

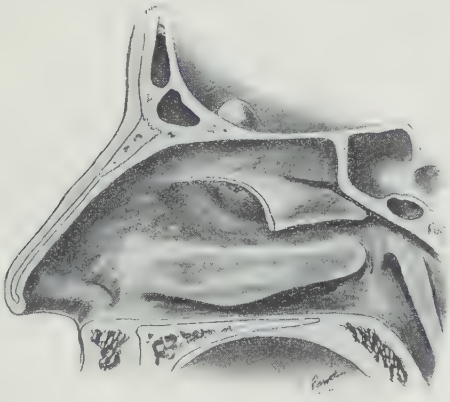
The technique is as follows: (*a*) Induce local anesthesia with a 10 per cent. solution of cocaine applied on a thin pledget of cotton over the whole of the middle turbinate. It may be necessary to apply a 20 to 30 per cent. solution, or even powdered cocaine with a delicate cotton-wound applicator to the less accessible areas.

FIG. 118



The removal of the anterior half of the middle turbinated body with Holmes' scissors.

FIG. 119



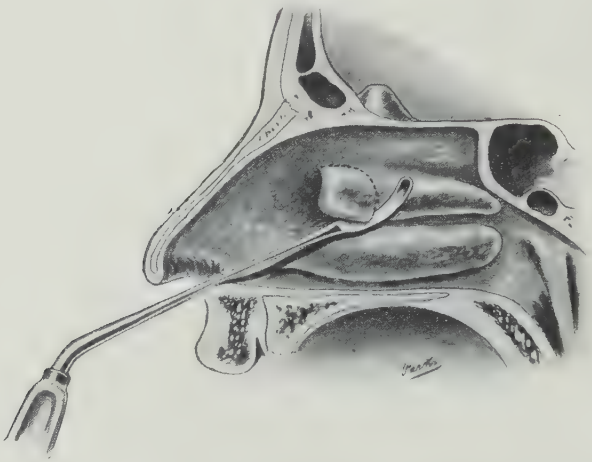
Anterior half of the middle turbinate removed with Holmes scissors, exposing the bulla ethmoidalis.

(*b*) Introduce the small swivel knife and engage the anterior attachment of the middle turbinate (Figs. 121 and 122), so that one prong tip is above and the other below the attachment.

(*c*) Carry the swivel blade backward with short strokes until the whole or a part of the middle turbinate is severed from its attachment. The severed middle turbinate does not pass between the prongs of the instrument, but is pushed downward beneath them. If only a portion of the

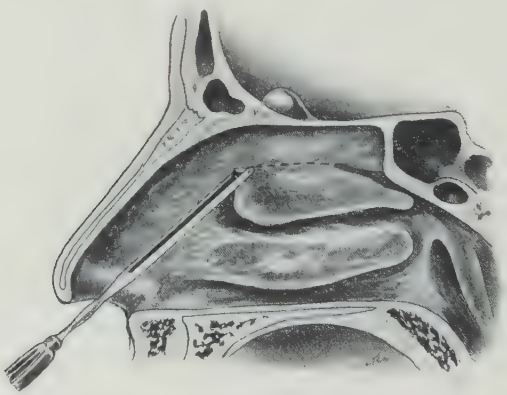
middle turbinate is to be removed, the swivel blade is directed downward through the turbinate at the desired point, or, failing in this, the swivel knife is removed and the loop of a snare is engaged over the detached fragment and the removal completed.

FIG. 120



The removal of the anterior portion of the middle turbinated body with the author's turbinal knife.

FIG. 121



The author's narrow swivel knife placed at the anterior attachment of the middle turbinate preparatory to removing it.

Remarks.—The swivel knife is not universally suited for turbinectomy or turbinotomy, although in many cases it is an ideal instrument for these purposes. In each case the instruments and mode of operation should be selected with reference to the conditions present rather than to follow blindly any described method of operating.

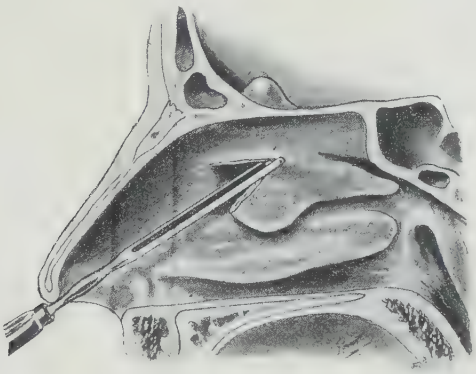
(*d*) The postoperative treatment should consist of the insufflation of bismuth powder, and, in case of severe persistent hemorrhage, the

nose should be packed with bismuth, or compound tincture of benzoin gauze.

Hemorrhage.—The middle turbinate is supplied with blood by the anterior and posterior ethmoidal arteries (A. ethmoidalis anterior et posterior) (Plate I, Fig. 1), and hemorrhage of considerable severity may occur either at the time of operation or at a later period. As a matter of fact, an oozing of blood continues in many cases for twenty-four hours.

The danger of septicemia and of meningitis is increased by nasal tampons, hence it is not advisable to pack the nose except in extreme necessity. Several cases of meningitis have occurred as a result of nasal tampons introduced after middle turbinotomy. The packing should be done with caution, and the gauze should be moistened with

FIG. 122



The removal of the middle turbinate with the author's narrow swivel knife.

the compound tincture of benzoin and squeezed until the excess of fluid is removed. If the operation is performed in a hospital, it is rarely necessary to pack the nose, as the patient remains quiet and severe hemorrhage rarely occurs. If it does occur, the house surgeon should be instructed to introduce the tampon.

The chief causes of complications and sequelæ after nasal operations are, namely: (*a*) The failure to sterilize the nasal chambers; (*b*) the use of nasal tampons; (*c*) ragged contused wounds; (*d*) blowing the nose, thus forcing infectious material into the sinuses and cranial cavity.

CHRONIC RHINITIS WITH COLLAPSE OF THE ERECTILE TISSUE

Definition.—This is not a true inflammatory disease, but is usually classed as such. It is a local manifestation of a general anemia; it is characterized by the collapse of the erectile tissue of the nose, and resembles atrophy in this region.

Etiology.—Its chief cause is general anemia. Atrophic rhinitis is also characterized by anemia that is secondary to the conditions causing the atrophy. In simple collapse of the "swell bodies" the anemia is

primary and the collapse secondary. It is most often found in women, as they are more subject to anemia. It is occasionally found in gouty individuals.

Symptoms.—The chief symptoms are dryness of the upper respiratory tract and patency of the nose. Upon anterior rhinoscopic examination the inferior turbinates appear quite small, on account of the collapse of the "swell bodies." Upon probe pressure the mucous membrane is found to be thin and tightly drawn over the underlying bone. The great space in the nasal chambers and the small size of the inferior turbinates at once suggest an atrophic condition, though true atrophy is absent; crusts and ozena are absent, nor is there a history of their previous presence. An examination of the blood shows anemia to be present. The sense of smell is unimpaired and ulceration of the mucosa and caries of the bone are absent. The condition is always bilateral, as it is due to constitutional rather than local causes.

Treatment.—The treatment should be directed to the anemia. It is necessary, therefore, to ascertain the type of the anemia by blood examinations and to carry out the treatment accordingly. I wish to suggest that an examination of the rectum will sometimes reveal ulcerations or other pathological processes that may be the cause of the anemia and the resultant collapse of the erectile tissue.

ATROPHIC RHINITIS

Synonyms.—Chronic dry rhinitis; simple mucous rhinitis; mucopurulent rhinitis; ozena.

Definition.—Atrophic rhinitis is characterized by a sclerotic change in the mucous membrane and occasionally of the underlying bone and by the presence of crusts and an offensive nasal breath. The conditions giving rise to these phenomena are varied and often complex.

Etiology.—The three causes of this condition are as follows:

(*a*) A simple atrophic process which is not dependent upon other local diseases of the mucous membrane. Meissner holds that atrophic ozena (see below) is due to a primitive or broad, shallow nose, and to a congenital development of pavement epithelium instead of the columnar or mucus-producing variety.

(*b*) Pressure necrosis due to excessive distention of the bloodvessels. This is a *cyanotic congestion* due to a heart lesion, and the general venous circulatory system participates in the sluggish venous flow. The mucosa covering the vessels is kept constantly stretched, and pressure atrophy results, as in red atrophy of the liver (D. Braden Kyle).

(*c*) Sclerotic atrophy due to a preëxisting inflammation of the sinuses during which there is an excessive proliferation of connective-tissue cells. These after a time become fibrous tissue and gradually cut off the blood supply and choke out the glandular and vascular structures of the membrane. The nutrition of the mucous membrane is diminished, and functional activity is diminished or destroyed.

These and various other theories are thought to be the cause, or causes of atrophic rhinitis. None of them is definitely proved, although the

one (*c*) advocated recently by Grünwald, and by Vieussens, Reininger, and Guns at the end of the seventeenth century, has rapidly gained ground in popular opinion. Those who hold to this theory believe that all or nearly all cases of atrophic rhinitis are due to suppuration of the accessory sinuses of the nose, more especially the ethmoidal and sphenoidal. My own experience is in accord with this view. I have seen many cases cured or greatly relieved by attention to the accessory sinuses. The ozena is invariably influenced favorably. In conjunction with Dr. Joseph C. Beck I have had skiagraphs of the sinuses made in cases of atrophic rhinitis, and without exception the sinuses appear cloudy, as they do in sinuitis, *i. e.*, their outline is poorly defined and the area of the cavities is opaque. This shows that in atrophic rhinitis the sinuses are often diseased, though it does not prove the disease of the sinus to be primary.

(*a*) **Simple Atrophic Rhinitis.**—Simple atrophy may take place in the nasal mucous membrane as well as in mucous membranes elsewhere in the body.

Etiology.—The etiology is not clear, but it is probable that the disease is due to the presence of some irritant in the blood, as in syphilis, tuberculosis, scrofula, etc. At any rate, the trophic nervous system is involved and nutrition modified.

Treatment.—The treatment should be addressed to the constitutional dyscrasia, upon the disappearance of which the atrophic and ozenic processes improve or disappear.

(*b*) **Atrophic Rhinitis Due to Pressure (Cyanotic Engorgement).**—*Etiology.*—(*a*) There may be some lesion of the heart, kidneys, liver, or lungs which causes a damming back of the venous blood upon the nasal mucous membrane, as well as elsewhere in the body. (*b*) The organs thus affected do not eliminate the waste products as rapidly as they should, and these are retained in the blood, where they act as irritants, and excite a slight inflammatory reaction. These two factors account for the phenomenon known as pressure atrophy as it occurs in the nasal mucosa.

Symptoms.—Although there is true atrophy, the membrane is congested to such a degree that there is nasal stenosis. The mucosa of the nose is swollen, purplish red in color, and inflamed. The ozenic odor may be slight. There is an exudation from the engorged vessels, but it is not a true mucous secretion. The skin of the nose may be red. There is a sense of fulness across the bridge of the nose, and frontal headache is commonly present. The conjunctiva may be injected, and this is attended by an overflow of tears.

D. Braden Kyle refers to a case due to organic mitral lesion. I have seen a case of this character in which the whole mucosa of the upper respiratory tract was cyanotic; the tonsils were enlarged and markedly blue from cyanotic congestion.

Prognosis.—This depends upon the curability of the lesions giving rise to the cyanotic congestion. In the cases referred to the patient had a valvular heart lesion.

It is obvious that the *treatment* in such cases must be palliative only.

(*c*) **Atrophic Rhinitis Due to Suppurative Sinuitis.**—*Etiology.*—All the causes given under the various types of catarrhal rhinitis may act as causes of this type of disease. The inflammation attending them is followed by a deposit of connective-tissue cells, which, after they become organized, cut off the blood supply and choke down the glandular tissue. The functional activity is gradually lost and the true mucous elements of the membrane finally disappear. The secretions become thick and inspissated. They dry upon the surface of the membrane, where, through biochemical changes, they develop the ozenic odor. Various theories have been advanced in explanation of the cause of the odor.

The following are suggestive but not conclusive:

(*a*) Simple decomposition of the mucopus.

(*b*) Degenerative changes in which certain fatty acids are liberated, giving rise to the odor.

(*c*) The presence of certain bacteria, as the Bacillus fœtidus.

Ozena a Symptom.—Ozena is not a disease, but a sign of certain diseased conditions. It is a "stench," and it is in this sense that the term is used. The fetid odor is associated with an inspissated secretion, which forms greenish crusts over the whole of the nasal mucous membrane. Other peculiar conditions may be associated with it, especially in those cases in which there is marked atrophy of the mucosa. For example, the nose may be broad and flat, the tip somewhat elevated, and the blood anemic. The anemia is secondary and not primary as in chronic rhinitis with collapse of the erectile tissue. The absorption of septic material and the loss of the respiratory functions of the nose are probably the chief causes of the anemia. It is a well-recognized fact that in mouth breathers from the presence of postnasal adenoids there is anemia, which is quickly cured after the removal of the adenoids.

The mucous membrane becomes atrophied in the later stages, and after a longer period the secretion and foul odor spontaneously cease and leave a comparatively clean but sclerotic membrane. The ozenic odor stops spontaneously after a number of years, hence it is a self-limited symptom. The mucous membrane, however, is left very much damaged. Its histological character and physiological function are changed or entirely lost.

The sclerosis and ozena in this type of atrophic rhinitis is in all probability due to a chronic sinuitis, or to other focalized suppurative processes, as has been shown by Grünwald in his work on *Nasal Suppuration.* In other words, the atrophy is not primary, but is secondary to a suppurative inflammation of the sinuses. Indeed, nearly all cases of atrophic rhinitis probably fall under this category. This subdivision of atrophic rhinitis is, therefore, from a clinical standpoint, of the greatest importance.

The *rationale* of the atrophic process is generally as follows:

The secretions from the sinuses, more particularly the fronta., ethmoidal, and sphenoidal, flow downward over the nasal membrane, where they dry, forming crusts. These undergo decomposition and irritate the underlying mucosa. There is, in addition, a mechanical

irritation from the shrinkage and contact of the crusts with the mucous membrane. The biochemical and mechanical irritation thus produced cause a proliferation of connective-tissue cells, which, when fully organized, contract and choke the normal tissues of the mucous membrane. Shrinkage and atrophy progress until the mucous membrane is replaced by a sclerotic tissue, devoid of mucous glands and columnar ciliated epithelium, pavement epithelium replacing the columnar type.

During the progress of the atrophic process the ozena is a symptom, but after the true mucous membrane is destroyed the mucous secretion and ozena disappear. Crust formation and ozena are self-limited phenomena, many years being required, however, to rid the patient of them.

Symptoms.—The symptoms vary with the state of advancement and activity of the process. The clinical picture presents the features shown in the comparative table given below. This is adapted from MacDonald's work on *Diseases of the Nose.*

Comparative Table of the Symptoms of Atrophic Rhinitis and Rhinitis with Collapse

Chronic Rhinitis with Collapse of the Erectile Tissue.	*Atrophic Rhinitis with Sclerosis and Mucous Secretion. Ozena.*
1. Chiefly in anemic women. The anemia is primary.	1. Chiefly in women and children: all subjects become anemic.
2. No peculiarity of physiognomy.	2. Small, sunken wide nose with wide nasal fossæ.
3. Mucous membrane anemic.	3. Mucous membrane anemic.
4. Collapse of erectile tissue; no tendency to atrophy.	4. Collapse of the erectile tissue with tendency to atrophy.
5. No ulceration.	5. Sometimes there is ulceration, and necrotic bone if the disease is of sinus origin.
6. Always bilateral, as it is of constitutional origin.	6. Usually bilateral; may be unilateral.
7. Spontaneous cure if the anemia is relieved.	7. After some years there is a tendency to improvement of the symptoms. The ozenic symptoms disappear as the atrophy becomes more complete.
8. Olfaction not affected.	8. Olfaction is often lost.
9. No characteristic odor.	9. Breath typically ozenic.
10. Little or no incrustation; if present, is limited to the anterior third of the middle turbinates.	10. Crusts are distributed over the entire mucous membrane.
11. Headache and dizziness absent.	11. Frontal headache and dizziness often present. Occipital headache may be present when the sphenoidal sinus is involved.

Treatment.—When seen in the early stage the treatment should aim at (*a*) the removal of the causes of the inflammation that produces the sclerotic process, and (*b*) intranasal cleanliness.

(*a*) **Removal of the Causes.**—The causes of the inflammation are numerous. Some have already been considered under acute catarrhal hyperplastic rhinitis, chronic suppurative sinuitis, and the congenital primitive nose with its pavement epithelium. Other causes are traumatism, deflections, and other obstructive lesions of the septum. By the removal of these predisposing causes of the inflammation, the sclerotic process may be modified or stopped altogether.

From the foregoing statements concerning focal suppuration within the sinuses and elsewhere in the nasal chambers, it is evident that in many cases the treatment should be addressed toward the cure of the suppuration of the sinuses, rather than to the atrophy resulting from it.

(*b*) **Intranasal Cleanliness.**—Intranasal cleanliness is obtained by the use of antiseptic douches containing a liberal amount of mild alkalies to soften and dissolve the crusts and tenacious mucopus. A solution of 8 grains of sodium bicarbonate to the ounce of water as hot as can be borne should be forcibly injected into the nostrils at frequent intervals during the day. A fountain syringe is well adapted for this purpose. The patient should be instructed to clear the nose by blowing after each injection. The injection may be administered by the physician at first, as the patient will not or cannot thoroughly cleanse his nose. To free the nostrils from crusts and tenacious mucus, a warm antiseptic aqueous solution of borax, sodium bicarbonate, oil of eucalyptus, carbolic acid, glycerin, and alcohol should be injected into the nostrils. A two-ounce hard rubber or an Alpha and Omega bulb syringe is well adapted for this purpose, as considerable force is necessary to dislodge the crusts.

Personally, I prefer to pack the nose with cotton-wool saturated with a 10 per cent. aqueous solution of ichthyol, which should be removed in from twenty to thirty minutes. The crusts, being softened, are easily detached by blowing the nose or by the use of a cotton-wound probe. This course of treatment, if faithfully carried out, will afford great relief. Mild astringent stimulating solutions, or powders, are of value in reducing the local infection. A powder containing 5 to 20 per cent. of silver nitrate, or a 1 to 2000 trichloracetic acid solution may be used for this purpose. The associated sinus disease should be treated as described under the Accessory Sinuses. Indeed, this is often the only method of treatment attended with success. Even this fails if the atrophy is far advanced.

Paraffin Injections in Atrophic Rhinitis.—Paraffin injections beneath the mucous membrane of the inferior turbinated body and of the septum have been used with great improvement of the symptoms. The crusts are either diminished or disappear altogether. Some writers recommend the use of paraffin in melted form, although the danger of thrombosis is ever present. More recently paraffin has been used in solid form in order to obviate this danger. A special syringe, adapted to the use of semisolid paraffin, has been devised by Dr. J. C. Beck for this purpose. With this device the danger of thrombosis is reduced to the minimum.

The injections should be made under local anesthesia. The amount injected at each sitting varies with the friability of the mucous membrane. In some cases only one or two minims or grains should be injected, a larger amount being liable to tear the mucous membrane. In other cases as much as one to two drams may be injected (Fig. 123). The injections should be made at intervals of from five to ten days, enough time being allowed between the sittings for the subsidence of the reaction.

Either the inferior turbinal (nasal concha) or the septum may be chosen for the site of the injections. The needle should be introduced a half-inch or more beneath the mucoperiosteum and a small amount of paraffin injected. It should then be withdrawn, a quarter of an inch and more paraffin injected, and so on until the needle is removed.

The effects produced are a lessening or the disappearance of the crusts, a thinning of the secretions, a sense of air passing through the nasal chambers, and occasionally edema of the eyelids. The good effects have remained for a period of two years and the indications are that they may last much longer. The lumen of the nasal chambers is diminished,

FIG. 123

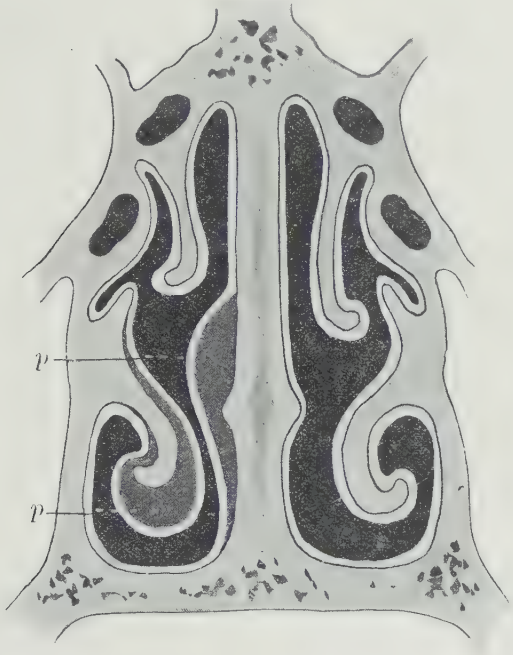


FIG. 123.—*p*, *p*, paraffin injected beneath the mucosa of the septum and the inferior turbinated body in atrophic rhinitis.

thus accounting in a measure for the lessened desiccation of the secretions. It is also quite probable that the irritation of the paraffin, a foreign body in the tissues, produces increased hyperemia and leukocytosis. Whatever the explanation may be, it appears that paraffin injections beneath the mucoperichondrium of the nasal septum and beneath the mucoperiosteum of the inferior turbinate materially improves the symptoms in atrophic rhinitis with incrustations. In those cases wherein the sinus origin of the suppuration and crusts is in doubt, and wherein the patient refuses operative interference on the sinuses when they are known to be the focal centre of the disease, paraffin injections

may be used with the reasonable assurance of an improvement of the symptoms, though a cure may not result.

SUPPURATIVE RHINITIS; NASAL SUPPURATION.

(A symptom, not a primary disease.)

Suppurative rhinitis has been described by various authors, notably by Bosworth in his work on the *Diseases of the Nose and Throat*. He described suppurative rhinitis in children as a primary disease, which, when neglected, results in atrophic rhinitis in adults. The trend of opinion is gradually relinquishing the view that primary suppuration of the nasal mucous membrane is often found. On the contrary, it is believed that it rarely exists except secondarily to sinuitis. Personally, I hold the latter view.

Pus in the nasal chambers is present in the later stages of acute coryza, which is an infectious disease and is usually complicated by a purulent infection of the sinuses. Purulent secretions may also accompany syphilitic, tuberculous, and gonorrheal processes in the nose. The specific exanthematous fevers are characterized by a purulent inflammation of the nasal and accessory sinus membranes. The various accessory sinuses, when affected by a purulent inflammatory process, discharge their purulent secretions into the nasal passages. Generally speaking, if after the nasal chambers are cleared of pus by mopping with a cotton-wound applicator the pus reappears within a few minutes in the middle meatus, it comes from the sinuses discharging into this meatus, namely, the frontal, anterior ethmoidal (including the bulla ethmoidalis), and the sinus maxillaris (antrum of Highmore). Occasionally one of the anterior ethmoidal cells discharges through the inner or median wall of the middle turbinate into the olfactory fissure or superior meatus. When the pus appears in the superior meatus, it is probably from the sinuses opening into the meatus, namely, the posterior ethmoidal and the sphenoidal sinuses. An occasional exception to this is when the sinus maxillaris (antrum of Highmore), the posterior and superior median wall of which is in relation to the superior meatus, discharges through a perforation into the superior meatus. Such a condition is rare, hence pus in this meatus as seen in the olfactory fissure is generally indicative of suppuration of the posterior ethmoidal and the sphenoidal sinuses. It is barely possible that there may be a focalized ulceration of the nasal mucous membrane in the superior meatus, and that the pus is from the meatus rather than the sinuses. It appears, therefore, that nasal suppuration is rarely, if ever, a primary disease, but that it is always, or nearly always, secondary to some other disease of the mucous membrane and bony walls of the nasal chambers or the accessory sinuses of the nose. Suppuration of the nose as a primary disease will not, therefore, be described, but the other diseases to which it is secondary are described, and the reader is referred to them for further information.

PLATE II

Anterior Reconstruction. (H. W. Loeb.)

On account of the multiplicity of lines, the individual ethmoidal cells are not shown; however, the two groups are represented, the anterior being lined horizontally and the posterior perpendicularly. The left sphenoidal sinus lies far above the right; its inner wall extends almost as far to the right as the outer wall of the right sphenoidal sinus.

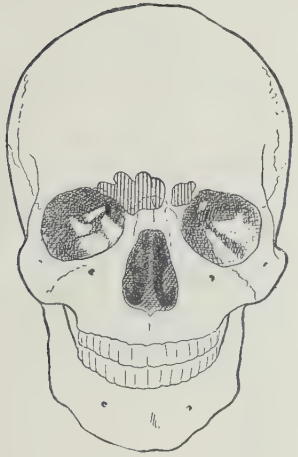
PLATE III

Left Lateral Reconstruction. (H. W. Loeb.)

In this and Plate II the frontal sinus is colored yellow, the maxillary purple, the sphenoid green, and the ethmoid red, the anterior group being lined horizontally and the posterior group perpendicularly. The ethmoidal cells are to be noted in two groups, the anterior two in number, and the posterior three. The first anterior cell is shown displacing the anterior wall of the frontal. The frontal is seen opening into the frontonasal canal. The antero-inferior wall of the second ethmoid constitutes the bulla ethmoidalis.

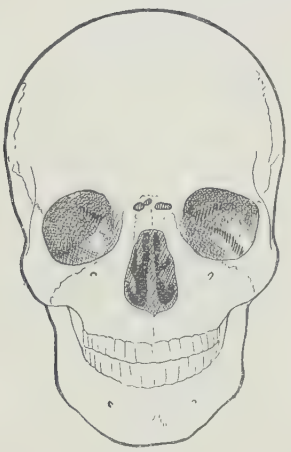
PLATE IV

Fig. 1



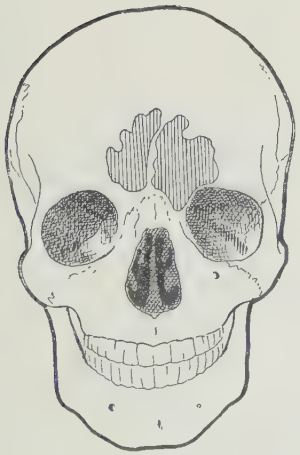
Large right frontal and a small left frontal sinus. (From author's skiagraph.)

Fig. 2



Absence of the frontal sinuses in a patient aged twenty-nine years. Small anterior ethmoidal cells are shown. This patient had extensive necrosis of the ethmoidal and sphenoidal bones, and secondary mastoiditis complicated by a brain abscess in the motor area for the arm and leg. The arm and leg on the opposite side were partly paralyzed. The ethmoidal and sphenoidal sinuses, mastoid and brain abscess were successively operated upon without result. (Author's case.)

Fig. 3



Very large frontal sinuses. (From author's skiagraph.)

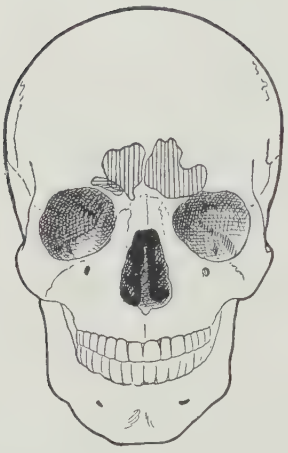
Fig. 4

Very large irregular right frontal and a small left frontal sinus. (From author's skiagraph.)

The Distribution of the Frontal Sinuses as Shown by Skiagraphy.

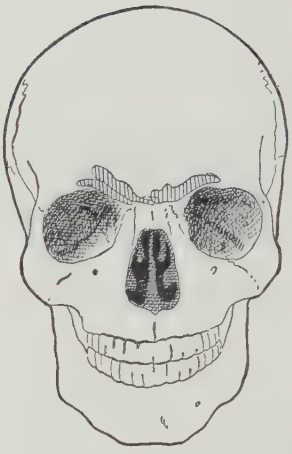
PLATE V

Fig. 1



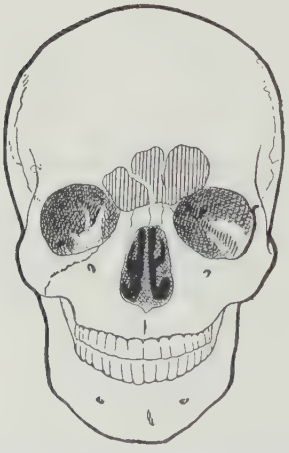
Large frontal sinuses and an anterior ethmoidal cell extending well over the right orbit. (From author's skiagraph.)

Fig. 2



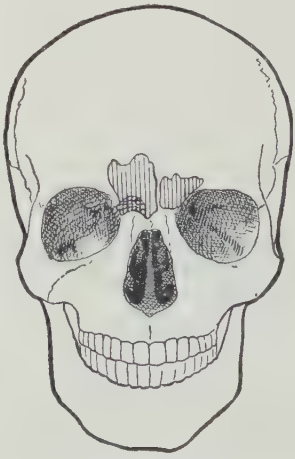
Narrow longitudinal frontal sinuses, the right having an ethmoidal cell encroaching upon its floor. (From author's skiagraph.)

Fig. 3



Very large left frontal sinus, almost divided by a septum. The left sinus extends about one-half inch beyond the median line. (From author's skiagraph.)

Fig. 4

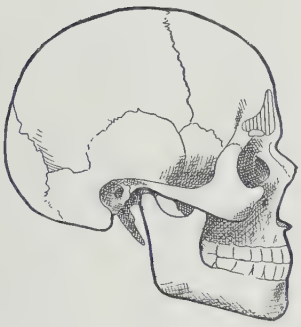


Large right frontal sinus with an anterior ethmoidal cell (bulla frontalis) encroaching upon its floor. (From author's skiagraph.)

The Distribution of the Frontal Sinuses as Shown by Skiagraphy.

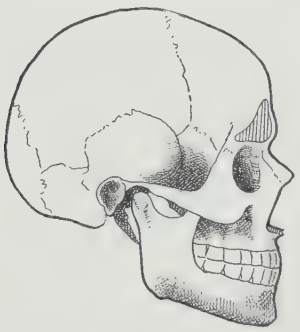
PLATE VI

FIG. 1



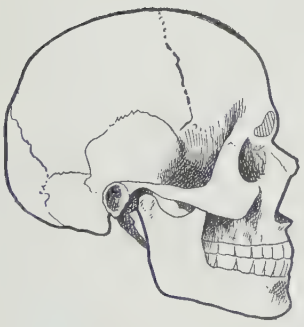
Side view of frontal sinus with great depth and upward extension. A small anterior ethmoidal cell, the bulla frontalis, encroaches upon its floor. (From author's skiagraph.)

FIG. 2



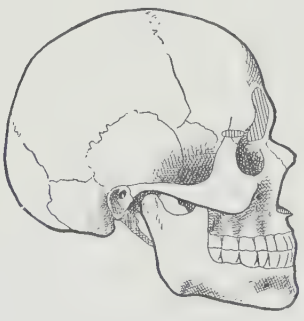
Another large frontal sinus with marked backward extension over the orbit. (From author's skiagraph.)

FIG. 3



Side view of the frontal sinus with limited upward extension and moderate backward extension. (From author's skiagraph.)

FIG. 4

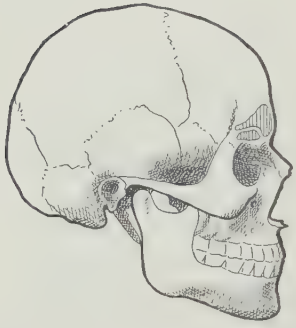


An unusual downward extension of the frontal sinus. (From author's skiagraph.)

The Anteroposterior Extension of the Frontal Sinuses as Shown by Skiagraphy.

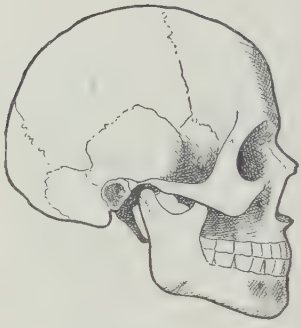
PLATE VII

Fig. 1



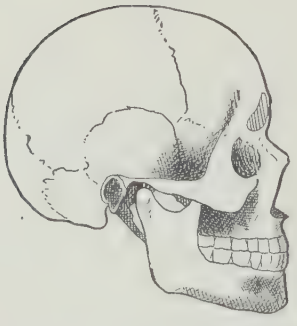
Frontal sinus with extreme extension backward, and with a large anterior ethmoidal cell encroaching upon the posterior portion of its floor. (From author's skiagraph.)

Fig. 2



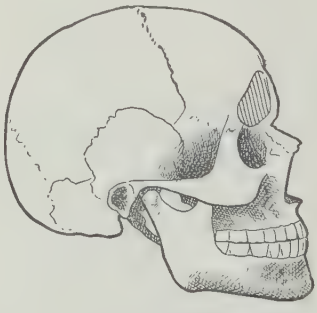
Side view showing absence of the frontal sinuses in a patient aged twenty-nine years. Anterior view shown in Plate IV, Fig. 2. (From author's skiagraph.)

Fig. 3



Side view showing a frontal sinus of moderate depth. (From author's skiagraph.)

Fig. 4



An extremely large and deep frontal sinus. (From author's skiagraph.)

The Anteroposterior Extension of the Frontal Sinuses as Shown by Skiagraphy.

CHAPTER IX

THE INDIVIDUAL SINUSES

The sinuses are divided for clinical purposes into two groups, namely, the anterior and the posterior sinuses. The anterior group is composed of the frontal, the anterior ethmoidal, and the maxillary sinuses. Hajek calls this group Series I. The posterior group is composed of the posterior ethmoidal and the sphenoidal sinuses, and is called Series II.

Our knowledge of the etiology, symptomatology, pathology, and surgical treatment of the sinuses has increased so greatly during the last ten years that it seems proper to depart from the traditional manner of presenting this subject, wherein each sinus is separately described and treated. As a matter of fact, a single sinus is rarely diseased, two or more being commonly affected at the same time. Indeed, it is not uncommon to find all the sinuses on one side of the head affected. The maxillary sinus is perhaps more often affected singly than either of the other sinuses. This is accounted for by the fact that in about one-half of the cases it is infected from the teeth rather than from the nose, whereas the other sinuses are nearly always infected from the nose. Having a common source of infection, they are, therefore, more often simultaneously diseased.

For this reason a general discussion of inflammation of the sinuses is to be preferred to a discussion of each sinus individually. Nevertheless, it will be advantageous to present the peculiar symptoms and other considerations of each sinus separately. The following considerations are therefore to be read in conjunction with the general description which follows.

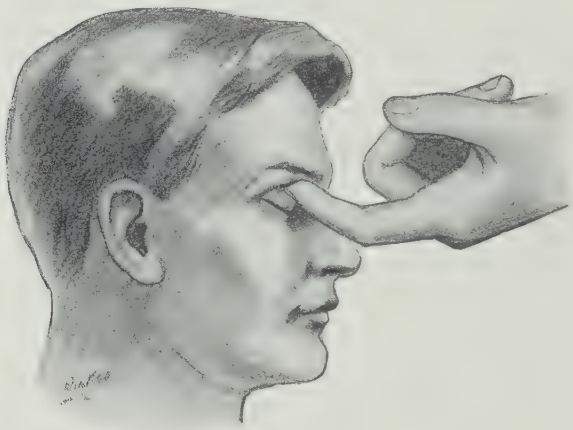
SERIES I

Frontal Sinus.—The frontal sinus is an extension upward of the ethmoidal cells between the plates of the frontal bone. The extension occurs at about the age of puberty, hence in infants and young children the frontal sinuses are absent. The size and shape of the frontal sinuses vary greatly in different individuals, and indeed the two sinuses often vary greatly in the same individual. References to Plates II, III, IV, V VI, and VII show some variations in the frontal sinuses, the drawings being taken from skiagraphs of some of the author's cases. These variations are of surgical interest, as the difference in size will often determine the method of operating. If there is a large and deep frontal sinus, great external deformity may follow the complete removal of the anterior wall. In such a subject the operation may be so executed as to avoid, or to greatly reduce, the probability of marked disfigurement.

H. W. Loeb's projections of the sinuses (Plate II and III) show more clearly than any other work the relations of the sinuses to one another and to the structures of the nose. The anteroposterior and lateral projections are shown. Plates IV, V, VI and VII also give a good idea of the distribution of the sinuses.

Skiagraphy.—The skiagraphic plate, if the exposure is properly made, affords good information concerning the presence or absence of disease in all except the sphenoidal sinus. It is not yet known what causes the cloudy appearance when the sinus is diseased. Coakley says it is not known whether it is due to the thickness of the inflamed membrane, to the presence of pus, or to the changed condition of the bone. I have a skiagraph of a patient affected with a severe chronic catarrhal sinuitis upon whom I performed a double Killian operation, in which the right

FIG. 124



The correct method of making pressure under the floor of the frontal sinus. Pressure is often made under the supra-orbital ridge, whereas it should be made much deeper.

frontal sinus as shown by the plate was cloudy, but less so than the left. Upon operating the right sinus was found to be free of pus, and its periosteum and mucous membrane were entirely gone. The bone was chalky white and slightly roughened. The left sinus was free of pus, but was filled with granulation tissue and viscid mucous secretion. The patient had complained for several months of an acrid secretion which irritated the nasal mucosa. This case is cited here, as it is unique, and demonstrates that a frontal sinus devoid of membrane periosteum, and purulent secretion gave a cloudy effect in the skiagraph, though not so pronounced as that given by the sinus in which the membrane and granulations were present. Pus was not present in either sinus.

Tenderness upon Pressure.—Tenderness over the frontal bone is rarely present in frontal sinuitis except in very acute cases with obstructed drainage. Tenderness is often present, however, when pressure is made

against the floor of the affected sinus near the inner angle of the orbital cavity (Fig. 124). The finger tip should be placed well under the roof of the orbit and the pressure directed upward. Pain is thus often elicited even in chronic catarrhal cases. Tenderness in this region does not, however, always indicate disease of the frontal sinus, as the anterior ethmoidal cells sometimes project beneath the floor of the sinus.

When such an anatomical deviation is present the surgeon may be led to a wrong conclusion. This difficulty may be obviated by having a skiagraph made, as it will aid in determining the position and condition of the frontal and anterior ethmoidal cells.

The tenderness present in frontal sinuitis is so nearly in the same position as that in ethmoidal sinuitis that a careful distinction should be made. In ethmoidal sinuitis the tenderness is usually located a little above the median palpebral commissure (inner canthus) of the eye and a little deeper in the orbital cavity than the canthus. The pressure should be made inward toward the median line, rather than upward, as in testing the frontal sinus.

Redness and Swelling.—Redness and swelling over the frontal region are only present in severe acute inflammation of the frontal sinus where the bone is affected by an infective osteomyelitis and the skin has yielded to the inflammatory process. There are perhaps a hundred cases of frontal sinuitis in which the redness and swelling are absent to one in which they are present. The day is past when a surgeon should wait for such symptoms before deciding to operate upon the frontal sinus. There are other positive indications of disease of the sinus to guide him to a diagnosis and to a choice of the mode of treatment.

Mucous Discharge.—While catarrhal inflammation of the sinuses is generally referred to in text-books, no clear idea of the symptomatology and diagnosis is given. The presence of pus in the nose has generally been considered an essential requirement in making a diagnosis. I have found it almost as easy to diagnosticate sinuitis without pus as with it. The symptoms are much the same as those in purulent sinuitis, except that pus is absent. The secretion is mucous or seromucous in character, and might easily escape observation. The patient often complains of a burning sensation in the anterior portion of the nasal passages or of fissures or excoriations at the margin of the nostrils as a result of the acrid catarrhal discharge.

Headache.—The patient generally complains of frontal headache which is limited to, or originates on, the side affected. The headache is often more severe during the night, especially upon awaking while in bed, or in the morning, than at other times. It is often confounded with eyestrain. Headache due to eyestrain is generally relieved upon closing the eyes, especially upon retiring for the night. The headache caused by frontal sinuitis (catarrhal or suppurative) is not aggravated by theatre-going; whereas if due to eyestrain, it is thereby aggravated.

Dizziness; Vertigo.—Dizziness or vertigo of slight degree is present in most cases, severe in others. It is often present in simple catarrhal inflammation, as well as in suppurative inflammation of the frontal and

ethmoidal sinuses. It is especially aggravated by stooping, or, if in a stooping posture, upon assuming the erect posture. Careful inquiry is often necessary to elicit this symptom, as the patient does not consider it of any significance.

Ocular Symptoms.—According to Fish, Zeim, Wood, Stucky, Coffin, and others (Eye in Relation to the Sinuses), inflammation of the frontal or any other sinus may give rise to morbid processes in any of the structures of the eye. This is accounted for by the free anastomosis of the veins of the sinuses with the ophthalmic vein. Congestion in the sinuses causes a like condition in the eye. Infection and toxemia are thereby favored; papillitis, choroiditis, optic neuritis, iritis, keratitis, etc., thus becoming established.

Intracranial Complications.—Extradural and brain abscess, meningitis, and sinus thrombosis may arise from sinuitis. Inasmuch as the posterior wall of the frontal sinus is thinner than the external or anterior wall, it is curious that intracranial complications are so rare. The superior, longitudinal, and the cavernous sinus occasionally become thrombosed in frontal sinuitis. Meningitis, which has its origin in the sinuses, is more frequently reported now than formerly, a fact significant of a better understanding of the subject.

Anterior Ethmoidal Sinuses.—The anterior ethmoidal cells vary in number from two to eight, and are smaller than the posterior cells. They all drain into the middle meatus. According to Logan Turner, the frontonasal canal opened into the infundibulum in about one-half of the specimens examined, and directly into the middle meatus in the remainder. The anterior cells are separated from the posterior cells by a thin transverse bony partition. The attachment of the middle turbinated body to the external wall of the nose also marks the line of division between the anterior and the posterior group of cells. The anterior cells lie in front of and below it, while the posterior cells lie above and behind it. Clinically the two groups of ethmoidal sinuses are, therefore, divided into anterior and posterior cells. The anterior cells belong to Series I, while the posterior cells belong to Series II.

Accessory ethmoidal sinuses are sometimes present in the middle turbinate and in the uncinate process, and when present drain into the middle meatus and belong to the anterior group or Series I.

The upper wall of the ethmoidal cells is a rather dense but thin plate of bone. The cribriform plate is not covered by the cells, but is freely exposed in the attic of the nose. While the bone is dense and not easily fractured by ordinary force exerted during an operation, its numerous openings render it a possible atrium for the conveyance of infection to the meninges. The outer wall of the ethmoidal sinuses is the *os planum* or *lamina papyracea* of the ethmoidal and the lacrymal bones. These plates of the bone are extremely thin, and form the inner wall of the orbital cavity. Should this plate of bone be perforated, orbital cellulitis, with protrusion of the eyeball, might result. In two of my cases orbital emphysema followed the ethmoidal operation.

In Fig. 125 is shown a case of ethmoidal suppuration in which the

lacrymal bone was carious and perforated. When first seen there was a large nipple-like projection of the skin at the inner angle of the orbit, or lateral wall of the nose, in this region The right eyelid was swollen and closed, while the left was less swollen and partially closed. The upper and lower lids of both eyes were discolored purple. Protrusion of the eyeballs was absent, as orbital cellulitis was not present. Had the perforation occurred more posteriorly through the os planum, orbital cellulitis would in all probability have occurred.

The patient had a similar attack one year previous to this one. The swelling subsided, but the nasal discharge continued, and the eye was uncomfortable.

FIG. 125

Empyema of the ethmoidal sinuses, with perforation through the lacrymal plate at the inner canthus of the right eye and marked bulging at this point. Both upper eyelids are edematous and purple. The right eye is entirely closed, the left almost. One year previously had a similar attack following scarlet fever. (Author's case.)

FIG. 126

Same case six days after operation. External wound gradually filled in by granulation and became closed in two months. (Author's case.)

Skiagraphs showed marked cloudiness in the ethmoidal region on the right side, while on the left it was less cloudy. The frontal sinuses were absent, or if present were very small. The lower meatus of the nose was quite open. Frontal headache and dizziness were prominent symptoms.

The nipple-like projection was incised at once and discharged a half-ounce of thick yellow pus. On the following day, under general anesthesia, the region was exposed by an external skin incision extending from a point below the nipple-like tumefaction to the middle of the right eyebrow. The lacrymal bone was almost entirely destroyed by necrosis. The frontal process of the maxilla was removed with rongeur forceps, thus fully exposing the anterior ethmoidal cells to operative interference The entire ethmoidal labyrinth, including the middle turbinate, was

removed. A curette (Fig. 127) was also used through the anterior nasal opening, to make sure that no remnants of the cells were left. The cranial plate and the os planum were carefully but thoroughly curetted until they were smooth.

The left side was operated on through the nose, the middle turbinate and the ethmoidal cells being removed in their entirety, in so far as they could be reached with the curette by this route.

Fig. 127

The author's ethmoid curette.

Fig. 126 shows the patient one week after operation. The edema and discoloration of the eyelids had entirely disappeared, and the wound in the lacrymal region on the right side permitted a clear view of the interior of the nose. The marked change in the facial expression is suggestive of the improved condition of the patient.

Fig. 128

Showing the thin orbito-ethmoidal wall partially destroyed. During ethmoiditis this wall may be broken or perforated, and give rise to orbital cellulitis. (Author's specimen.)

The Maxillary Sinus (Antrum of Highmore).—The maxillary sinus, the third and last sinus belonging to Series I, is the largest, and, according to the prevailing opinion, is more frequently diseased than either of the other sinuses in both series. Personally, I question this statement, as according to my own observations the ethmoidal and frontal sinuses are more frequently involved. Our knowledge of the symptomatology of disease of the sinuses in general has greatly increased during the past five or ten years, with the result that ethmoidal, sphenoidal, and frontal sinuitis are diagnosticated twenty times as often as they were ten years ago. While the antrum is still a frequent seat of disease, the ethmoidal and the frontal sinus occupy a more important place. The diagnosis of antral inflammation has been understood for many years, and this has given rise to the impression that it is much more common than inflammation in the other sinuses. It may be infected from the nose or the teeth, the cases probably being about equally divided between these two sources of infection. On account of the dental origin of so many cases of maxillary sinuitis, it is more often affected singly than either of the other sinuses, in which the infection is almost always of nasal origin.

When the infection is of nasal origin, quite naturally more than one group of sinuses is simultaneously affected.

The ostium maxillare is situated in the upper portion of the naso-antral wall as far removed from the floor of the sinus as possible. This apparently renders the drainage of the secretions quite difficult or impossible, except as they overflow when the antrum is filled. This is not the case, however, as there is but little secretion in the sinus in health—only enough to keep the mucous membrane moist. The epithelium of the antral mucous membrane is of the modified ciliated columnar variety, though it is but slightly developed and in patches. The wave-like motion of the ciliæ aids in carrying the scanty secretions to the ostium maxillare at the top of the sinus, where it is discharged through the infundibulum into the middle meatus.

In the course of severe or long-continued inflammation of the mucous membrane of the antrum, the ciliæ are injured or destroyed, and the secretions are retained in the antrum because they are not carried to the ostium maxillare. The secretions are greatly increased in quantity, a fact which still further tends to promote the accumulation within the sinus.

The second bicuspid and the first and second molar teeth are in close relation to the floor of the sinus. Indeed, they sometimes project into the bony cavity, being only covered by mucous membrane. A suppurative process around the root of either of these teeth might easily affect the mucous membrane of the sinus through the lymphatics and blood-vessels. Indeed, an infection of the crown of the teeth may extend through the lymphatics to the antrum.

The superior wall or roof of the sinus is crossed in its central portion by the infra-orbital nerve, which lies in a groove on the broad inferior side of the plate of bone. It is covered by mucous membrane, and may be easily injured during the curettement of the sinus.

As it is a nerve of sensation rather than of motion, it regenerates readily after being injured, even if long portions of it are removed. Motor nerves do not thus readily repair.

SERIES II

Series II is composed of the posterior ethmoidal and the sphenoidal sinuses, and their ostei open into the superior meatus of the nose.

Posterior Ethmoidal Sinuses.—The posterior ethmoidal are usually fewer in number and larger in size than the anterior ethmoidal cells. Sometimes they occupy nearly all the ethmoidal labyrinth, extending to the anterior portion of the nose, and sometimes the anterior cells extend backward almost to the sphenoidal bone.

The ostia open into the posterior portion of the superior meatus and drain upon the posterior half of the middle nasal concha (turbinated body). As the middle turbinate slopes slightly downward and backward, the secretion flows toward the posterior choana, though it also flows over the median border of the turbinate through the olfactory fissure or

space between the turbinate and the septum, hence a purulent secretion in the olfactory fissure is usually indicative of posterior ethmoidal suppuration. It may, however, indicate sphenoidal disease, or a combined empyema of the ethmoidal and sphenoidal sinuses. The secretions may also be forced into this position from the middle meatus by snuffling the nose.

The ostia of the posterior cells are not visible by either anterior or posterior rhinoscopy, nor are they accessible to the probe or cannula.

The symptoms of posterior ethmoidal suppuration are not so distinct as those in either of the cells comprising Series I. As the posterior cells are deeply situated, external tenderness is not present. Exophthalmos may result from the retention of the purulent secretion in the cells, the os planum forced outward behind the eyeball, causing it to protrude forward. This also gives rise to diplopia and strabismus and to a circumscribed visual field, especially for colors. The ocular disturbances are extremely rare in proportion to the number of cases in which the posterior ethmoidal cells are diseased. According to my own clinical observations, the ethmoidal sinuses (anterior and posterior) are more often diseased than the maxillary sinus, which is generally regarded as the most frequently affected. The ethmoidal sinuses are so situated in the upper and narrow portion of the nasal chambers that a moderate deviation of the septum or an enlargement of the middle turbinate closes the olfactory fissure and thus blocks ventilation and drainage of the superior meatus and accessory cells. For these reasons the posterior ethmoidal cells are often the seat of disease.

The secretion in the posterior portion of the olfactory fissure is significant of ethmoidal suppuration, though the pus may come from the sphenoid. Indeed, the posterior ethmoidal and sphenoidal cells are so closely associated that when one is diseased both are often affected. A postrhinoscopic examination showing purulent secretion on top of the middle turbinate is almost certain evidence of disease of the posterior ethmoidal and sphenoidal cells. Crusts and secretions in the vault of the epipharynx are likewise indicative of the same affection.

Sphenoidal Sinus. —The *ostium sphenoidale* is situated in the anterior wall of the sphenoidal sinus near the top of the cavity, though it is occasionally a little lower down. It is near the septum of the nose and is hidden from view by the close approximation of the middle turbinate to the septum. If there is marked atrophy of the turbinate, or if the septum deviates to the opposite side, it may be seen by anterior rhinoscopy. The opening varies from $\frac{1}{2}$ to 4 mm. in diameter.

The purulent secretion flowing from the ostium either drains directly through the posterior choana into the epipharynx or on to the posterior end of the middle turbinate. Ocular inspection can usually only be made after the removal of the entire middle turbinated body.

The pain or headache occurring in sphenoidal inflammation is usually felt in the occipital region on the affected side, though in some cases it is diffused and ill defined. Catarrhal inflammation causes the same headache as suppurative inflammation, though it may not be so severe.

The ocular symptoms usually ascribed to suppuration of the sphenoidal sinus are those dependent upon the compression of the optic and oculomotor nerves. The optic nerve passes over the roof of the sinus, hence in closed empyema in which the thin bony wall of the roof is softened, compression or even destruction of the optic nerve may take place. Optic neuritis may be followed by atrophy and blindness. Optic neuritis may be toxic in origin, the noxa originating in the infected sinuses. I have seen several cases of neuritis and blindness which were apparently of toxic origin, as there was no retention of secretion. If the pressure reaches the sphenoidal fissure, the oculomotor nerves, the third, fourth, and sixth, become involved and strabismus in some form follows. Intense neuralgia may result from a neuritis of the ophthalmic division of the fifth nerve.

Other ocular lesions arising in the course of inflammatory diseases of this and all the other sinuses are referred to in the paragraph on the Eye in Relation to the Sinuses.

DIFFERENTIAL DIAGNOSIS

To illustrate the methods of differential diagnosis, a series of hypothetical cases will be given, assuming the symptoms characteristic of the simple and combined empyemas of the various sinuses in the open, closed, and latent forms.

Simple empyema refers to those cases which are limited to one group of cells, as the maxillary sinus, frontal, anterior ethmoidal, posterior ethmoidal, or the sphenoidal sinus.

Open empyema refers to an empyema, either simple or combined, in which the ostia are open and permit of drainage and ventilation.

Closed empyema refers to those cases in which the ostia are closed by pathological changes and the secretions are retained and cause pressure.

Latent empyema refers to those cases in which the ostia are open, but the secretion is so slight that it is not demonstrable, except by irrigation of the affected sinus.

The ostia of the sinuses are so situated that they drain into either the middle or the superior meatus of the nose. The sinuses situated anteriorly drain into the middle meatus, while those situated posteriorly drain into the superior meatus.

The anterior group, or those draining into the middle meatus, are the antrum, the frontal and the anterior ethmoidal cells. These have been designated by Hajek as Series I.

The posterior group, or those draining into the superior meatus, are the posterior ethmoidal and the sphenoidal sinuses. These are designated as Series II. For the sake of brevity and clearness these terms will be continued. Having defined the terms, we are ready to recite a series of hypothetical cases, illustrative of the symptoms and procedures necessary to arrive at a positive differential diagnosis between empyema of the various sinuses or combinations of them.

Case I.—(*a*) Unilateral discharge from the nose.

(*b*) No pain.

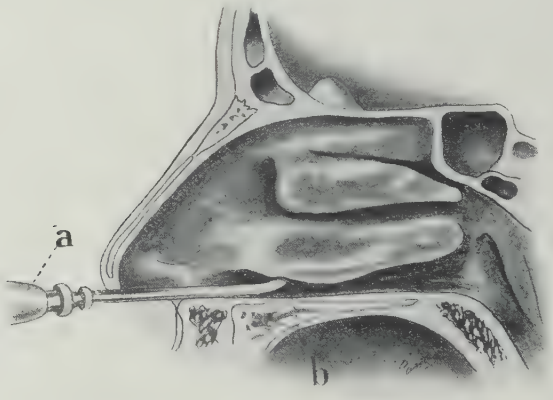
(*c*) Subjective fetid odor.

(*d*) There is an ulcer at the root of the second bicuspid tooth on the side of the nasal discharge.

(*e*) Anterior rhinoscopy shows pus in the middle meatus.

The conclusion, based upon the above data, is that one or more of the anterior group of cells, Series I, is involved. While the ulcerous bicuspid suggests the antrum as the sinus most probably affected, it is by no means proved nor are the frontal and anterior ethmoidal sinuses known to be free. To differentiate still further the focal centre of infection the following procedure must be instituted:

Fig. 129



Introducing a trocar and cannula into the maxillary antrum beneath the inferior turbinate for diagnostic purposes.

Remove the secretions from the middle meatus with the douche or a cotton-wound probe, and place the patient in Escat's position, *i. e.*, the head thrown forward with the affected side turned upward to help the flow of pus from the antrum. After the patient has remained in this position for a few minutes the middle meatus should be reëxamined, and if pus is found, the antrum is probably involved. This is not absolutely established, however, as the pus might have come from the frontonasal canal. To establish still further the diagnosis, introduce a cannula and trocar through the naso-antral wall in the inferior meatus, under cocaine anesthesia (Fig. 129), and irrigate the antrum. If pus is found the antrum is involved. The diagnosis is not yet complete, as it remains to be demonstrated whether the frontal and anterior ethmoidal cells are affected. If after thorough irrigation of the antrum pus does not reappear in the middle meatus, the probabilities are strongly in favor of a simple empyema of the antrum. This is true in view of the fact that the flow of pus from the frontal sinus is nearly constant, as its outlet when the patient is in a sitting posture is usually in the most dependent

PLATE VIII

Transillumination of the Antrum.

Right side normal, *i e.*, pupillary reflex and crescent of light present. Left side diseased.

portion of the sinus. In this case pus does not reappear in the middle meatus for several hours, unless the patient assumes Escat's position, hence the condition is probably a simple empyema of the antrum.

To strengthen the diagnosis still further, *transillumination* of the antrum and frontal sinus should be performed. If the side involved shows opacity over the lower eyelid, a non-luminous pupil, and the absence of the sense of light with the eyes closed, empyema of the antrum is indicated. If, in addition, transillumination of the frontal sinus is negative, the diagnosis of a simple empyema is fairly well established.

The anterior ethmoidal cells are still to be considered. Transillumination does not help us here. The bulla ethmoidalis belongs to the anterior ethmoidal cells, and if it is enlarged toward the septum, or downward against the uncinate process, it is probable that the anterior ethmoidal cells are involved.

If pus is removed by irrigation from the frontal sinus, the case is one of combined empyema of Series I. Skiagraphy shows the frontal and ethmoidal areas clear while the antrum upon the affected side is cloudy.

Diagnosis.—Simple, open empyema of the maxillary antrum.

Case II.—(*a*) Unilateral discharge of pus from the nose.

(*b*) Dull aching pain in the left cheek bone.

(*c*) Pus in the middle meatus.

(*d*) Slight tenderness over the cheek bone on pressure.

(*e*) Case under observation for several days; pus not always found in the middle meatus.

(*f*) Outer nasal wall on left side bulges toward septum.

(*g*) Pus occasionally discharged in great quantities, after which the dull ache in the malar region is relieved.

After performing the procedures described in Case I the purulent secretion is excluded from the frontal and anterior ethmoidal cells, and is localized in the maxillary antrum. The retention of the purulent secretion gives rise to the pain and tenderness over the left cheek bone and to the bulging of the outer nasal wall toward the septum. At times the pressure of the purulent secretion was great enough to force it either through the ostium maxillare or the accessory ostia, which were closed by the swollen mucous membrane. The pain caused by the pressure was relieved after each spontaneous discharge.

Diagnosis.—This is a case of simple, closed empyema of the antrum.

Case III.—(*a*) No nasal discharge.

(*b*) There is a previous history of nasal discharge from the right side.

(*c*) Frequent attacks of frontal headache on the right side.

(*d*) Mental depression.

(*e*) Aprosexia.

(*f*) Transillumination of antrum and frontal sinus is negative.

(*g*) Pus not present in either the middle meatus or the olfactory slit.

(*h*) Irrigation of the sinus through a puncture in the inferior meatus (Fig. 129) shows a very small amount of pus.

(*i*) Irrigation of the frontal and anterior ethmoidal cells is negative.

(*j*) Irrigation of antrum continued until pus disappears.

(*k*) Supra-orbital pain, mental depression, and aprosexia disappear.

(*l*) Skiagraph shows cloudiness of antral area, while the frontal and ethmoidal are clear.

Diagnosis.—Latent empyema of the maxillary sinus.

Case IV.—(*a*) Unilateral nasal discharge.

(*b*) Supra-orbital pain and tenderness on percussion.

(*c*) Pressure on the roof of the orbit (floor of frontal sinus) elicits pain.

(*d*) Pus present in the middle meatus.

(*e*) When wiped away it reappears after a few minutes.

(*f*) Escat's position of the head has no influence on the flow of pus.

(*g*) Lying upon the back checks the flow.

(*h*) Frontal headache beginning on the affected side, more marked in the morning.

(*i*) Dizziness upon stooping.

(*j*) Transillumination shows the crescentic light over the lower eyelid, the red pupillary reflex, and the sense of light in both eyes with the lids closed.

(*k*) Transillumination of the frontal sinus seems to show diminished luminosity on the affected side, although the difference between the two might easily be accounted for by anatomical variations.

(*l*) Puncture of maxillary sinus through the inferior meatus negative.

Fig. 130

Frontal sinus cannula.

(*m*) The cannula (Fig. 130) is introduced into the frontonasal canal and irrigation through it brings pus. Pus reappears in the middle meatus in a few minutes.

(*n*) Skiagraphs show cloudiness of the frontal sinus, the ethmoidal and antrum being clear.

Diagnosis.—Simple open empyema of the frontal sinus.

Case V.—(*a*) Constant nasal discharge on the right side.

(*b*) Supra-orbital headache on the right side.

(*c*) Tenderness and swelling over the right eyebrow.

(*d*) Anterior rhinoscopy; septum deviated to right in the region of the middle turbinate. Polypi in the middle meatus on right side.

(*e*) Probe shows polypi attached to uncinate process and the middle turbinal.

(*f*) Provisional diagnosis: Series I involved, probably localized in the frontal or the frontal and anterior ethmoidal sinuses.

(*g*) Transillumination of maxillary sinus shows faint crescent and pupillary reflex. Frontal sinus opaque.

(*h*) Polypi removed.

(*i*) Maxillary sinus punctured through inferior meatus and odorless pus is washed out.

(*j*) Frontal sinus irrigated through cannula. Pus abundant.

(*k*) Frontal sinus irrigated daily, maxillary occasionally; pus absent in maxillary after the first irrigation.

(*l*) At end of six weeks frontal sinus still discharges pus.

(*m*) Radical external operation; caries and polypi found in frontal sinus.

Diagnosis.—Empyema of frontal sinus with secondary involvement of the maxillary sinus, which acts as a reservoir, but is not a focal centre of disease.

Case VI.—(*a*) Patient complains of purulent crusts in the right nostril and in the epipharynx on rising. Hawks up crusts from the epipharynx.

(*b*) Dull headache variously located; sometimes it is frontal, then vertexial, and then occipital.

(*c*) Mental depression and aprosexia.

(*d*) Anterior rhinoscopy; septum deviated to right in region of middle turbinal. Olfactory slit narrow and filled with pus and crusts. Small polypi springing from above the middle turbinal.

(*e*) Posterior rhinoscopy shows purulent secretions flowing over the posterior end of the right middle turbinal and the posterior epipharyngeal wall. Crusts not found, as they form at night when the position of the head and the quietness of sleep favor accumulation.

(*f*) Middle meatus free from pus.

(*g*) Provisional diagnosis: Empyema of Series II.

(*h*) A cannula is passed into the sphenoidal sinus through its ostium. Irrigation shows no pus.

(*i*) A curved silver probe introduced through the olfactory slit shows bare, rough bone in the superior meatus.

Diagnosis.—Open empyema of the posterior ethmoidal cells. The irrigation of the sphenoidal sinus eliminates it from consideration, and as Series II is only composed of the sphenoidal and posterior ethmoidal sinuses, the empyema is located by exclusion in the posterior ethmoidal cells. This is still further substantiated by the presence of bare, rough bone in the superior meatus.

Case VII.—(*a*) Patient complains of the formation of crusts in the epipharynx, also of postnasal "dropping."

(*b*) A subjective sense of odor is present, even in the absence of such an odor.

(*c*) Vertexial and occipital headache.

(*d*) Field of vision, especially for colors, diminished.

(*e*) Mental depression.

(*f*) Anterior rhinoscopy; olfactory slit occasionally filled with pus, though it is usually clear.

(*g*) Probing shows the mucous membrane of the superior meatus intact, while probing of the sphenoid sinus shows roughened bone and bleeding.

(*h*) Posterior rhinoscopy; purulent secretions on posterior end of right middle turbinated body and upon the posterior wall of the epipharynx.

(*i*) Irrigation of the sphenoidal sinus shows pus in considerable quantities.

(*j*) Transillumination of maxillary and frontal sinuses negative.

(*k*) Examination of the fundus oculi shows slight papillitis.

Diagnosis.—Open empyema of Series II, probably focalized in the sphenoidal sinus. If the treatment of the sphenoid is followed by the disappearance of all symptoms, the diagnosis is positive. If the purulent discharge continues, the posterior ethmoidal cells should be removed; and if a cure follows, the diagnosis of combined empyema of the sphenoidal and posterior ethmoidal sinuses is established.

Case VIII.—(*a*) Intense headache at the vertex and occiput.

(*b*) Crust formation and postnasal dropping, yellow in color.

(*c*) Subjective sense of odor.

(*d*) Sudden blindness in the right eye.

(*e*) Great mental depression and aprosexia.

(*f*) Dizziness complained of.

(*g*) Anterior rhinoscopy shows pus and crusts in the olfactory fissure.

(*h*) Transillumination of the maxillary and frontal sinuses is negative.

(*i*) Probing of the middle and superior meatuses is negative.

(*j*) Cannot locate the ostium of the sphenoid on account of the great swelling.

(*k*) The middle turbinate is removed and the ostium sphenoidalis is filled with granulation tissue bathed in pus.

(*l*) The anterior wall of the sphenoid is removed, the cavity curetted, and granulation tissue and pus are found in considerable quantities.

(*m*) After the removal of the middle nasal concha (turbinated body) no pus is seen coming from the region of the posterior ethmoidal cells.

Diagnosis. Simple closed empyema, granulations, and caries of the walls of the sphenoidal sinus on the right side.

The sudden blindness may be accounted for by pressure upon and inflammation of the optic nerve, or by venous stasis or toxemia.

Case IX.—(*a*) Supra-orbital, vertexial, and occipital headache.

(*b*) Purulent discharge from the right nostril into the epipharynx.

(*c*) Subjective sense of odor.

(*d*) Strabismus of the right eye.

(*e*) Transillumination shows opacity of the right lower eyelid (left negative) and absence of red pupillary reflex, also opacity over the right frontal sinus.

(*f*) The bulla ethmoidalis is enlarged downward and inward, and there are polypi in the middle meatus.

Provisional diagnosis of empyema of Series I and II is made. It is still a question as to the exact localization of the suppuration. It seems probable that all the sinuses in Series I and II are involved, although this is not yet proved.

(*g*) The blunt probe is used, and shows bare rough bone in the superior meatus and in the region of the uncinate process (the inner and inferior lip of the hiatus semilunaris). This makes it quite probable that the posterior ethmoidal, anterior ethmoidal, and the antrum are involved.

When the bulla ethmoidalis is enlarged downward the discharge of pus is blocked in the infundibulum and is pent up in the anterior ethmoidal and the frontal sinuses. The pus under these circumstances often breaks through the lateral wall of the nose into the antrum. The enlargement of the bulla (one of the anterior ethmoidal cells) is in itself significant of a diseased process in this group of cells.

(*h*) The anterior end of the middle turbinal and the polypi in the middle meatus are removed.

(*i*) The maxillary sinus is irrigated through a puncture in the inferior meatus and much pus removed, but it continues to discharge.

(*j*) The frontal sinus is irrigated through a cannula and a copious discharge of pus follows and persists.

(*k*) The bulla is broken down with a curette, and pus wells from its interior. A polypus also protrudes from its cavity. The remainder of the middle turbinate is resected and the posterior ethmoidal cells are thoroughly removed by curettement. After a time the discharge of pus ceases.

Having demonstrated the persistent presence of pus in all the sinuses embraced in Series I and II a positive diagnosis may be made.

Diagnosis.—Combined empyema of all the accessory nasal sinuses of one side of the head. A radical external operation and intranasal operations may or may not be indicated. All the sinuses may be drained by operative procedures through the nose and a cure effected without external operations in many cases.

Note.—While the foregoing series of hypothetical cases does not exhaust the list of possible and actual combinations of empyema of the accessory nasal sinuses, it illustrates fairly well the data and methods of procedure necessary to arrive at a diagnosis. Nor should it be understood that the data used in the above series is in strict accord with the clinical aspect of every case having the diagnosis given above. Other symptoms and pathological conditions are found, and great anatomical asymmetry often complicates the diagnosis. What is given above is in the main true. Much that is left unsaid is also true. It is obvious that in a limited number of hypothetical cases all the clinical and pathological data cannot be given.

CHAPTER X

GENERAL CONSIDERATIONS IN REFERENCE TO THE SINUSES

The nasal accessory sinuses in man are the residual olfactory organs. In his primeval state the acute sense of smell was necessary, as it is in some lower animals. In the process of evolution the large distribution of the olfactory nerve has become less and less necessary, hence the sinuses are being gradually closed off from the nasal chambers until only small openings are present in man. Inflammation of the lining mucous membrane of the walled-off spaces becomes, therefore, a frequent pathological process. If the sinuses were open more to ventilation and drainage, inflammatory processes within them would occur less frequently, because the perpetuity and destructiveness of the process depend very largely upon the lack of normal ventilation and drainage. It follows, therefore, that when inflammation of the sinuses is present the first principle of treatment is to establish ventilation and drainage. This may only mean that the swollen and inflamed mucous membrane around the cell openings should be depleted by the application of adrenalin, cocaine, or antipyrine, or it may mean that some surgical procedure should be instituted for their relief. Whichever may be necessary, ventilation and drainage of the sinuses is of prime importance, and the removal of the morbid material is secondary to this.

Etiology.—The etiology of the inflammatory diseases of the nasal accessory sinuses of the nose, like that in other mucous-lined cavities of the body, is largely embraced in those conditions which interfere with the drainage and ventilation of the cavities. (See Etiology of Inflammations of the Nose and Accessory Sinuses, Chapter VI.) When there is good drainage and ventilation, inflammation is rare, except in those cases subjected to a virulent infection or in which the resistance is lowered by some dyscrasia. The local expression of a constitutional dyscrasia, as syphilis, tuberculosis, etc., or a carious process in some contiguous organ, as a tooth, may cause inflammation of a sinus, even though the drainage and ventilation of the cells is normal. Aside from these and other local and constitutional diseases which cause sinuitis, it may be said that the anatomical configuration of the interior of the nose, whereby the drainage of the secretions and the ventilation of the sinuses are interfered with, plays an important role in the etiology of inflammation of the sinuses.

The constitutional diseases having most to do with the causation of sinuitis are syphilis and tuberculosis. When there is a granulomatous infiltration in the outer wall of the nose, the ulcerative process may invade the sinuses and give rise to inflammatory symptoms, as pain,

tenderness, suppuration, headache, dizziness, etc. Likewise, when tuberculous infiltration and subsequent degeneration are focalized in the outer wall of the nose, the sinuses may participate in the process, or the ostia of the sinuses may become closed from swelling of the mucous membrane, and thereby obstruct the drainage and ventilation.

Diseases of the contiguous anatomical structures, as the teeth, hard palate, and outer wall of the nose, may give rise to inflammation of the mucous membrane of the sinuses by an extension of these cavities, and by blocking the cell openings or the infundibulum, so that drainage and ventilation are impaired or altogether lost.

Caries of the root of a tooth located beneath the floor of the maxillary sinus (antrum of Highmore) may cause empyema of the antrum by infection through the carious fistula thus formed, or by way of the vessels and lymphatics. It has been estimated that nearly one-half of all empyemas of the antrum have their origin in diseased teeth, while the remainder are due chiefly to intranasal diseases and anatomical deformities of the nose. Nasal polyp is also regarded as a cause of sinuitis, although I believe the polyp is more often the result than the cause. However this may be, it is certain that the presence of a nasal polyp aggravates an existing sinuitis, and that its removal is often attended by an apparent rather than a real cure of the inflammation.

Foreign bodies in the nasal passages may cause sinuitis by erosion and subsequent infection of the nasal mucosa, by directly blocking the cell openings, or by erosion through the outer nasal wall into the sinuses.

Nasal operations may result in sinuitis by reactionary infection and inflammation, which may extend directly through the outer nasal wall or *via* the cell openings into the sinuses. In hospital practice particularly, infection from other patients may give rise to sinuitis.

Nasal dressings may cause a damming up of the secretions which undergo decomposition and infection, and thus give rise to inflammation of the sinuses. Too much emphasis cannot be laid upon the untoward results of intranasal tamponing, as it is a fruitful source of inflammatory disease of the nasal and sinus mucous membranes. Personally, I have abandoned intranasal dressings except in those cases in which there is severe hemorrhage, and in which a dressing must be introduced to hold the septum in position after certain operations for the correction of deviations. Even then they should not be left in position an hour longer than is absolutely necessary to accomplish their purpose.

Venous stasis from intranasal pressure may cause sinuitis. The pressure may be due to some anatomical or pathological departure from the normal, as a deviation of the septum pressing against the outer wall of the nose, or to gummatous swelling of the septum.

These and other pathological lesions of the adjacent structures may cause sinuitis. All cases should, therefore, be carefully studied in order to determine the predisposing cause of the inflammation.

The Exciting Causes. The exciting causes of inflammation of the sinuses are the various microörganisms causing the exanthematous and

other infectious fevers. It is well known that coryza is often one of the early phenomena in this class of cases, and that it is due to micro-organisms and their toxins. The inflammation usually extends to the sinuses, where it may remain in a latent or chronic form. In some cases it is only after many years that the involvement of the sinuses becomes obvious enough to attract the attention of either the patient or the physician.

It is probably true that the inflammation thus started is more likely to become chronic in those cases in which the cell openings are more or less blocked by anatomical deviations of the septum or other obstructive lesions of the nose. If, for example, the septum in its upper portion is deviated to one side, and lies against the middle turbinate, the sinuitis which develops during an attack of one of the infectious fevers is more likely to continue into the chronic form than it is where no such obstructive deformity of the septum exists.

Hajek has emphasized the causative relation of influenza to inflammation of the sinuses. Indeed, he claims that it is probably the most frequent source of infection.

Pathology.—The pathological changes which occur in the mucous membrane and bony walls of the sinuses in the course of suppurative inflammation are what might be expected in a mucous-lined cavity. Much discussion has arisen on this subject between anatomists and clinicians. Anatomists have found less marked changes, probably because they only examined such cases as came to them from the dead-house, while clinicians describe much more extensive changes in living cases, from whom specimens were removed during life, or upon the postmortem table. I prefer to base the pathology upon the clinical rather than upon the anatomical data.

Acute inflammation of the sinuses may be divided into the exudative and the diphtheritic, although the latter is rarely present and is not a true diphtheritic membrane.

The *exudative* inflammation may be serous, fibrinous, seropurulent or purulent in character, according to the intensity of the inflammatory process.

For didactic purposes the changes which occur in the tissues may be studied in the following order, which represents the usual sequence of the pathological events:

(*a*) The submucous tissue is infiltrated with serum, while the surface is dry. Leukocytes also fill the meshes of the submucous tissue.

(*b*) The capillaries are dilated, and the mucous membrane is red in consequence.

(*c*) After a few hours, or a day or two, the serum and leukocytes escape through the epithelial covering of the mucosa, where they become admixed with bacteria, epithelial debris, and mucus. In some instances capillary hemorrhage occurs and blood becomes mixed with the secretions. The secretions, at first thin and watery, later become thicker and tenacious, on account of the coagulation of the fibrin of the serum.

(*d*) In many cases resolution by the absorption of the exudate and the cessation of the discharge of the leukocytes takes place in from ten to fourteen days.

(*e*) In other cases, however, the inflammation passes from the catarrhal to the purulent type, the leukocytes being thrown out in immense numbers. Resolution is still possible, although not probable, as the tissue changes are not yet of a fixed type. Unless the process is speedily arrested the tissue changes become permanent and chronicity is established.

(*f*) If the ostia of the sinuses are open the discharge of pus may continue indefinitely with little or no pain. If, on the contrary, they are closed, the purulent secretion is retained, and pressure symptoms, as pain, swelling, and tenderness, arise. If the discharge cannot escape through the ostia the point of least resistance bulges before the pressure of confined pus. The points of least resistance vary in different cases, although there is reasonable constancy in their location.

The points of least resistance in the sinuses are as follows, due allowance being made for anatomical variations:

(*a*) In the *frontal sinus* the inferior wall is the thinnest, especially three-quarters of an inch from the median line over the anterior ethmoidal cells, hence the frequent involvement of these cells in frontal empyema. Clinically, we often see cases in which there is a sudden gush of pus into the nasal chamber, after which the pain and other pressure symptoms are relieved. It is probable that in these cases the floor of the frontal sinus yielded to the pressure of the pent-up pus, which may have discharged through the anterior ethmoidal cells, though it may have escaped through the frontonasal canal.

(*b*) In the *antrum* the most vulnerable point in the nasal walls is the pars membranaceæ, the membranous portion of the middle meatus. The anterior and superior walls are sometimes thin, and may bulge, or become perforated by the pressure of the retained pus. One of the characteristic symptoms of antral empyema is the tenderness and swelling over the anterior (canine fossa) wall. Bulging of the upper or orbital wall causes an interference with the external muscular apparatus of the eyeball. Perforation in the orbital wall, or roof of the antrum, gives rise to an abscess of the orbit, or orbital cellulitis.

(*c*) In the *ethmoidal sinuses* the point of least resistance is, perhaps, difficult to define, on account of the complexity of the ethmoidal labyrinth, it being composed of several pneumatic spaces. The lamina papyraceæ (paper plate) separating the cells from the orbital cavity is quite thin, as its name implies, and may be the seat of bulging and perforation. The pressure may extend toward the orbit and give rise to a lack of balance of the external muscles of the eyeball, strabismus being the most common expression. The inner or nasal aspect of the ethmoidal cells is more thin, and in empyema may be distended until it presses against the septum.

(*d*) In the *sphenoidal sinus* the point of least resistance is in the upper wall, or roof, which is in close relationship to the optic nerve; hence, the ocular disturbances often found in closed empyema of this sinus.

In chronic inflammation by far the greater number of observations have been made on the antrum, because it is more accessible to inspection and operation through the canine fossa. There is no particular reason, however, why similar changes may not occur in the other sinuses. I will therefore describe in general the pathological changes which occur in the entire sinus labyrinth, pointing out the changes peculiar to each group of cells, in addition to the changes common to them all. In general, it may be said that the pathological changes in the accessory sinuses of the nose correspond with the descriptions in general pathology.

The slighter changes are quite like those in acute suppurative inflammation affecting other mucous membranes and bone tissue. The mucous membrane may present a granular surface, villous and fungoid excrescences, granular, cushion-like thickening, etc. In the older cases there is thickening from deposit of hyperplastic and pyogenic membrane The membrane may be destroyed in spots by ulceration, exposing smooth, bare bone, or it may be soft or rough from caries. In some cases necrosis and bone sequestra are present, or they may be absorbed. A microscopic examination of the secretions of the mucous membrane sometimes shows a loss of the epithelium and glands, which are replaced by connective tissue. Ulcerations of the membrane are often surrounded by granulation tissue, especially if there is necrosis of the bone. Granulation buds may encroach upon the periosteum and thus unite the bone and mucous membrane. When this happens the bone is superficially absorbed and somewhat roughened in consequence. Osteophytes, or bony scales or plaques, resulting from plastic exudation sometimes form on the surface of the bone.

Polypi have been found in all the sinuses, although they are more common in the antrum and ethmoidal cells. They are much more common in the ethmoidal cells than is generally supposed. Their hidden location within the small ethmoidal spaces renders their diagnosis rather difficult. In the antrum, however, they are more easily diagnosticated, as they may be exposed through the canine fossa. As this sinus is quite large, the polypi are easily seen and diagnosticated. They have been found in the frontal and sphenoidal sinuses, although not so frequently as in the antrum and ethmoidal cells. The polypi in the ethmoidal cells are usually quite small, on account of the limited space within the cells, whereas in the antrum they are much larger. In empyema of the ethmoidal cells the thin lamina papyraceæ separating the cells from the orbital cavity may be perforated or entirely destroyed by the suppurative process. The same is true of the cranial plate separating the cells from the anterior hemisphere of the brain. In the latter case the meninges are exposed to infection, and may be the seat of meningitis, brain abscess, or epidural abscess. Such an exposure of the meninges may exist in cases of latent ethmoidal empyema, with no other symptoms than a slight headache and mental irritability. A slight intranasal operation, especially on the middle turbinated body, may light up the slumbering fires and rapidly lead to a dangerous, or even a fatal, meningitis. The cases of meningitis occurring after intra-

nasal operations are probably to be explained in this way, as has been shown by Grünwald in his work on *Nasal Suppuration.*

Thrombosis of the longitudinal and cavernous sinuses occasionally complicates ethmoidal empyema. Retrobulbar suppuration, or orbital cellulitis, is a comparatively infrequent complication of ethmoidal empyema.

In frontal empyema the floor and posterior wall are most often the seat of destructive changes. The floor near the median line is in apposition with the anterior ethmoidal cells and nasal septum, hence the cells and septum are frequently more or less involved in the carious and necrotic retrograde changes. The anterior ethmoidal cells are always filled with pus in cases of frontal empyema.

Symptomatology.—The Objective Symptoms.—The objective symptoms may be extranasal or intranasal.

The extranasal symptoms are those changes in the appearance of the skin of the face and of the fundus of the eye as shown by ophthalmoscopic examination. In addition to the objective signs, the results of transillumination and of skiagraphy afford important objective information.

The intranasal objective signs of disease of the sinuses are those changes in the appearance of the outer walls of the nasal chambers and the location of the secretion as it drains from the affected cells.

Extranasal Objective Symptoms.—(*a*) When any of the sinuses contiguous to the skin of the face are involved (frontal, anterior ethmoidal, or antrum) there may be redness, swelling, and heat of the skin covering the affected area. If, for instance, the frontal sinus is acutely inflamed there may be swelling, redness, and heat of the skin in the frontal region; likewise in the malar region in antral disease and at the inner angle of the orbit in anterior ethmoidal disease (Fig. 125). Tenderness upon pressure (a subjective symptom) is also present when redness and swelling are found.

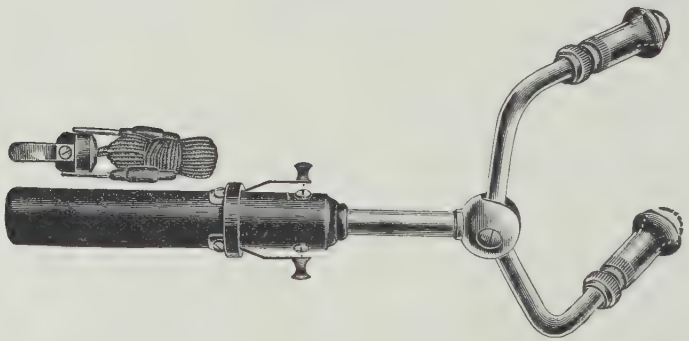
(*b*) The fundus of the eye sometimes affords very useful and important objective evidence of inflammation.

(*c*) Transillumination of the face affords objective information as to the condition of the maxillary sinus, and sometimes of the frontal sinus, but none in reference to the other sinuses. In transillumination of the antrum three points should be noted, namely: (1) The red pupillary reflex, (2) the crescent of light corresponding to the position of the lower eyelid, and (3) the sense of light in the eye when closed. If the red pupillary reflex and the crescent of light are absent the antrum is probably affected. Note both sides at once, and thus determine which one, if either, is affected. A comparison of the lower portion of the field of illumination may be very misleading, as the anterior wall of the antrum varies greatly in density, irrespective of the disease present. The orbital or upper wall of the antrum is, however, more nearly uniform in its density in all cases, and affords a fair opportunity for a comparison of the transilluminated light through the two orbital plates; that is, when both orbital plates of the antrum are healthy the

amount of light transmitted through them is about equal; whereas when one is thickened by an inflammatory exudate the transmission of light is interfered with, hence the crescent of light is dimmed or altogether absent. Likewise when both orbital plates are healthy (antral disease absent) the light transmitted into the interior of the eyeball is shown in the red pupillary reflex in each eye; whereas if one antrum is involved the pupillary reflex is absent upon that side and present on the other. The sense of light (eyes closed) is present on the healthy side and absent upon the diseased side in maxillary diseases.

Transillumination of the frontal sinuses is an uncertain means of diagnosis, as the anterior wall often varies so much in thickness on the two sides in the same individual. The hooded lamp should be placed under the floor of the frontal sinus at the upper and inner angle of the orbit and the two sides compared. Dr. Birkett has devised a double lamp (Fig. 131), so that both sides can be illuminated at once, to facilitate

FIG. 131



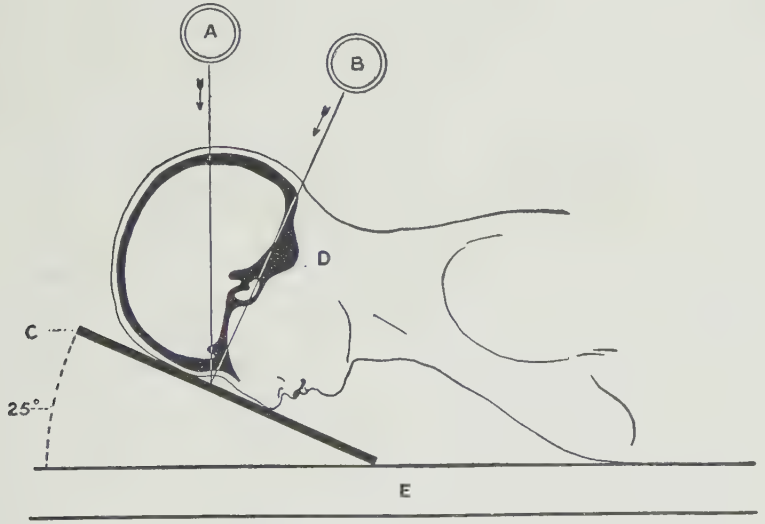
Birkett's transilluminator for the simultaneous illumination of both frontal sinuses.

comparison. If the lamp is not placed well under the supra-orbital ridge the skin transmits the light and may thus lead to a false deduction. Taken as a whole, transillumination of the frontal sinuses is not a reliable procedure.

Skiagraphy.—Skiagraphy of the accessory sinuses of the nose should be a routine practice when access is had to a competent radiographer. Prof. Gustav Killian first practised it in diseases of the nasal accessory sinuses. Personally, I have skiagraphs made of all my private sinus cases and find them of the greatest diagnostic aid, especially in determining the extent of the suppurative involvement. During the past year I have had the plates made with first one orbit on the negative, and then the other orbit thus placed. These oblique positions give perspective views of all the sinuses from the frontal to the sphenoidal, and enable the attending surgeon to locate the purulent inflammation in one or more of the cells. The great difficulty has been to find a radiographer who understands the technique well enough to produce clear skiagraphic plates.

To get a plate with clearly defined outlines of the sinuses, and with a clear definition of their area, it is necessary so to place the *x*-ray tube as to avoid the heavy bone of the floor of the cranium, as it would interfere with the passage of the rays through the head. The *x*-ray tube should be applied, therefore, to the back of the head at a point above the occiput and floor of the cranium, as shown by the line *A* in Fig. 132. If the tube is applied at *B*, the rays would have to pierce through the dense bone of the occiput and the long axis of the plate of bone forming the floor of the cranium before reaching the frontal and ethmoidal sinuses, thereby interfering with the formation of a clear shadow of the dense bone forming the walls of the sinuses and the production of a clear definition of the area of the sinus cavities. If, however, the *x*-ray tube is applied at *A*, midway between the occiput and the vertex, the rays have an unimpeded course of the frontal and ethmoidal sinuses, and the outline and area of normal sinuses will be clear and well modulated. The delineation of the

FIG. 132



Schema showing the proper position for making a skiagraph of the frontal and ethmoidal sinuses: *A*, the proper angle for passing the *x*-rays through the head; *B*, the improper angle, as the rays must pass through a great deal of dense bone (*D*) to reach the sinus; *C*, an 8 x 10 inch photographic plate against which the forehead should rest; *E*, the table upon which the patient lies. The forehead should be placed upon a triangular block with an inclination of 25 degrees, as this is more comfortable to the patient and renders the line (*A*) perpendicular to the table.

maxillary sinuses is not so clear, as the rays must pass through more bone tissue to reach it. A clear skiagraph of this sinus is not so essential, as this sinus is easily and successfully examined by transillumination with an electric lamp in the mouth.

The advantages derived from skiagraphy of the accessory sinuses in diagnosis are:

(*a*) If a sinus is healthy, its outline on the plate or negative is clear and distinct (light) and its area is clear and dark. If the sinus is diseased,

its outline is less clear and distinct and its area is cloudy or hazy upon the negative or plate. Prints from the plates are rarely satisfactory for diagnostic purposes.

(*b*) The dimensions of the frontal sinuses are clearly defined, thus affording the surgeon positive information as to the extent of exposure necessary before he begins an external operation. A skiagraph through the lateral dimensions of the head shows the depth of the frontal sinus, thus affording the surgeon additional data as to the probable deformity to be expected should the Killian operation be performed. The wider and deeper the frontal sinus the greater is the deformity following the complete removal of the anterior bony wall of the sinus. The information gained from the two views of the frontal sinus may cause the operator to determine either to select or reject a given method of operating. If, for example, the skiagraph shows a small, shallow frontal sinus, the Killian operation might be chosen in preference to other methods, as it is a thorough and satisfactory method of operating, and would in such a case be followed by little or no external deformity. If, on the other hand, the plates show a large and deep frontal sinus the surgeon might be influenced to adopt some other method of operating which would not be attended by such marked external deformity.

(*c*) In some instances, when the frontal sinus *seems* to be involved, the skiagraph will show a total absence of disease, and sometimes of the sinus, information of no small consequence to both the surgeon and the patient.

Remark.—According to my observations the skiagraph does not differentiate between a catarrhal and a suppurative sinuitis.

The Intranasal Objective Symptoms.—(*a*) The contour of the outer nasal wall sometimes affords information as to the condition of the sinuses. In closed empyema of the antrum the inner wall of the antrum may be pushed toward the septum. Likewise in empyema of the bulla ethmoidalis its median wall may be distended so as to close the hiatus semilunaris, and impinge against the external surface of the middle turbinal.

(*b*) The texture of the mucous membrane of the nose, especially that portion of it covering the middle turbinated body, is sometimes indicative of sinus disease; that is, when the mucosa of the anterior end of the middle turbinate is boggy and velvety in texture, it usually signifies the existence of an inflammation of the ethmoidal cells.

(*c*) Polypi are often associated with disease of the sinuses, and are, I believe, usually secondary to the inflammation.

(*d*) Pus within the nasal chambers is usually significant of empyema of the sinuses. The nasal mucosa is rarely the focal centre of suppurative inflammation, whereas the sinuses are commonly the focal centre of such an inflammation. The presence of pus in the nasal chambers should, therefore, excite suspicion of the existence of an inflammation of the sinuses. (To determine which of the sinuses is involved, see General Diagnosis.) In a general way it may be stated that pus in the middle meatus signifies an involvement of the frontal, anterior ethmoidal, or the maxillary sinus, as these cells drain into the middle meatus. If pus is seen in the olfactory fissure (between the septum and middle turbinate)

the posterior ethmoidal or the sphenoidal cells are involved, as these cells drain into the superior meatus above the middle turbinate.

Subjective Symptoms.—The subjective symptoms of inflammation of the sinuses have reference to the sensations of pain and of pressure, the equilibrium of the mind, and the impairment of the special senses.

(*a*) Pain referable to the region of the sinus involved may or may not be present. In active inflammation of the antral or frontal sinus pain is often distinctly referred to the region involved. In the deeper sinuses, as the ethmoidal and sphenoidal, the pain is vaguely deep seated in the head, or it is referred to the periphery of the head without reference to the location of the sinus. For example, sphenoidal inflammation may give rise to pain in the occipital or to the frontal region. As a matter of fact, inflammation in any or all of the sinuses usually causes pain in the frontal region. These pains are almost universally called headaches by the patient.

(*b*) Headache is, therefore, one of the most common and significant signs of sinuitis, though it may be present when the middle turbinal presses against the septum. This condition is often mistaken for eyestrain. Refraction is rarely satisfactory, and only when the anterior end of the middle turbinate is removed is the headache relieved and glasses accepted. In many cases glasses are not necessary. Headache has multitudinous causes, and is not, therefore, pathognomonic of inflammatory or other diseased conditions of the sinuses. Headache may signify eyestrain, but in this case it is usually bilateral, whereas in sinus disease it is more often unilateral, or, if not unilateral, more pronounced on one side, or it begins as a unilateral headache and extends to the other side. The headache which originates in a sinus is increased upon stooping forward and upon sudden jarring of the body. It may persist upon closing the eyes upon retiring, or in a darkened room; whereas if it is of ocular origin it disappears under such conditions.

The headache of ocular origin is greatly increased upon prolonged reading and upon attendance at the theatre. The headache caused by attendance at the theatre is so characteristic of ocular disturbance that it may be termed "theatre pain." This type of pain is not characteristic of sinus disease.

The pains and headache due to disease of the frontal sinus may assume the form of sharp, shooting pains through the eyes, or they may be dull and heavy, and nearly constant; or they may consist of a dull feeling in the forehead, which is aggravated by leaning forward, and which in females is especially well marked during each menstrual period (H. M. Fish). Pressure under the floor of the sinus at the inner angle of the orbit usually elicits pain in these cases.

(*c*) *Tenderness upon Pressure.*—Tenderness and pain upon finger pressure may be present in disease of those sinuses contiguous to the surface of the face, viz., the frontal, anterior ethmoidal, and the maxillary sinuses.

For the examination of the frontal sinus, pressure should be made over the anterior wall above the supra-orbital ridge, and under the floor of the sinus near the inner angle of the orbit.

In the examination of the anterior ethmoidal cells, pressure should be made at the inner angle of the orbit against the orbital plate of the ethmoid.

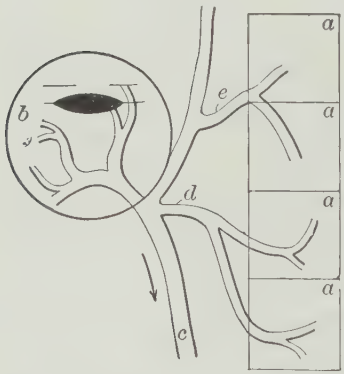
In the examination of the antrum of Highmore, pressure should be made over the canine fossa of the superior maxilla.

(*d*) *Disturbance of Equilibrium.*—Giddiness and vertigo or a momentary sense of blurred or darkened vision and imminent fainting are frequently present in disease of the sinuses. All these symptoms may be aggravated or produced by stooping forward. The patient should be carefully questioned in regard to these symptoms, as otherwise they may be overlooked.

(*e*) *Disturbances of the Special Senses.*—The olfactory, visual, and auditory senses are frequently disturbed or altogether lost in sinuitis.

The olfactory sense may be perverted (parosmia), the patient apparently perceiving odors that are not in evidence to normal noses. A more common symptom is the loss of olfaction (anosmia). This is accounted for by the blocking of the olfactory fissure by the tissues in the region of the middle turbinate. The ventilation of the superior meatus of the nose is thereby prevented, hence the loss of the sense of smell. In some cases this may be due to the degeneration of the terminal filaments of the olfactory nerve, although in most cases coming under my observation the sense of smell is regained after opening the olfactory fissure either by removing the obstructive tissues or by resorting to some surgical procedure, as the removal of polypi, a portion of the middle turbinate, or correcting a deviation of the septum.

Fig. 133



Schema showing the venous connections of the ethmoidal cells with the eyeball: *a*, *a*, *a*, *a*, anterior and posterior ethmoidal cells; *b*, eyeball; *c*, superior ophthalmic vein; *d*, posterior ethmoidal vein; *e*, anterior ethmoidal vein.

The ocular function may be disturbed or altogether lost in the course of sinus disease. The disturbance may be due to either arterial or venous congestion, and to toxins, or to thrombosis of the veins intercommunicating between the sinuses and the eye. The morbid process in the eye may take the form of a papillitis, neuroretinitis, retrobulbar disease, keratitis, errors of refraction or of accommodation, photophobia, epiphora, choroiditis, marginal blepharitis, iridocyclitis, conjunctival injection, restricted field or loss of vision.

Relation of the Eye to Disease of the Sinuses.—The intimate relation between the veins of the nose and accessory sinuses and of the eye (Fig. 133), as demonstrated by Dr. H. M. Fish, Dr. W. C. Posey, and others, shows how reasonable is the assumption that many of the ocular lesions heretofore attributed to auto-intoxication from the intestines,

gonorrhea, syphilis, and rheumatism, may, in many instances, be due to an extension of the disease from the sinuses to the ocular apparatus *via* the veins and lymphatics.

According to Posey, the extra-ocular muscles may become paretic or paralyzed from inflammation of the sinuses, because the nerves which supply the muscles are in close anatomical relationship with the walls of the sinuses and may be paralyzed by pressure or by toxic influences. The levator, superior oblique, and superior rectus muscles are in relationship with the floor of the frontal sinus, and paralysis of them is indicative of disease of the frontal sinus. The internal rectus muscle is in relationship to the inner orbital or ethmoidal wall and paralysis of this muscle is indicative of disease of the ethmoid cells. The inferior oblique and the inferior rectus muscles are in relationship to the superior wall of the antrum (floor of the orbit) and paralysis of either of these muscles is indicative of disease of the antrum. As the nerves which supply all these muscles pass in apposition or close approximation to the sphenoid sinus, disease of this sinus may involve one or more of the muscles, hence, each case must be carefully studied before the location of the inflammation can be determined. Paresis of either of these muscles causes a type of diplopia or squint. Diplopia may also be due to retrobulbar pressure causing displacement of the eyeball.

Optic neuritis or other disease of the eye and adnexa is frequently due to disease of the nasal accessory sinuses, more particularly the ethmoid and sphenoid sinuses. C. R. Holmes reviewed the literature on the subject and found several cases on record. In one case the patient died of cerebral hemorrhage, and at the autopsy it was found that the roof of the sphenoid, including the bone and dura, was destroyed.

Three cases of optic neuritis with partial and complete blindness have come under my observation and operative treatment within the past two years. The first case was referred to me by Dr. J. G. Huizinga with the diagnosis of optic neuritis due to ethmoidal and sphenoidal disease. His diagnosis was confirmed by Drs. C. A. Wood and G. F. Suker. The patient was thirty-five years of age and was single; syphilis had been excluded. His vision was $\frac{3}{200}$. The defective vision had been present for four months. I performed an ethmoidal exenteration, and removed the anterior wall of the sphenoidal sinuses upon both sides. The vision rapidly improved to $\frac{20}{40}$, where it has remained two years after the operations.

The second case had been under treatment with electricity, etc., for eighteen months and the vision had gradually declined. At the end of this time the case was referred to me by Dr. J. E. Colburn for operation upon the ethmoidal and sphenoidal sinuses. After the operation vision continued to decline.

The third case was referred to me by Dr. G. F. Suker for operation upon the ethmoidal and sphenoidal sinuses. The patient was forty-two years old; syphilis was excluded. He was totally blind, not being able to see a lighted match. The blindness had been present for two weeks. I operated upon the right ethmoidal and sphenoidal sinuses at

once and the vision began to improve. Ten days later I operated upon the left side. The vision receded for two or three days and then began to improve rapidly, until at the end of six weeks it was normal.

The *auditory functions* may be more or less disturbed by disease of a sinus. The discharge from the sinuses into the epipharynx may cause infection of the mucous membrane of the Eustachian tube and middle ear. Sinuitis may indirectly be the cause of catarrh of the middle ear or of suppurative otitis media and mastoiditis. In addition to the foregoing aural complications, there is another symptom which I have not seen mentioned in the literature, namely, a momentary roaring accompanied by a fulness in the ears and dulness of hearing. These phenomena are especially likely to occur on bending forward.

The Principles of Treatment.—The cure of inflammation of a sinus depends upon three things, namely: (*a*) The establishment of free drainage and ventilation, (*b*) the removal of the morbid material, and (*c*) the elevation of the opsonic index by the administration of autovaccines.

In those cases in which the interference with drainage and ventilation is due to a simple hyperemia of the mucous membrane the local application of cocaine, antipyrine, or adrenalin may be quite sufficient to establish a cure. In such subjects the morbid material is the secretion, hence drainage removes it. On the other hand, in those cases in which there is a marked obstruction due to a deviation of the septum or to hyperplasia or cystic enlargement of the middle turbinate, it is often necessary to resort to surgical measures in order to give relief. Furthermore, in those cases in which the sinus is filled with granulation tissue and the bony walls are necrosed the establishment of drainage even by surgical means may not effect a cure; the morbid material (granulations and necrotic bone) must also be removed.

The Indications.—An appreciation of these fundamental principles enables the surgeon to decide upon the method of treatment in each case. In the following discussion of the treatment the foregoing principles will be constantly referred to, with a view to enabling the student and practitioner to elect the proper mode of treatment in the cases coming under his observation. Before entering upon a detailed description of the various modes of treatment a general discussion of the varying conditions to be met will be given.

Acute catarrhal sinuitis is usually an extension of a similar inflammation of the nasal mucosa to the sinus, in the course of a coryza or cold in the head. The mucous membrane of the nose and sinuses is hyperemic and swollen. The ostia and the infundibulum may be closed from swelling of the mucous membrane. The obvious indication is to relieve the swelling by the local application of certain drugs; surgical intervention is rarely necessary.

Acute suppurative sinuitis occurring in the course of coryza is characterized by hyperemia and swelling of the mucous membrane of the nose and sinuses, and the indications are to reduce the swelling by local medicinal applications, as in the acute catarrhal variety.

Chronic catarrhal sinuitis due to pressure in the middle turbinate

region necessitates the removal of the tissue which causes the pressure. If the mucous membrane is chronically swollen, temporary relief may follow the application of antiphlogistic drugs, as adrenalin. If the secretions have dried and blocked the cell openings, probing may afford temporary relief. In most cases the middle turbinate is enlarged from hyperplasia (see Hyperplastic Rhinitis, Polyp), or from bullous enlargement which blocks the infundibulum. In some cases, therefore, it is necessary either to straighten the septum or remove a portion of the middle turbinate in order to give permanent relief. The bulla ethmoidalis may also block the infundibulum and prevent drainage and ventilation of the sinuses in Series I.

Chronic suppurative sinuitis, with obstructive lesions, necessitates their removal, whether they be of septal, turbinal, or other origin. In this case there is simple obstruction, and no morbid material other than pus is present; hence the removal of the obstructive lesion permits of drainage which removes the pus. The foregoing statement does not apply, however, to all cases, as the drainage of pus from the cells is not altogether dependent upon free cell openings, because in most of the cells the opening is near the upper limit. The ciliated columnar epithelium which lines the cells, though limited in distribution, carries the secretions up to the cell openings, where it is discharged into the nasal cavity. If, therefore, the ciliæ are destroyed by the inflammatory process, the removal of the obstructive lesions does not necessarily establish free drainage. In such cases it may be necessary to institute operative procedures in order to open the cells at their most dependent portion, or to exenterate them in their entirety (ethmoidal). In some cases the mucous membrane and the ciliated epithelium can be restored to their normal integrity and functional activity by lavage, or by negative air pressure, as recommended by Bier.

Chronic sinuitis, without obstructive lesions of the septum or the middle turbinated body, implies a hyperplasia of the mucous membrane with a loss of the columnar ciliated epithelium of the sinuses, at least in certain areas. These cases, according to Uffenorde and Skelleren, are not attended by suppuration. My personal observations do not confirm their view, as I have often opened the frontal sinuses and have found both hyperplasia of the mucous membrane (polypi) and pus. I have, however, more often found only polypi present. I do not understand that hyperplastic tissue is immune to pus producing microorganisms, but, on the contrary, I can conceive on both theoretical and clinical grounds that purulent secretions may and do accompany hyperplastic rhinitis and sinuitis. The treatment should therefore either be directed to the regeneration of the mucous membrane by negative pressure, and the resultant hyperemia and increased nutrition, or by opening the cells and establishing free drainage by some operative procedure.

Chronic suppurative sinuitis, with granulations, polypi, or necrosis of the bone, is only amenable to surgical treatment. No treatment other than this will establish drainage and ventilation and remove the morbid material.

Treatment.—The principles of treatment having been given, only the technique will be described in this section.

Treatment of Acute Catarrhal Sinuitis.—Acute catarrhal sinuitis usually involves all the accessory sinuses, and the indications call for the reduction of the swelling of the mucous membrane for the purpose of opening the ostia of the sinuses. The following technique is usually successful:

(*a*) Apply adrenalin, 1 to 2000, on thin pledgets of cotton, to the swollen middle and inferior turbinates to reduce the swelling.

(*b*) Apply a 4 per cent. solution of cocaine to reduce the swelling and to relieve the hypersensitiveness of the mucous membrane.

(*c*) Apply a 10 per cent. solution of antipyrine over the same area to prolong the ischemic effects of the adrenalin and cocaine.

(*d*) Use a 0.5 per cent. solution of menthol or other bland aromatic oily solution with a nebulizer every two or three hours.

The solutions of adrenalin, cocaine, and antipyrine should be used as often as the nasal chambers feel "stuffy," or the headache and sense of pressure return.

In addition to the foregoing local remedies, those which are usually given in acute coryza may be administered, but they are of value only in the early stage. (See Treatment of Coryza.)

Heat from a 500 candle-power lamp applied over the face sometimes affords speedy relief. The lamp should be passed back and forth before the closed eyes, at a distance of from twelve to eighteen inches for twenty to thirty minutes. The good effects are due to the increased hyperemia and leukocytosis, and to the improvement of the nutrition. While germicidal properties are claimed for the light of this lamp, the effects are probably due to the increased leukocytosis and nutrition of the tissues. I have thus treated chronic cases in which the purulent discharge and pain ceased, but returned after a few weeks. Whether persistent use of the light will cure these cases I am not prepared to state.

Treatment of Chronic Catarrhal Sinuitis.—This is a more difficult type to treat successfully on account of its chronicity, which of itself may imply that anatomical barriers existed during the acute stage which prevented resolution. These barriers, if present, must be overcome before a cure can be permanently established. The anatomical barriers to resolution may consist of hypertrophic or hyperplastic changes in the mucous membrane of the nose, especially in the region of the cell openings and the olfactory fissure, or they may be due to ethmoidal cells in the middle turbinate or to deviations of the upper portion of the nasal septum.

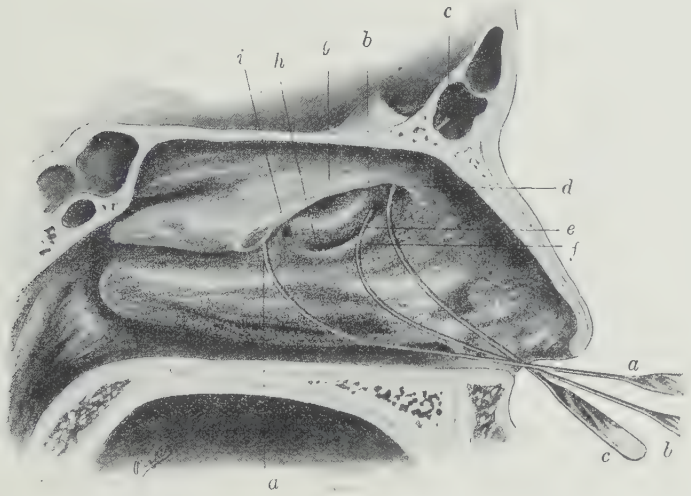
The swelling of the mucosa may be somewhat reduced by the local applications of adrenalin, cocaine, and antipyrine. In addition to this the hypertrophic or hyperplastic rhinitis should be surgically treated after the manner described under these diseases.

If these measures fail, more radical surgical procedures, such as are used in obstinate cases of suppurative sinuitis, may become necessary. Probing of the frontonasal canal sometimes affords relief, although the

removal of the anterior end of the middle turbinate and the curettement of the ethmoidal cells may be necessary.

Treatment of Chronic Suppurative Sinuitis.—In the simpler form of sinuitis, that is, when there are no granulations nor carious bone, the lavage of the affected sinus with antiseptic, alkaline, or stimulating solutions is sometimes followed by a cure. The lavage of the frontal sinus may be performed through the frontonasal canal, except in those cases in which it is absolutely closed by an enlarged bulla or by an enlarged middle turbinated body.

FIG. 134



Probing the frontal sinus. The anterior half of the middle turbinated body is removed to show the anatomical landmarks: *a*, *a*, the probe in the first position beneath the middle turbinate and posterior to the bulla ethmoidalis; *b*, the probe in the second position beneath the middle turbinate and in front of the bulla ethmoidalis; *c*, *c*, the probe in the third position introduced through the frontonasal canal into the frontal sinus; *d*, the nasal end of the frontonasal canal; *e*, the lip of the uncinate process; *f*, the inner wall (uncinate process) of the infundibulum; *g*, the ostium bulla ethmoidalis; *h*, the ostium maxillare; *i*, an accessory opening into the maxillary sinus. (Drawing from a specimen loaned by Dr. Ira Frank.)

Lavage of the Frontal Sinus.—An understanding of certain anatomical peculiarities of the region of the infundibulum and the frontonasal canal will materially aid in the lavage of the sinuses. The hiatus semilunaris, the infundibulum, and the frontonasal canal will be clearly defined, as much confusion appears in the literature concerning them. The terms are often used as synonymous, whereas they are distinct anatomical entities.

The hiatus semilunaris is a slit-like crescentic-shaped opening in the outer wall of the nose. It is the opening of the infundibulum into the middle meatus. Its inner lip is the upper margin of the uncinate process of the ethmoid bone.

The infundibulum is a deep, narrow groove or gutter in the outer wall of the nose (Fig. 134, *f*), the inner wall of which is the uncinate process. The frontonasal canal drains into the infundibulum in about one-half

of the subjects, whereas in the remainder it drains a little anterior to it directly into the middle meatus (Turner).

The frontonasal canal is a closed tubular duct extending upward and forward from the middle meatus or the infundibulum, as the case may be, to the frontal sinus. Its opening into the floor of the frontal sinus is known as the ostium frontale. In rare instances the ostium opens high upon the posterior wall of the sinus.

Having defined the parts concerned in probing or irrigating the frontal sinus, certain anatomical peculiarities which influence the procedure will be given brief notice.

The hiatus semilunaris is the key to the probing, as it is the opening into the infundibulum, which must be entered to reach the frontonasal canal in about one-half of the cases. The bulla ethmoidalis is situated just above the hiatus, and when large it encroaches upon the slit-like opening and partially or completely closes it. Occasionally there are accessory cells in the uncinate process, which also obstruct the hiatus. In other cases the middle turbinate closely hugs the outer wall of the nose and blocks the hiatus (Sluder). When either of these anatomical peculiarities is present the introduction of the probe or the cannula is rendered difficult or impossible. If the frontonasal canal opens in front of the infundibulum the probe or cannula may be passed into it even though the hiatus is closed.

FIG. 135

Holmes' malleable frontal sinus probe.

Another difficulty sometimes encountered in probing is, that the probe may enter the ostium of one of the anterior ethmoidal cells instead of the frontal sinus. Some of the anterior cells may open into the infundibulum on its outer wall, while others open into the frontonasal canal. The anterior cells are usually located external to the infundibulum and the frontonasal canal, and their ostia open into the infundibulum and frontonasal canal, through the outer wall. In probing, therefore, the point of the probe should be kept against the inner or mesial wall of the frontonasal canal in order to avoid the ostia on its outer wall.

Probing is generally more difficult in those subjects in which the frontonasal canal empties into the infundibulum than when it empties directly into the middle meatus. In the former case the canal is often tortuous and narrow, while in the latter it is usually straighter and of larger caliber.

The middle turbinate is sometimes so close to the hiatus, especially when the turbinate contains an accessory cell, that it is difficult to enter it with a probe or cannula. In this event the removal of the anterior third of the middle turbinate overcomes the difficulty.

Technique of Probing the Frontonasal Canal.—First cocainize the parts. Then introduce a fine silver probe (Fig. 135), bent at its distal end to an angle of about 135 degrees, between the anterior third of the middle turbinate and the outer wall of the nose. Keep the tip of the probe against the outer surface of the turbinate and pass it forward and upward through the hiatus into the infundibulum, where it readily enters the frontonasal canal even to the ostium frontale (Fig. 134). After engaging in the middle meatus it should be passed into the infundibulum and canal for about 6 to 8 cm. to reach the frontal sinus.

Irrigation of the frontal sinus is accomplished through a silver cannula, which is introduced in the same manner as described for the introduction of the probe. The syringe is attached to the cannula, and the sinus gently irrigated with warm normal salt or boric acid solution.

Lavage of the Maxillary Sinus.—This can rarely be effected through the cell opening on account of its hidden position in the infundibulum, and on account of its forward and downward direction from the infundibulum to the antrum. The opening into the antrum is not directly through the lateral wall of the nose, but it is more like a canal extending obliquely downward and forward through the thickness of the wall. The canal or opening is furthermore somewhat hidden by the unciform process, or lip, of the hiatus semilunaris. Some writers have claimed that they could irrigate the antrum through its normal opening, but a casual study of the anatomical peculiarities of the region will convince anyone that it is a physical impossibility, except in rare instances. In a certain number of cases there are accessory openings into the antrum (Fig. 134, *i*), which when present may be utilized for purposes of irrigation. Then, too, the lamina membranacea of the naso-antral wall may be perforated with the tip of the cannula and irrigation performed through it. In view of the foregoing facts it is rarely possible to irrigate the antrum through the normal ostium, hence an artificial route should be chosen, the most available one being beneath the inferior turbinated body, a curved trocar and cannula being used for the purpose. The technique is as follows:

(*a*) Anesthetize the mucous membrane of the inferior meatus with a 5 per cent. solution of cocaine.

(*b*) Introduce the trocar and cannula beneath the inferior turbinate posterior to the anterior antral wall, and direct it upward and outward, a little above the floor of the nose, in order to avoid the thick wall of bone at this point. In some cases, especially when a maxillary cyst is present, the floor of the antrum is quite high and it is not possible to introduce the trocar beneath the inferior turbinate.

(*c*) After penetrating the naso-antral wall, remove the trocar, leaving the cannula in position.

(*d*) Attach the rubber hose of the syringe to the cannula and irrigate with normal salt or other solution chosen for the purpose.

(*e*) By cocainizing the area daily, the irrigations may be continued indefinitely through the artificial opening.

Lavage of the Antrum through the Alveolar Process.—This may be done after having performed the Cooper operation, so named after Sir Astley Cooper, who introduced it to the profession.

The technique is as follows:

(*a*) Select a place where a tooth has been extracted below the antrum, or if a tooth is decayed beyond repair, extract it for the purpose, and drill a canal into the floor of the sinus. This is Cooper's operation.

(*b*) Through this opening a cannula is introduced and the antrum irrigated with normal salt or any solution desired.

(*c*) The canal thus made should be kept open by means of a hard or soft rubber or gold tube made for the purpose. The tube should be flanged on the lower end to prevent it slipping upward into the antrum.

(*d*) A plug should be introduced into the tube to prevent the entrance of food into the antrum. This method is obsolete.

Lavage through a Canal External to the Teeth.

(*a*) Cocainize the gums.

(*b*) Drill a canal through the upper and external part of the alveolar process at a point between the first and second biscuspids, avoiding the roots of the teeth. This method is practically obsolete.

(*c*) Proceed thereafter as in the Cooper operation.

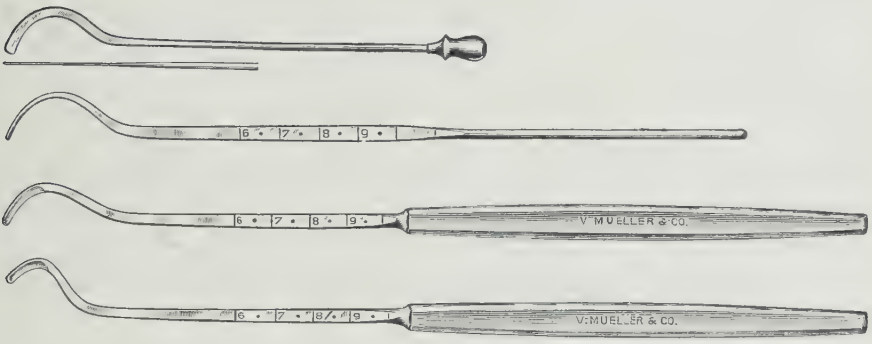
Lavage of the Ethmoidal Cells.—This is often impossible except in the case of the anterior cells which drain into the frontonasal canal. The bulla ethmoidalis, one of the anterior cells, does not drain into the frontonasal canal, but drains directly into the middle meatus, and its ostium is situated at its upper median wall beneath the attachment of the middle turbinated body.

The technique for the lavage of the anterior cells opening into the frontonasal canal is the same as for the frontal sinus, this being introduced into the canal only to the second position (Fig. 134); indeed, both sets of cells are often irrigated at the same time. Their ostia are bathed with the irrigating fluid and the accumulated pus in the canal is removed, thus facilitating the drainage of the cells.

Lavage of the sphenoidal sinus is possible when the middle turbinate, or a deflection of the septum, does not prevent the introduction of the sphenoidal cannula into its opening. When such an obstruction is present it may become necessary to first remove it by some surgical procedure before the irrigations can be practised. I generally use a silver Eustachian catheter in place of a sphenoidal cannula, and find the curve used for the inflation of the ear the correct one for irrigation of the sphenoidal sinus. Myle's cannula may be bent to reach any sinus, and is smaller than the Eustachian catheter. A. H. Andrews has devised a curved cannula (Fig. 136) which can be introduced into the sphenoidal sinus without the preliminary removal of the middle turbinated body. This is a decided advantage, as it renders the treatment of empyema of this sinus a very simple procedure. Should granulations be abundant, it may be necessary first to remove the middle turbinate and then the anterior wall of the sphenoidal sinus, and curette its interior.

The special curve of Andrews' cannula enables the operator to insinuate it through the olfactory fissure into the spheno-ethmoid fossa, and to engage the tip in the ostium sphenoidale by rotating it (Fig. 137). When it has been introduced, the patient should be instructed to lean

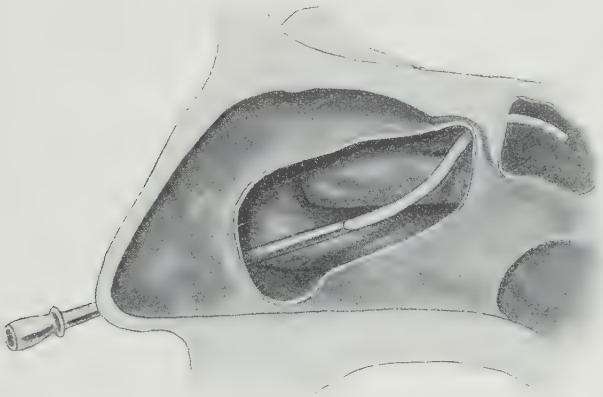
FIG. 136



Andrews' sphenoidal probe cannula and knives.

forward and open his mouth; then the hose of the syringe should be attached to the cannula and the sinus irrigated. If the patient's head is inclined forward and the mouth open the fluid will not enter the Eustachian tube.

FIG. 137



Irrigation of the sphenoidal sinus with Andrews' curved cannula.

General Remarks Concerning Lavage or Irrigation of the Sinuses.— Lavage of the sinuses in suppurative inflammation is, upon the whole, an unsatisfactory therapeutic measure. Formerly it was in vogue with dentists and surgeons for the treatment of antral empyema. Many cases were thus treated daily, for weeks and months, and some were cured, or apparently cured, while others continued to suppurate uninterruptedly.

If lavage is useful at all it is in the simple suppurative cases uncomplicated by granulations and necrosis. The removal of the purulent secretions gives the ciliated epithelium a chance to regenerate. It should also be borne in mind that the mucous membrane does not tolerate lavage indefinitely, as it is not accustomed to the presence of large quantities of aqueous solution, hence irrigation is a doubtful procedure. If after a few days' or weeks' trial the case does not greatly improve, irrigation should be discontinued and some other method of treatment, probably surgical in character, instituted.

Treatment by Negative Air Pressure.—Bier has demonstrated the therapeutic value of this method of treatment in inflammations. Sondermann, Brawley and others have also reported favorably upon the use of negative pressure by means of an exhaust pump. The *rationale* of this meathod of treatment consists chiefly in the increased hyperemia of the mucous membrane lining the cells. The local nutrition is thereby improved, the cell resistance and leukocytosis increased, and the infective process checked. That such changes do take place in some cases thus treated is probably true. It is not claimed that all cases are amenable to this treatment. Let it be understood, therefore, that negative air pressure should be used only as a tentative measure, and if a cure does not follow within a few weeks, it should be abandoned and some other treatment substituted for it.

Technique.—(*a*) The apparatus necessary for producing negative pressure in the sinuses consists of either a hand pump or other device for exhausting the air in the nasal chambers. Brawley's apparatus is operated by attaching it to a faucet of the washbasin, the negative pressure being regulated by the amount of water turned on.

(*b*) Insert the nasal tips into the nostrils and bring the soft palate into apposition with the pharyngeal wall by swallowing. With practice the patient soon learns to maintain this condition for several minutes.

(*c*) While the air is thus exhausted the pus is drawn from the sinus into the rubber tubing, from whence it flows into the reservoir bottle. In this way several drams or ounces of pus may be removed in the course of a half-hour.

(*d*) Daily seances should be maintained until improvement begins, or until the surgeon is convinced that this method of treatment is inadequate for the case.

Drs. Dabney and Pynchon have each devised an exhaust apparatus, having the appearance of a spray tube, which is operated with a compressed-air tank. These are ingenious and practical instruments.

With either apparatus the patient is instructed to swallow, thus closing off the pharynx from the epipharynx and nose. The suction, after a little practice on the part of the patient, maintains the palate muscles in this position for an indefinite period of time. The patient during this process breathes through the mouth.

Vaccine Therapy.—Vaccine therapy in suppuration of the accessory sinuses of the nose, throat, ear, and meninges promises much for the future, but unfortunately it is at present far from universally successful.

In a recent exhaustive review of the literature on this subject by Dr. Reik, both views and results were at great variance. Some writers reported excellent results in one or a series of cases, while others reported negative or occasional apparent success. Some used autogenous vaccines, others stock vaccines. The autogenous vaccines appeared to give better results. Some reported good results without operation, others, only after operation. Some found better results in acute cases, others in chronic cases. One writer, Dr. Nagel, insisted that success depended upon the manner in which the vaccine was prepared, *i. e., the least amount of heat that will kill the bacteria should be used in preparing the vaccine.* She reported *forty suppurative ear* cases treated with autogenous vaccine prepared in this way with thirty-nine cures. All the cases have been under observation one or two years. She did not, however, particularize the cases by giving the full clinical histories. It may be assumed, however, that they were of several types, as they were selected at random from Dr. C. M. Cobb's clinic at Harvard Medical College. Dr. Cobb corroborated Dr. Nagel in every particular, as the work was done under his observation and direction. A report of this character from highly credited authority leads one to hope much good may be gained by vaccine therapy. It also suggests that the great discrepancy in the results obtained by other observers may have been due to the faulty method employed (too great heat, etc.) in the preparation of the vaccine. In the meantime we should continue to use vaccine therapy and insist that the vaccine be prepared with the least degree of heat that will kill the bacteria. This and other improvements in the preparation, and administration of the vaccine, together with the selection of suitable cases, may bring this method of treatment to the degree of efficiency it seems upon theoretical grounds to deserve.

Dr. J. F. McKernon believes that in vaccine therapy we have an aid, first, in wound repair after mastoid operations following scarlet fever and measles; second, in hastening resolution of the accompanying purulent otitis media; and third, in increasing the patient's resistance to the disease by neutralizing the poison in the system, and allowing a more rapid tissue repair. In other words, he believes that healing is hastened and depression and scarring are diminished by autogenous vaccines in mastoid cases following scarlet fever and measles.

Technique.—The preparation of the vaccine is a laboratory process requiring about forty-eight hours for its completion and will not be described. The practitioner should remove some of the purulent secretion from the nose, throat, ear, or other part involved with a cotton-wound applicator, great care being observed to secure it from or as near the original point of infection as possible, and to avoid contact of the swab with other regions which may contain extraneous micrococci which have nothing to do with the infection. The swab thus secured may be sealed in a test-tube or smeared at once on a suitable culture media and delivered to a competent laboratory expert who will prepare a vaccine from it. The laboratory worker should indicate the strength or dosage of the vaccine, which should be injected every three days into the

muscular tissue of the arm, thigh, back, or chest at the discretion of the attending physician.

I have personally used autogenous vaccines in a few cases of extradural abscess of otitic origin with apparent benefit, though in one case without effecting a complete cure. In this case a radical mastoid operation was first performed to relieve the meningeal abscess (following diphtheria). The acute manifestations rapidly subsided, but a purulent secretion, small in amount, continues, after the lapse of five months, to discharge from the opening made in the tegmen antri.

CHAPTER XI

THE SURGERY OF THE ACCESSORY SINUSES

THE "KEY" TO DISEASES OF THE SINUSES, OR THE "VICIOUS CIRCLE" OF THE NOSE

In the chapter on the Etiology of the Inflammatory Diseases of the Nose and Accessory Sinuses it was shown that the chief predisposing cause of inflammation of the sinuses is an obstruction in the region of the middle turbinated body and the hiatus semilunaris. The obstructive lesion may be a deflection of the nasal septum, an enlarged or cystic middle turbinate, an enlarged bulla ethmoidalis, or cells in the uncinate process, the median wall of the infundibulum (Figs. 138 to 144). As the frontal, anterior ethmoidal, and the maxillary sinuses drain into the infundibulum (exceptions noted, p. 164), an obstruction in this region may occlude either or all of these sinuses. When either of them is the seat of inflammation it is always advisable to make a careful examination of this region. The area to be thus examined is shown in Fig. 145 within the circle. These structures may be designated the "key" to inflammation of the sinuses, or the "vicious circle" of the nose. Being the key to the etiology of infection, it is also the key to the treatment of the infection; that is, if the obstruction predisposing the sinuses to infection is located within the area of the circle, it is obvious that if this area is freed from obstruction the chief etiological factor will have been removed, and having been removed the infectious process tends to subside.

The following principle may, therefore, be given as a working basis in the treatment of inflammatory diseases of the sinuses composing Series I. (See Chapter IX.)

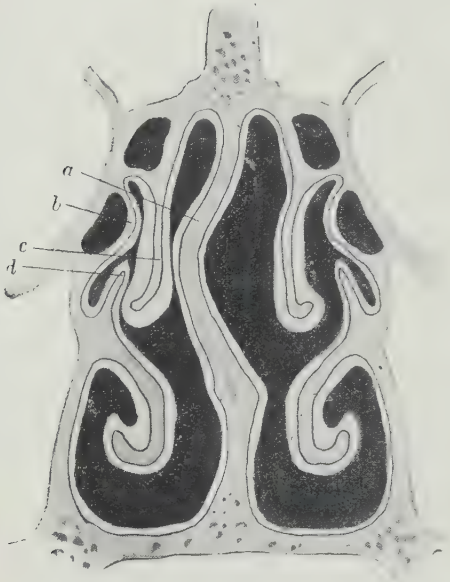
Remove the obstruction within the "key," or "vicious circle," before attempting more radical measures.

By so doing the drainage of the sinuses may be established and a cure result. This principle is of so nearly universal application that it forms a good working basis, and, if observed, will prove of inestimable value, as it will often obviate the necessity of resorting to the more radical operations in the treatment of the sinuses. Should the recommendations given above fail to relieve the disease, the more radical operative procedures may be performed in due time.

Various writers have made clinical observations that meningitis is more likely to follow the radical external operation if an intranasal operation is performed a few days prior to the radical operation. The following deduction is, therefore, obvious:

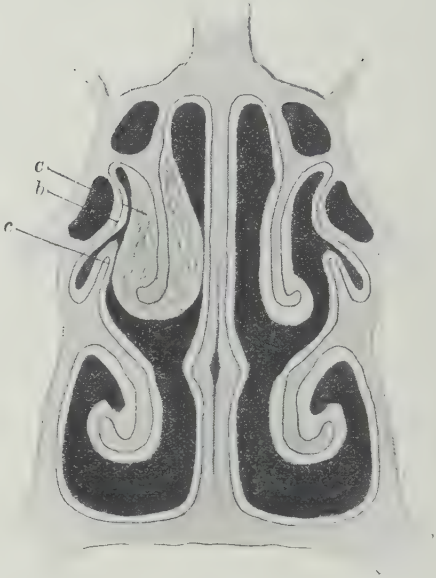
Never perform a preliminary intranasal operation a few days before a radical operation on a sinus.

Fig. 138



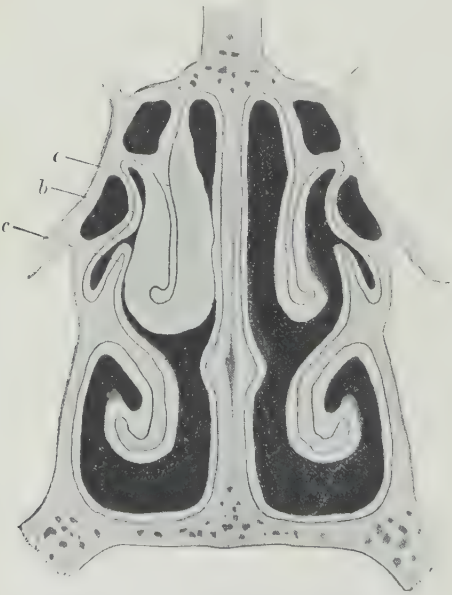
A high deviation of the septum, causing closure of the infundibulum: *a*, high deviation of the septum; *b*, inner wall of the bulla ethmoidalis; *c*, middle turbinate crowded against the outer wall of the nose and blocking the drainage of the infundibulum.

Fig. 139



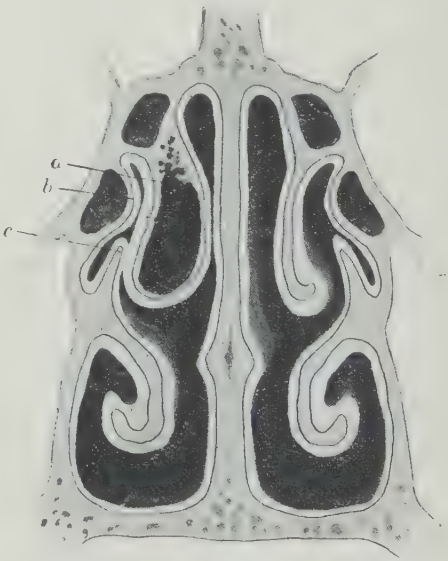
Cross-section through the nose: *a*, hyperplasia of the middle turbinated body, which crowds upon the uncinate process (*c*) and closes the infundibulum.

Fig. 140



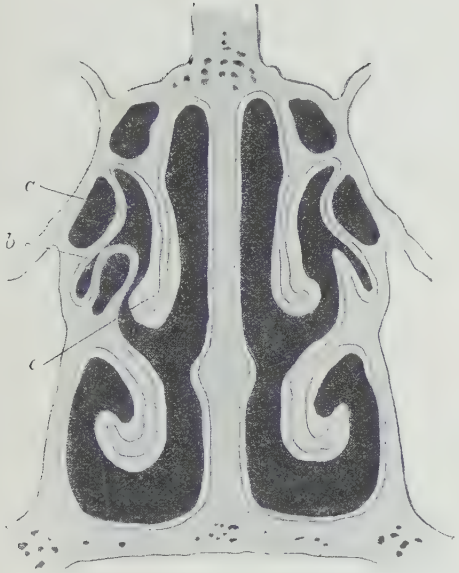
Edema of the mucous membrane of the middle turbinate, blocking the infundibulum: *a*, edematous middle turbinate; *b*, bulla ethmoidalis; *c*, uncinate process or inner wall of the infundibulum

Fig. 141



A large cell in the middle turbinated body, occluding the infundibulum: *a*, cell in middle turbinate; *b*, the inner wall of the bulla ethmoidalis; *c*, the uncinate process or inner wall of the infundibulum or gutter.

Fig. 142

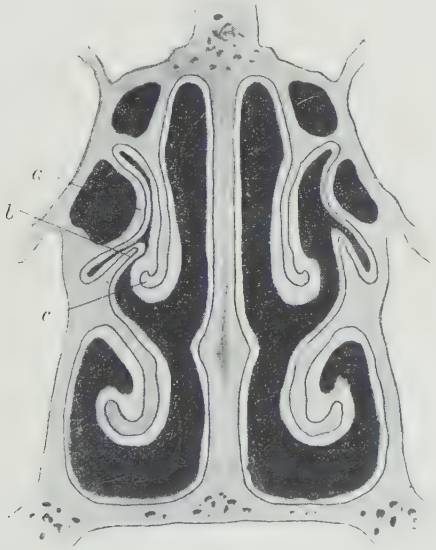


Cell in the uncinate process (*b*) blocking the infundibulum; *a*, bulla ethmoidalis; *c*, middle turbinated body.

Fig. 143

The middle turbinated body (*a*) clinging to the outer wall of the nose and blocking the infundibulum; *b*, inner wall of the bulla ethmoidalis; *c*, uncinate process or inner wall of the infundibulum.

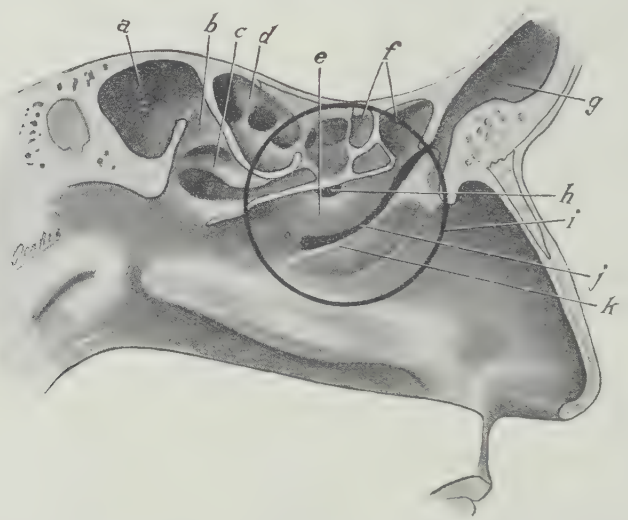
Fig. 144



Enlargement of the bulla ethmoidalis, blocking the infundibulum: *a*, the inner and distended wall of the bulla ethmoidalis, crowding inward and downward against the uncinate process and blocking the infundibulum; *b*, the uncinate process; *c*, the middle turbinate, which, on account of the bulging bulla, appears to be the cause of the blockage, whereas the bulla blocks.

Several days or a few weeks should elapse between them, to allow a wall of protecting granulation tissue to be formed. An additional reason for delaying the radical operation is, to allow sufficient time to elapse to determine whether the intranasal operation is adequate to cure the disease. I have seen serious cases cured most unexpectedly under such treatment. I wish to state most emphatically, however, that, having found the simple intranasal operation ineffective, the surgeon should unhesitatingly perform a more radical operation. My plea is for rationalism rather than against radicalism. I do not plead for so-called "con-

FIG. 145



The "vicious circle" of the nose: *b*, the sphenó-ethmoidal fossa; *c*, the superior turbinated body; *d*, posterior ethmoidal cells; *e*, bulla ethmoidalis; *f*, anterior ethmoidal cells draining into the frontonasal canal; *g*, frontal sinus; *h*, the ostium of the bulla ethmoidalis; *i*, hiatus semilunaris; *k*, the uncinate process or outer wall of the infundibulum or gutter on the outer wall of the nose into which the frontal, anterior ethmoidal, and maxillary sinuses usually drain. The high light below and anterior to *j* and *k* indicates the inferior boundary of the infundibulum or gutter into which the sinuses drain. The middle turbinated body is removed to exhibit the anatomical details beneath it.

servatism," a term which has been used to justify timidity and surgical inefficiency. The true conservative is a rationalist who dares to refrain from radical procedures, and yet who dares to undertake them when indicated.

SURGERY OF THE FRONTAL SINUS

How to Choose a Sinus Operation.—In a study of the operative indications, the sinuses naturally fall into three groups, namely: (1) The maxillary, (2) the posterior ethmoidal and sphenoidal, and (3) the frontal and anterior ethmoidal sinuses. This subdivision is due to the anatomical arrangement of the drainage system of the sinuses, and the relation of some of the teeth to the floor of the antrum.

The drainage and ventilation of the antrum are effected through the osteum maxillare, which opens into the bottom of the infundibulum, a gutter-like depression in the outer wall of the nose, beneath the middle turbinated body. In about half of the cases the sinus has additional openings above and posterior to the infundibulum in the membranous portion of the nasomaxillary wall. It would be of interest to know whether the accessory openings influence the frequency or character of the infection of this sinus. Theoretically the accessory ostea should reduce the frequency and severity of the infection and inflammation on account of the better drainage and ventilation they afford. If, for example, the osteum maxillare is obstructed, the accessory ostea would still afford ample drainage and ventilation and thus reduce or prevent infection and inflammation. If, however, there are no accessory ostea and the osteum is obstructed, the conditions are more favorable for infection and inflammation. We propose, therefore, to base our study upon the broad principle of adequate drainage and ventilation, and certain other conditions which we believe are of secondary importance.

1. *The Maxillary Sinus.*—When the maxillary sinus is the site of infection and purulent inflammation, how shall we determine what type of operation is required to establish free drainage and ventilation?

Several factors enter into the equation. Of these the first is, Was the infection of intranasal or of dental origin? If of dental origin, the proper treatment of the carious tooth, combined with simple puncture through the nasomaxillary wall, may be all that is required to effect a cure. The acuteness and chronicity of the disease also influence the method of treatment. If the attack is acute and primary, astringent remedies, cocaine, adrenalin, and antipyrine, locally applied, or simple puncture and lavage may be effective. If the attack is an acute exacerbation upon an old chronic inflammation these remedial measures would in a large number of cases only relieve and not cure the disease.

If the exacerbation is severe and one of several such manifestations, the nasomaxillary wall should be extensively resected. The partial removal of this wall is usually attended by a continuation of the disease, as the opening rapidly closes by granulations from its margins.

If the conditions described in the preceding paragraphs are present and polypi are found in the maxillary cavity, the complete removal of the nasomaxillary wall except by the Canfield-Ballenger operation, may not establish a cure, as the polypi and diseased mucous membrane are not accessible through the nose, and simple drainage and ventilation are not always followed by the regeneration of the diseased mucous membrane. In such cases the Caldwell-Luc, the Denker, or the Canfield-Ballenger operation is indicated. The latter operation is the best and simplest.

2. *Posterior Ethmoid and Sphenoid.*—When confronted with posterior ethmoidal and sphenoidal disease there is no choice as between an intranasal and extranasal operation, as these cavities can only be reached through the nasal chambers, unless, indeed, they are approached *via* the inner angle of the orbit, a route much less desirable than the intra-

nasal one, except when the anterior ethmoidal cells are also extensively diseased, in which case it may be necessary to operate *via* the external route.

If the case is catarrhal, or only discharges pus during an attack of acute coryza, the removal of the middle turbinal may be all that is necessary. Such cases are usually characterized by headache, more pronounced in its beginning on one side, often present upon awaking in the morning, and dizziness upon stooping. Objectively the nasal cavities may be free of purulent secretion, except when the patient is suffering from acute coryza, at which time purulent secretion is present. The middle turbinal is usually bullous and lies against the septum. Such cases are often cured by the removal of the middle turbinal. If in addition to the above symptoms there is a chronic purulent secretion discharging through the olfactory fissure, and through the choana into the epipharynx, and if polypi are found in the olfactory fissure the middle turbinal and posterior ethmoidal cells should be completely exenterated. The anterior and a portion of the inferior walls of the sphenoidal cavity should also be freely removed. The complete exenteration of the posterior ethmoidal cells is not always easy, or even possible to do, in some cases, as the anatomical arrangements of the cells in relation to the sphenoid and the other structures of the head renders them inaccessible except by jeopardizing vital structures. Dr. Wales has a bony specimen in which the cells pass around the side of the sphenoid sinus to its posterior aspect, and seem to communicate with the mastoid cells. In such a case it is obviously impossible to completely exenterate the ethmoidal cells. I have a case under my care in which the posterior ethmoidal cells extend backward along the side of the sphenoid sinus on the right side, and in so far as I am able to demonstrate they may extend behind it. I have been unable in this case to completely check the ethmoidal discharge; vaccine treatment might be of great value here.

If the sphenoidal involvement is acute or subacute, the removal of the middle turbinated body will usually establish adequate drainage and ventilation. If it is chronic and the mucous membrane is edematous or has undergone hyperplastic changes (polypi), it will be necessary to first remove the middle tubinated body and then completely remove the anterior wall of the sphenoidal cavity, especially at its lower portion, where the wall may be from one-eighth to one-quarter of an inch in thickness. A Hajek or Fletcher punch forceps should be used for this purpose, as they are powerful enough to cut the heavy bone.

The Frontal and Anterior Ethmoidal Cells.—As the frontal and anterior ethmoidal cells are nearly always simultaneously involved, it is necessary to consider the surgical indications together; indeed, this fact throws a suggestive side light upon the choice of an operation. It is obvious that if both systems of cells are simultaneously involved, an operation must be chosen that will adequately drain both. If the disease is primary and acute, the application of ischemic remedies, as cocaine and adrenalin, to the upper and anterior portions of the nasal chambers may establish good drainage and ventilation. If this fails, the removal of the anterior

half of the middle turbinal may clear the region of the infundibulum (vicious circle) of the obstruction, and thus establish free drainage and ventilation. If, after removing the middle turbinal, polypi are found in the region of the hiatus semilunaris, they should be removed.

If the polypi rise from the interior of the anterior ethmoidal cells, it will be necessary to partially exenterate them *via* the nasal route. If the bulla ethmoidalis is enlarged and overhangs the hiatus, it should be removed to establish drainage and ventilation of the infundibulum. Indeed, any obstruction in the "vicious circle of the nose," such as a high deviation of the septum crowding the middle turbinated body against the hiatus semilunaris, a bullous or hyperplastic middle turbinal, an enlarged or overhanging bulla ethmoidalis or cells in the uncinate process which obstruct the infundibulum, should be removed.

If the case is mild and chronic the procedures above enumerated may effect a cure, though in many instances they will fail. If they fail the case may still be treated through the intranasal route, by enlarging the frontonasal duct and removing the floor of the frontal sinus. The choice of operation under these conditions lies between the Ingals, the Halle, and the Good operations. The Ingals operation enlarges the frontonasal duct with a pilot burr, the Good operation with a rasp, while the Halle operation removes the floor of the frontal sinus with a series of specially devised burrs. The Ingals operation does not establish as large an opening as the Good and the Halle operations. It also necessitates the use of a drainage tube for several weeks or months.

If the case is chronic and is attended by acute exacerbations with frontal tenderness, an external operation should usually be performed, as hyperplastic changes (polypi) are usually present and cause obstruction. Of the external operations I can only recommend the Killian, as it also includes the exenteration of the anterior ethmoidal cells. Of the thirty-five Killian operations performed by me the external deformity has been an almost negligible quantity except in one case in which the frontal sinuses were large and deep.

Special Indications.—There are certain special indications for operations upon the sinuses which have not been given in the preceding portions of this section. They are (*a*) ocular symptoms, (*b*) skiagraphic findings, (*c*) intracranial complications, and (*d*) hay fever.

(*a*) *Ocular Symptoms.*—It is now generally admitted that certain eye symptoms are caused by sinus infection, whether the end results of the infection are catarrhal inflammation, hyperplasia (polypi), or suppuration. It is beyond the province of this section to enter into a general discussion of eye symptoms in relation to sinus disease, hence I will limit my remarks to a few ocular indications for the surgery of the sinuses.

Asthenopia or imbalance of the muscles of accommodation is frequently due to irritation within the ethmoidal cells. I have repeatedly proved this by the proper attention to the ethmoidal labyrinth, a procedure which has been followed by the complete relief of the asthenopia. Whereas, before the treatment of the ethmoidal disease the patient could

not be properly fitted with reading glasses, after it he could be perfectly fitted, or, as I have found in a number of cases, glasses were not needed.

In these cases it is often only necessary to fracture the anterior portion of the middle turbinal and force it toward the septum, or remove a portion or all of the middle turbinated body. In others it may be necessary to exenterate some or all of the cells.

Partial or complete blindness may be due to infection of the sinuses, the blindness in all probability being due to the absorption of toxic material from the affected sinuses. I have seen and operated upon three cases of toxic amblyopia of sinus origin. In one case of complete blindness of recent origin I did a double exenteration of the ethmoidal and sphenoidal sinuses, which was followed by the return of vision to normal. In the second case the result was not as good, as the blindness had been present for four months before the exenteration. In the third case no improvement followed the operation, progressive blindness having been present for nearly two years before operation.

These cases are referred to here for the purpose of emphasizing sudden or rapidly developing blindness as a possible indication for the surgical treatment of the sinuses. Pressure paralysis of the optic and motor nerves of the eye may also constitute indications for operation upon the ethmoidal and sphenoidal sinuses.

(*b*) *Skiagraphic Indications.*—Skiagraphs of the sinuses show the presence of disease, but not the exact pathological condition present. For example, given four cases, the first with catarrhal inflammation, the second with simple suppurative inflammation, the third with polypi and suppuration, and the fourth with the frontal sinus denuded of its mucous membrane, the skiagraphic plates will each be cloudy over the frontal sinus, but will not indicate the pathological differences. The plates will, however, show anatomical points which will aid in choosing an operative procedure.

First, the size and depth of the frontal sinus is shown. If deep and large the operator should hesitate to do a Killian operation on account of the great deformity which would probably result; he should choose the *Killian plus the osteoplastic bone flap* shown in Beck's operation. If the sinus is not deep and large, and all other symptoms indicate an external operation, the Killian operation should be elected.

Second, the plate will show the presence or absence of septa in the frontal sinuses (Coakley). If absent, and the case is simple, an intranasal operation may be performed with a reasonable assurance of success. If septa are present and almost completely divide the sinus, an intranasal operation will probably fail.

Third, the skiagraphic plate will show whether or not the ethmoid cells extend over the orbital roof. If they do, an intranasal operation upon the ethmoidal labyrinth will probably fail to effect a cure. Such a case will probably require a Killian operation if the frontal is also involved, and a Moure operation if the ethmoidal alone is involved.

(*c*) *Intracranial Complications.*—The significance of brain abscesses and meningitis complicating infection of the nasal accessory sinuses is

sometimes an important one. When brain abscess complicates the sinus disease the affected sinus or sinuses should be radically operated and the abscess drained. When a circumscribed extradural abscess (meningitis) complicates the disease, the sinuses should not be operated without due precautionary measures. To operate under such conditions would in all probability excite an acute exacerbation of meningitis, cause it to become diffuse, and hasten a fatal issue, as occurred in one of my cases.

(*d*) *Hay Fever.*—Whether hay fever is ever due to sinus disease is, I presume, still an open question. The late Dr. Schadle called attention to the apparent relationship of antrum disease to hay fever, and cited some cases which were greatly benefited by lavage of the affected antra. Pynchon has since then also made similar observations. Personally, I have not observed such a relationship. I have, however, seen hay fever subjects who were afflicted with ethmoidal and frontal disease. In one case I did a complete exenteration of both ethmoidal labyrinths, and afterward did a double Killian operation upon the frontal sinuses. The patient after four years is completely relieved of the hay fever symptoms. Previous to the operation the patient was compelled to sleep in a sitting posture for three months each summer and autumn. This, and other cases of a similar nature, have led me to infer that sinus disease, especially anterior ethmoidal and frontal, may be a more frequent cause of hay fever than heretofore suspected. I suspect that catarrhal frontal and ethmoidal sinuitis is a more frequent cause of hay fever than the suppurative type. The constant discharge of acrid mucus over the area of distribution of the anterior ethmoidal nerve might well render this area hypersensitive to the pollen or other irritating substances which are the active causes of hay fever. I do not claim, nor do I believe, that sinus disease is a constant factor in the etiology of hay fever. I only suggest that it is sometimes a cause. If this is true, hay fever may be an indication for the surgical treatment of the sinuses, more especially the frontal and anterior ethmoidal.

Surgical treatment of frontal sinuitis may be divided into (*a*) intranasal, and (*b*) extranasal operations.

The intranasal operations consist in the removal of obstructions within the "key," or "vicious circle," and in the more extensive operations of Halle, Good, and Ingals.

THE AUTHOR'S OPERATIONS WITHIN THE "VICIOUS CIRCLE"

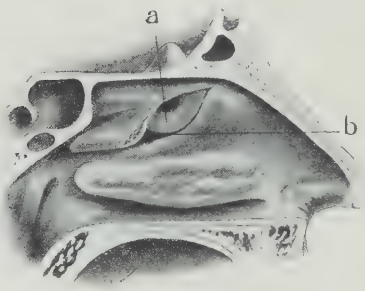
Intranasal Operations for Frontal, Anterior, Ethmoidal, and Maxillary Sinuitis.—(*a*) Local cocaine anesthesia should generally be depended upon, though general anesthesia is preferable in certain cases.

(*b*) Remove the middle turbinated body or such part of it as obstructs the area within the circle shown in Fig. 145. Even though the middle turbinate does not actually obstruct the hiatus and infundibulum, it may be necessary to remove a portion of it to expose the field to surgical

intervention. Physiologically there is little objection to the removal of this structure. The olfactory nerve is not distributed to its mucous membrane, and the "swell bodies" are rudimentary. The method of its removal should be selected with reference to the anatomical conformation and the individual preference of the surgeon. The author's turbinal knife is usually well adapted to the purpose.

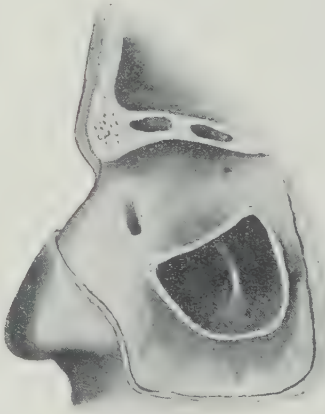
(*c*) Remove all of the anterior ethmoidal cells that can be reached with the curette, Grünwald forceps, or other instruments. Owing to the wide variation in the distribution of the anterior ethmoidal cells, the area of curettement varies in each case. In some subjects all the cells are not accessible to the curette. Occasionally one of the cells extends over the orbital roof posterior to the frontal sinus, as shown in Fig. 147. In other cases a cell encroaches upon the floor of the frontal

FIG. 146



Showing a large bulla ethmoidalis (*a*) encroaching upon the hiatus semilunaris; (*b*) the hiatus semilunaris. The middle turbinate has been removed. (Dr. W. A. Fisher's specimen.)

FIG. 147



The anterior cell is the frontal sinus; the posterior one is one of the anterior ethmoidal cells extending half-way across the orbital cavity, and is inaccessible to operation except by bent curettes through the nasal chambers. The author recently operated on three such cases. (Dr. W. A. Fisher's specimen.)

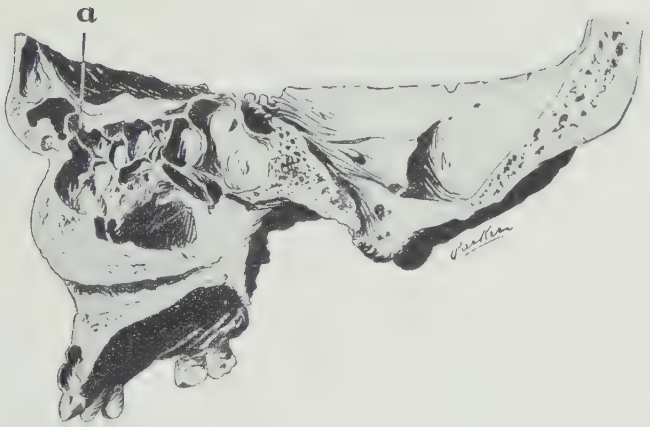
sinus and forms the so-called bulla frontalis, as shown in Fig. 148. The dense bone of the frontonasal spine of the superior maxillary bone often shields some of the most anterior of the cells from the curette. For these reasons the total exenteration of the anterior ethmoidal cells with the curette is not always possible by the intranasal route. As a consequence the frontonasal canal and the infundibulum cannot always be cleared of obstructive lesions. Drainage and ventilation of the frontal sinus are not, therefore, always possible by this method of operating.

Should the subsequent course of the frontal sinuitis prove the inadequacy of the operation, either the Halle, Good, or Ingals or one of the external operations is recommended. After an experience in more than

four hundred cases operated on *via* the "vicious circle" of the nose, I am convinced that but few cases of frontal and ethmoidal sinuitis require more radical surgical interference. In only 3 per cent. of the cases was it necessary to perform an external operation. As the infundibulum is the outlet of the drainage system of the sinuses comprised in Series I, and as the anatomical deformities of the septum, middle turbinate, and bulla ethmoidalis often obstruct the drainage and ventilation of the infundibulum, it is a rational conclusion that if the obstructive anatomical lesion is removed, drainage will be restored and the infection and inflammation cured.

Hemorrhage is the most troublesome complication attending this operation. The parts are chiefly supplied by the anterior and posterior ethmoidal and a branch of the sphenopalatine artery (Plate I, Fig. 1). They are of considerable size and may bleed freely, though in my experi-

FIG. 148



Showing the nasal sinuses of the right side of the head. The naso-antral wall, inferior turbinate, and the middle turbinate are removed. One of the anterior ethmoidal cells (*a*) projects into the floor of the frontal sinus and forms the so-called bulla frontalis. (Author's specimen.)

ence they rarely do so. The hemorrhage, though not profuse, usually continues for about twenty-four hours. A firm tampon of gauze in the upper portion of the nasal cavity readily checks it. Fortunately it is rarely necessary to introduce a tampon for this purpose. The presence of the tampon may prove as serious as the operation, as it may fracture the orbital plate and expose the orbital contents to infection. A tampon should not, therefore, be introduced except in case of severe hemorrhage. Drainage is of more importance than the control of a slight hemorrhage. Place the patient in a hospital if possible, as the hemorrhage can be kept under better control than it can if the patient is at home.

After-treatment.—Instruct the patient to introduce a pledget of cotton in the vestibule of the nose and to remove and renew it as often as it becomes soiled with blood and secretions. This protects the denuded surfaces from being irritated by the inspiratory current of air and prevents the blood trickling over the upper lip. A dusting powder of bismuth-

iodine should be insufflated once or twice daily. Healing usually occurs in about fourteen days, and if the exenteration is complete the space in the ethmoidal region should be free and roomy. For a few days after the operation small pledgets of cotton, saturated with a 10 per cent. aqueous solution of ichthyol, should be introduced every four hours into the attic of the nose to promote osmosis and asepsis of the surgical field.

HALLE'S OPERATION ON THE FRONTAL SINUS

Max Halle, of Berlin, secures entrance to the frontal sinus by the intranasal route by means of burrs and a protector to the internal plate of the frontal bone. The chief source of danger attending this operation is the injury of the internal plate of the frontal bone, thereby opening an avenue of infection to the meninges and brain. The grooved protector is intended to prevent injury of this plate, and it should always be used.

The anatomical barrier to the removal of the floor of the frontal sinus is the backward projection of the spina nasofrontalis of the superior maxillary bone. This dense, heavy bone was regarded as an insurmountable barrier to the floor of the frontal sinus by the intranasal route, until Halle recently called attention to his method of operating.

Indications.—The Halle operation is indicated in those cases of frontal and anterior ethmoidal sinuitis which have resisted the removal of the anatomical obstructive lesions within the "vicious circle" of the nose, and in which there is no fulminating symptom, as meningitis, orbital abscess, or external perforation. When these symptoms are present an external operation should be performed. (See How to Determine Which Operation Should be Elected.)

Technique.—(*a*) Induce local anesthesia with cocaine.

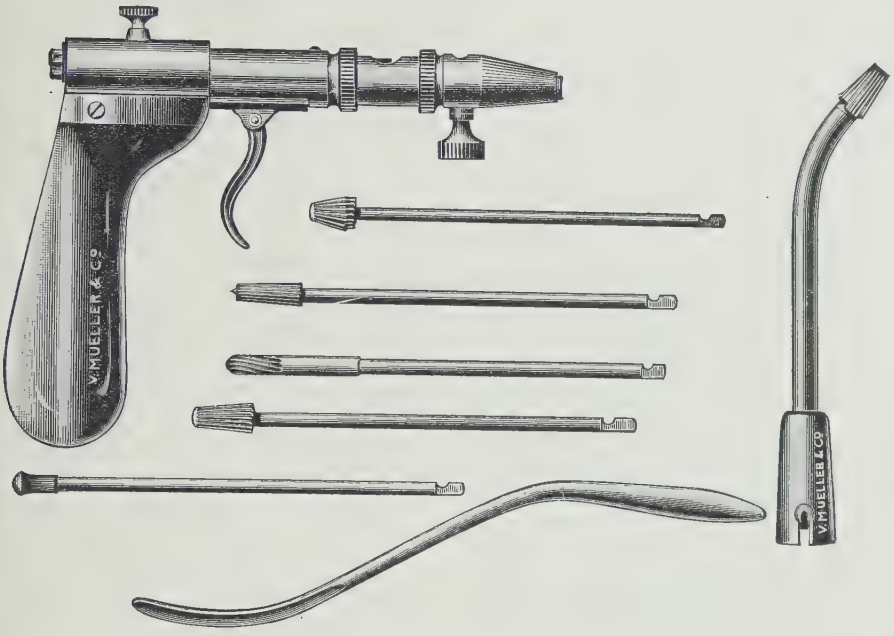
(*b*) Introduce a probe into the frontonasal canal for a distance of $2\frac{1}{2}$ to 3 cm. after it enters the infundibulum or hiatus semilunaris, as when it is passed upward and forward this distance it has entered the frontal sinus.

(*c*) Introduce the protector beside the probe for the same distance.

(*d*) Next engage the pointed drill (Fig. 149) against the under and posterior border of the spina nasofrontalis, just in front of the protector. Direct the drill forward and upward and remove enough of the bone to allow the blunt-pointed drill to be introduced. The sharp-pointed drill should only be used to make an opening large enough to permit the introduction of the blunt-pointed one, as to use it further might lead to injury of the internal plate of the frontal bone. The blunt drill will not do this.

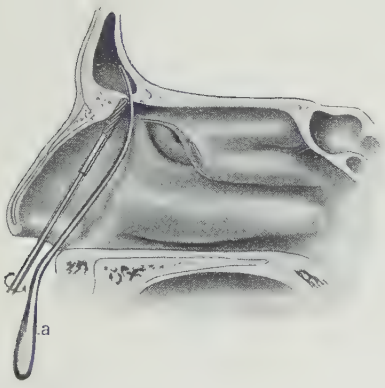
(*e*) With the blunt drill remove enough of the bone to permit the introduction of the pear-shaped drill (Fig. 150), the thickened portion of which is rounded and polished. According to Halle, the blunt or bulbous drill can inflict no serious injury to the meninges or brain, provided a little care is exercised. The entire floor can be drilled away with it, and so large a part of the external plate of the frontal

FIG. 149



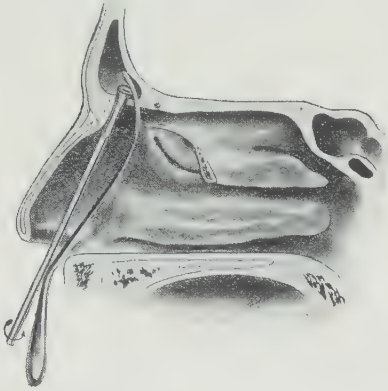
Halle's frontal sinus drills and handle.

FIG. 150



Halle's first step in removing the nasal process which forms the floor of the frontal sinus at its inner extremity. A metal protector (*a*) is introduced into the frontonasal canal to prevent injury to the inner or cranial wall of the frontal sinus. The pointed burr is only used to begin the operation, after which blunt, smooth-tipped burrs are used, as they will not penetrate the inner or cranial bony wall of the sinus if they should accidentally come in contact with it.

FIG. 151



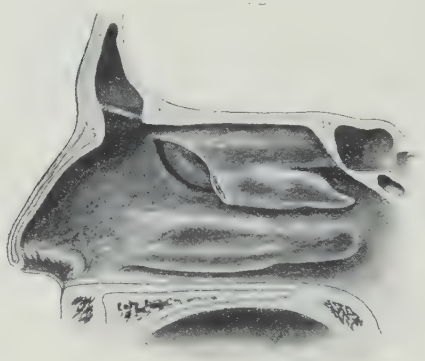
The round-tipped burr removing the floor of the frontal sinus by the intranasal route. The protector is in position and the rounded, polished tip of the burr renders injury to the cranial wall of the sinus improbable.

bone in a downward direction that the instrument can be felt from without. It is necessary that the assistant take the precaution to push his finger well into the orbit, so that he can control the head of the instrument (drill) and prevent it going too far to the front or the sides.

The mucous membrane of the frontal sinus may thus be exposed to ocular inspection and treatment through the nose if enough of the bone is removed, as shown in Fig. 152.

(*f*) The after-treatment consists in first packing the sinus with iodoform gauze, and the subsequent use of alcohol, protargol, or the nitrate of silver to retard granulations and to promote the formation of epithelium. At a later period Halle instructs the patient to introduce a large cannula several times a day to prevent the formation of granulations and adhesions, though this should preferably be done by the removal of the granulations, caustic applications, etc., by the surgeon.

Fig. 152



The intranasal operation of Halle completed. The floor of the frontal sinus is widely opened and permits curettage and free drainage of the sinus.

(*g*) The anterior ethmoidal cells and middle turbinated body of the "vicious circle" are also removed in this operation. The posterior cells may also be removed at the same time by either of the methods described elsewhere in this chapter.

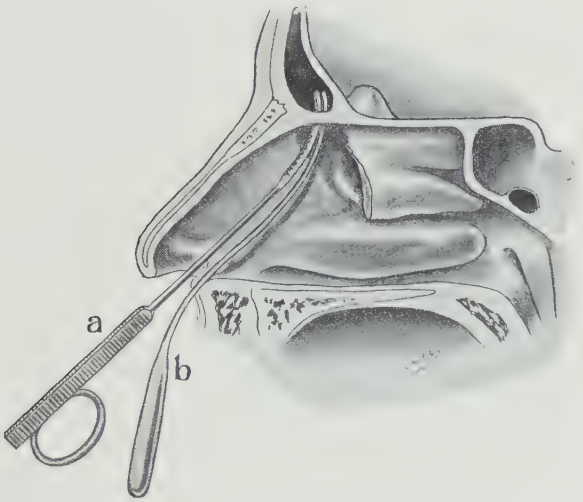
Good's Operation.—The first step of this operation is the removal of the anterior portion of the middle turbinated body, a procedure which, as I have shown, will often effect a cure of the frontal sinuitis, especially if it is of the simple catarrhal type, and is characterized by exacerbations of acute coryza.

The second step of the operation consists in the introduction of the guard and guide into the frontonasal canal (Fig. 153, *b*). The guard should have the normal curve of a frontal sinus probe or cannula, and is introduced with the same technique.

The third step of the operation consists of the introduction of the curved frontal sinus rasp into the frontonasal canal, in front of the guide, which is slightly hollowed or grooved. It may be necessary to use a little force, as the canal is too narrow to admit the rasp without crushing some of the anterior ethmoidal cells along its outer side. The rasp should be introduced until its tip emerges in the cavity of the frontal sinus (Fig. 153). The file-edge of the rasp should face anteriorly and outward, while the smooth surface should face posteriorly and medianward. The object of the rasp is to enlarge the frontonasal canal by removing some of the anterior ethmoidal cells, and to remove the floor of the frontal sinus.

After-treatment.—When the frontonasal canal has been enlarged and the floor of the frontal sinus removed, the wound may be maintained in a patulous condition by the use of a gold filigree tube, or, if a sufficiently large opening is made, the tube may be omitted. When the tube is not

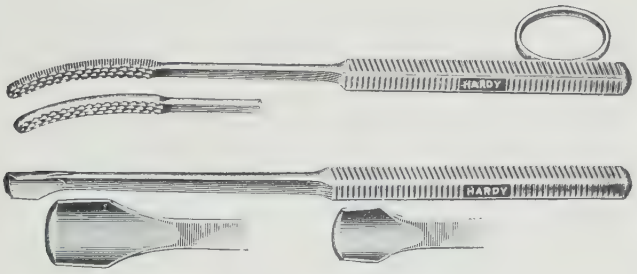
FIG. 153



Good's intranasal frontal sinus operation. *a*, Good's rasp removing the floor of the frontal sinus; *b*, the guide and protector in position.

used the area should be closely watched for exuberant granulations, which, if found, should be reduced with a bead of fused chromic acid crystals. The frontal sinus should be irrigated daily with boric acid solution until the purulent secretion ceases.

FIG. 154



Good's rasps and chisels.

This operation should not be undertaken unless it has first been demonstrated that a frontonasal probe will enter the frontal sinus *via* the frontonasal canal. If this cannot be done the rasp file might be misdirected, the posterior wall of the frontal sinus penetrated, and meningitis incited.

The Ingals Operation.—According to E. Fletcher Ingals, the author of this operation, from 95 to 98 per cent. of all cases of empyema of the frontal sinus may be cured by his operation. This accords with the results obtained by my intranasal operations. (See "Vicious Circle" of the Nose and the Exenteration of the Middle Turbinate and the Ethmoidal Cells *en masse*, and the various operations upon the tissues within the area of the "vicious circle".) As my experience broadens I am inclined to modify my original opinion as to the percentage of cures by operations *via* the intranasal route. I still believe, however, that a large percentage can be cured in this way.

The objections offered to the Ingals operation are: (*a*) That the internal plate of the frontal sinus may be injured, which would give rise to meningitis, though the guard and guide now used with the instrument will probably prevent such an accident, as with it the burr may be drawn forward away from the internal plate; (*b*) injury of the fossa ethmoidalis, which is a point in the anterior fossa near the cribriform plate, to which the dura is closely adherent, and which is regarded as especially susceptible to meningitis.

The Technique.—Ingals has performed all his operations under cocaine anesthesia, though a general anesthetic may be administered. The cocaine (20 per cent. in 2 to 1000 adrenalin) is injected into the frontonasal canal with a small curved cannula fitted to a hypodermic syringe. The cannula is inserted by the same technique which is used in probing the canal to the floor of the frontal sinus. From $\frac{1}{4}$ to $\frac{1}{2}$ ♏ is then injected, the cannula slightly withdrawn, and the same amount again injected. This process is repeated until the whole length of the frontonasal canal is cocainized. Two or more introductions of the syringe-cannula may be necessary to produce complete anesthesia.

If the anterior end of the middle turbinate has not been previously removed this region should also be cocainized.

1. Remove the anterior end of the middle turbinate. This should be done two or more weeks before the Ingals operation, or else just preceding it, preferably the former, because this procedure alone is sometimes followed by a cure of the empyema of the frontal sinus. (See "Vicious Circle" of the Nose.)

2. Introduce the probe-pilot into the frontonasal canal.

3. Slip the pilot-burr over the probe-pilot until the burr is at the lower extremity of the frontonasal canal. If it is desirable to protect the internal plate of the frontal bone from injury, the pilot-burr may be protected by a guard, as shown in Fig. 155. With this device the pilot-burr may be drawn forward, away from the posterior wall of the frontal sinus.

4. When all the parts of the instrument are adjusted the burr is gently pressed upward. It usually cuts its way into the frontal sinus in two or three seconds. It may be passed up and down through the opening thus made two or three times to insure a clear passage.

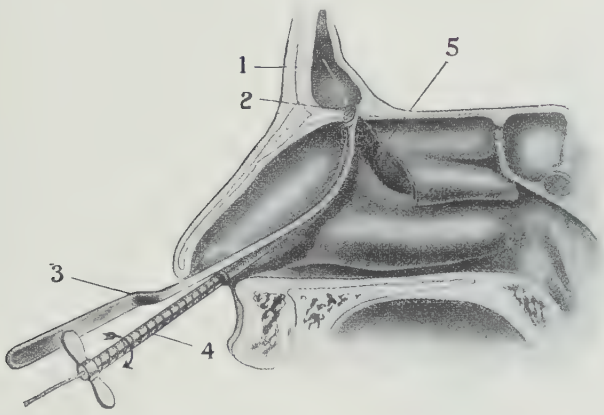
5. Introduce a one-inch strip of sterile gauze saturated in a 20 per cent. solution of the chloride of zinc into the enlarged frontonasal canal,

having previously swabbed the nasal mucous membrane with vaseline. Leave the gauze in place for about five minutes, to insure its caustic action. The gauze should be introduced through a suitably curved uterine packer.

6. A gold drainage tube is introduced into the enlarged frontonasal duct as follows:

The wire applicator of the uterine packer is first enveloped with a flexible spiral shield. The drainage tube is then slipped on the end of the applicator and introduced into the lower opening of the canal. The spiral shield is then pressed upward against the drainage tube, forcing

FIG. 155



The Ingals intranasal frontal sinus operation: 1, the pilot-probe over which the pilot-burr is placed; 2, the pilot-burr; 3, the guide with which the pilot-burr is drawn forward away from the posterior wall of the frontal sinus; 4, the flexible shaft; 5, the frontonasal canal.

it to the full depth of the canal. The applicator and spiral tube are withdrawn and the operation thus completed. Before introducing the gold drainage tube its spring ends are capped with a No. 2 gelatin capsule, which is further protected by a coat of vaseline to prevent it melting too rapidly when it comes in contact with the tissues. The capsule holds the flaring segments of the tube in position while it is being introduced. The capsule is dissolved in about five minutes and the segments of the tube spring apart and hold it in position.

The tube should be worn for about four months, though to wear it for a much longer period would not cause great inconvenience.

The frontal sinus should be irrigated daily through the tube.

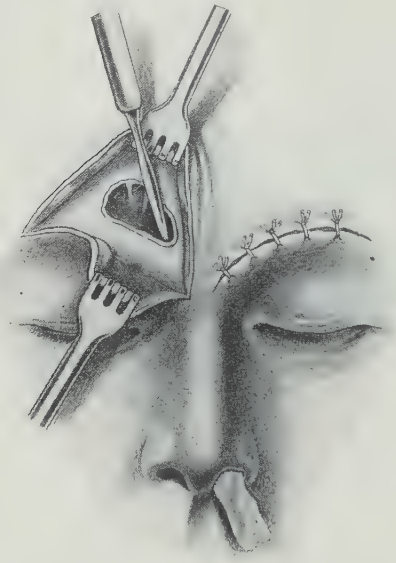
External Surgery of the Frontal Sinus.—On account of its location, the frontal sinus is sometimes less successfully treated by the intranasal route than either of the other sinuses. It is, therefore, necessary to resort to external methods of operating in a considerable number of chronic cases. The method of Hajek-Luc, or Ogston-Luc, as it is sometimes called, is one of the most efficient in uncomplicated cases of

chronic empyema of the frontal sinus. This method is not adapted, however, to those cases in which the anterior ethmoidal cells are to be exenterated. In such cases it is necessary to remove the floor of the frontal sinus and the processus frontalis of the superior maxillary bone to give access to the anterior ethmoidal cells. The posterior ethmoidal and sphenoidal cells are accessible by the intranasal route.

The Hajek-Luc Operation.—(*a*) The skin of the forehead and around the eye should be thoroughly cleansed and covered with a moist dressing twenty-four hours previous to the operation.

(*b*) The patient is placed upon the operating table and anesthetized.

FIG. 156



The Hajek-Luc operation. The anterior wall of the frontal sinus is removed, and the anterior ethmoidal cells are being removed through the floor of the frontal sinus with a curette. The left side has been operated on, a gauze wick introduced through the anterior ethmoidal wound and drawn out through the nostril.

(*c*) The dressing is then removed and the parts again washed. It is not necessary to shave the eyebrow, as it can be easily cleansed and is useful as a landmark; though I prefer to shave it, because it interferes with the removal of the stitches.

(*d*) An incision is made, beginning at the temporal end of the eyebrow and extending to the bridge of the nose (Fig. 156). A second incision may be started where the first leaves off, and extended upward as far as the upper limit of the frontal sinus, a fact which should be determined beforehand by skiagraphy.

(*e*) The skin and periosteum within this triangular incision are turned upward, thus exposing the outer plate of the frontal bone.

(*f*) A liberal portion of the bone is then chiselled away, thus exposing the frontal sinus to inspection and curettage.

(*g*) After determining the outline of the sinus and the character and location of pathological lesions, the morbid material is removed with a curette, and if bony septa are present they are broken down (Fig. 156).

(*h*) The frontonasal canal must be enlarged as much as possible, to establish free drainage into the nose. This is done by breaking down the anterior ethmoidal cells with a curette (Fig. 156), through the floor of the frontal sinus.

(*i*) A large rubber tube is inserted into the enlarged frontonasal canal and left in position for several weeks, or until all discharge ceases. The

nasal end of the rubber tube is seized with forceps from time to time, and moved up and down, to prevent adhesions. When all discharge ceases the tube is withdrawn through the nose.

(*j*) After inserting the rubber tube into the frontonasal opening the external wound is closed and allowed to heal by primary intention.

Advantages of the Operation.—The advantages of this method of operating are: (1) It avoids disfigurement, as the wound heals by primary intention; (2) the frontonasal canal is enlarged, the anterior ethmoidal cells eradicated; and (3) as they are invariably involved in frontal sinuitis, this operation is advantageous, because they are opened and drained in its performance.

Disadvantages of the Operation.—Relapse occurs in about 50 per cent. of the cases, because the curettement cannot be done thoroughly, as the ethmoidal cells are not accessible through the frontal wound. Suppuration of the scalp has been reported, and the operation has been followed by sinuitis on the opposite side. Severe intracranial complications have also been reported. Tilley cites one death in 5 cases.

Lermoyez reports 9 cases in which there were 8 relapses; 5 of the cases were subsequently cured by Kuhnt's operation, 1 by the repetition of the Hajek-Luc operation, while 2 died of meningitis (slow septicemia). It appears, therefore, that this method, while apparently very simple, is sometimes followed by very serious sequelæ. In view of these facts, it is usually better to adopt Kuhnt's operation, or at least a combination of the two. I believe this operation fails in such a large percentage of cases because the obstruction in the "vicious circle" of the nose is not removed; indeed, it is probable that this latter procedure alone would have given far better results than that given in the above statistics for the Hajek-Luc operation.

Kuhnt's Operation.—The object of Kuhnt's operation is to obliterate the frontal sinus by granulation from the bottom of the cavity. He resects the entire anterior wall (Hajek-Luc removes only a portion of it) and a portion of the floor or superior orbital wall. Curettement is thoroughly performed, but the frontonasal canal is not disturbed, as to do so he thinks may lead to reinfection of the sinus from the nasal fossa. Kuhnt does not close the external wound, but leaves it open for the introduction of the dressings and for drainage. A cure takes place in from three to six weeks. Relapse and sequelæ, according to Kuhnt, are rare, and recovery is the rule.

Disadvantages.—(1) External drainage and dressings must be continued for several weeks. (2) When a cure is accomplished the patient is more or less disfigured. (3) The anterior ethmoidal cells are unopened, though they are always simultaneously involved. (4) Diplopia has frequently followed, from injury of the pulley of the superior oblique muscle, or from inflammatory infiltration about the pulley or within the muscle.

The Kuhnt-Luc Operation.—This operation is a combination of the method of Kuhnt and Hajek-Luc, and consists in the free removal of the anterior wall of the frontal sinus, the enlargement of the frontonasal

canal, and the introduction of the funnel-shaped rubber tube, together with the closure of the primary skin incision. This gives a fairly good cosmetic result with frontonasal drainage and a partial ablation of the anterior ethmoidal cells, as in the Hajek-Luc operation, while it avoids, in a measure, the disfigurement attending external drainage, as practised by Kuhnt. There is more or less depression of the skin, which is caused by the removal of the bone, but this can be corrected, in a measure, by subsequent paraffin injections.

Kuster's Osteoplastic Operation.—A modification of the operation just detailed consists of making an osteoplastic flap instead of chiselling away the outer bony wall. The bony flap is formed by making a narrow incision with a V-shaped chisel along the upper border of the supra-orbital ridge for the whole length of the sinus. The incision is then extended upward into either end of the supra-orbital incision in directions corresponding to the outline of the sinus as shown by a skiagraph previously made. This incision may also be made with a narrow-bladed rongeur forceps, or the De Vilbiss bone-cutting forceps. After the bony incision above the supra-orbital ridge is made it is enlarged somewhat at either extremity to admit two rongeur forceps by means of which the bony plate is broken off and left attached to the soft tissue above. Considerable care must be exercised in handling the bony flap and soft tissues while they are being retracted, lest they be separated. The next step in the operation consists of the incision of the membranous lining of the sinus and the removal of the floor of the sinus. This is followed by a very thorough curettement of the anterior ethmoidal sinuses through the floor of the frontal sinus. After carefully cleansing the sinuses the wound is packed with gauze moistened with the compound tincture of benzoin. The external wound is closed with sutures, and on the fifth or sixth day two of the centre stitches are removed and the dressing taken out.

The object of this method of operating is the same as that of Kuhnt's operation. The eye symptoms are also the same. As Canfield has pointed out, there may be some deformity on account of the osteoplastic flap being lifted outward at its lower border by adhesions at the upper border of the bone flap to the posterior wall of the sinus, and subsequent contraction of the same. Again, the lower border of the osteoplastic flap is lifted outward somewhat by the removal of the gauze dressing. The lower border of the osteoplastic flap thus dislocated sometimes forms a ridge, which may be removed or corrected by a secondary operation. I see no reason why the wound should be packed as described. A better plan would be to pass a small wick of gauze through the enlarged frontonasal opening, to maintain its patency for a few days, and then to withdraw it altogether. This would obviate opening the external incision, as recommended, and would give a better cosmetic effect. A thorough exenteration of the anterior ethmoidal cells and the establishment of good drainage as recommended by me will nearly always be followed by a cure of the disease without an external operation. (See "Vicious Circle.")

Beck's Double Osteoplastic Operation.—The method of procedure is as follows (for Indications see How to Determine Which Operation Should be Elected):

1. An incision is made through the skin and subcutaneous connective tissue through the upper margins of the eyebrows, then downward and inward as far as is usually done in the Killian operation. These two incisions are then joined by means of a transverse incision across the bridge of the nose.

2. This skin and subcutaneous flap are then dissected upward until the upper limits of the frontal sinuses are exposed. This is determined by means of a celluloid tracing of the radiogram placed upon the frontal bone.

The Preparation of the Celluloid Tracing.—Take a piece of ground celluloid film, about three inches square, place over the radiogram (glass plate) negative, which is either in the transilluminating box or against a window glass. Trace the outlines of the sinuses with ink. The outline of the supra-orbital margins is made for the purpose of getting a fixed point. The celluloid model can be sterilized in bichloride of mercury and alcohol.

If the sinuses extend very high on the forehead, it may become necessary to make two small perpendicular incisions at the extreme limits of the flap over the external canthi.

3. Place the celluloid tracing of the radiogram over the frontal region and incise the periosteum all around the upper and lateral margins of the same, but not over the supra-orbital borders or at the root of the nose.

4. With a flat chisel the external table of the frontal sinus is then penetrated along the whole course of the above described tracing through the periosteum; this also severs the attachment of the septum of the frontal sinuses from the posterior surface of the external table.

5. This osteoperiosteal flap is then slightly pried open by means of a chisel, and a Gigli saw is inserted beneath the bone flap and carried down to its supra-orbital attachment.

6. The saw should be made to cut from within outward; a few strokes severing the bone, care being taken to preserve the periosteum intact. Great care must be taken not to cut through this structure; indeed, the entire thickness of bone should not be sawn through, as it will readily break when it is everted downward over the nose. The skin flap is then reflected upward and the periosteal bone flap downward, thus exposing both frontal sinuses. The right side (Fig. 157) shows the granulations removed, and the drill in operation enlarging the frontonasal canal. The left side shows the cavity filled with granulations and pus.

7. If only one sinus is to be exposed, the technique varies only in the osteoperiosteal flap, and in making the incision within the limits of the frontal sinus septum and the lateral limit of the sinus. The skin flap may be made by making a perpendicular incision from the internal angle of the orbit to the height of the frontal sinus, as indicated by the radiogram.

8. Thoroughly eradicate the diseased mucous membrane, but do not curette it; and enlarge the natural opening into the nose, using the

Halle trephine or Good's rasp for this purpose. Also remove the most anterior ethmoidal cells as completely as possible through the floor of the sinus. This can only be done completely by opening through the lateral wall of the nose, as in Killian's operation. This constitutes the weakness of Beck's operation.

9. Introduce a large rubber tube with a wick of gauze in its lumen into the enlarged frontonasal canal. The upper end of the wick is loosely folded within the cavity of the frontal sinus, while the other end is brought down to the floor of the nose, so that a small portion protrudes through the vestibule.

FIG. 157

Beck's osteoplastic operation upon the frontal sinus. The right side shows the probe in the frontonasal duct, and the frontal sinus freed of granulations and pus. The left sinus is still filled with granulations and pus.

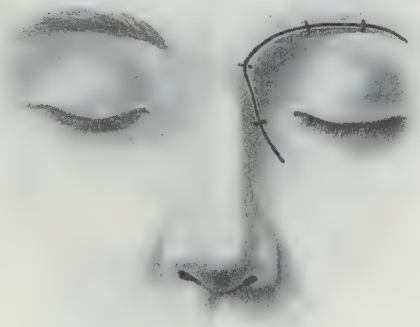
10. Replace the osteoplastic flap in its natural position. Bring the skin flap to its natural position and suture with silkworm gut, using the Halsted subdermal suture, with a few horsehairs, over the bridge of the nose.

The After-treatment.—The gauze should be removed on the day following the operation and on the third or the fifth day a gold or silver filigree tube should be inserted. In one case Dr. Beck used no tube, and four months after the operation the opening was sufficiently large to permit ventilation and drainage, the patient finally recovering.

The use of douches and blowing the nose should be avoided for several days after the operation. Indeed, the patient should snuff the secretions from the nose.

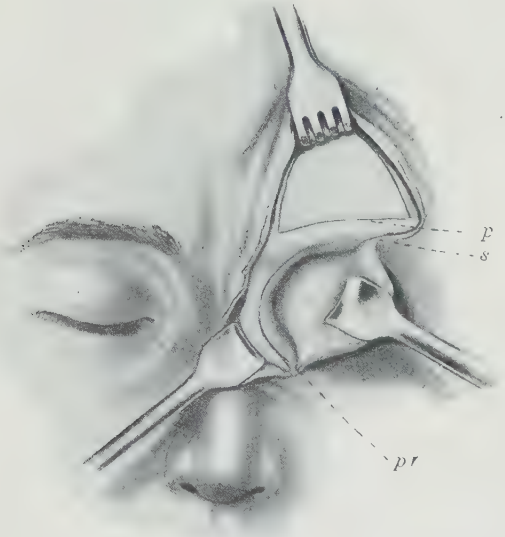
If this operation fails it may be converted into the Killian operation at a subsequent time.

FIG. 158



Killian's incision, with cross-cuts for guides in suturing.

FIG. 159



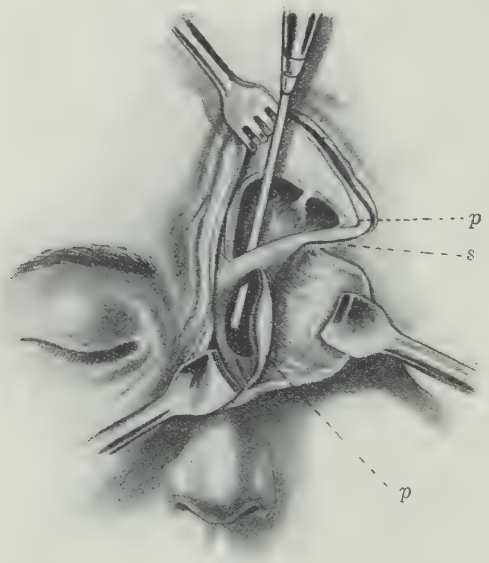
Showing the retraction of the skin flaps in the Killian frontal sinus operation: *P*, the periosteal incision 5 mm. above the skin incision; *S*, the skin incision 5 mm. below the periosteal incision; *P*1, the periosteal incision at the side of the nose.

The Killian Operation (for Indications see How to Determine Which Operation Should be Elected).—*Technique.*—After having prepared the field of operation, and having administered a general anesthetic, an incision is made through the eyebrow (previously shaved), beginning at

its temporal end, extending to the median line at the root of the nose, and then curving downward and outward below the base of the nasal bone (Fig. 158).

The periosteal incisions are two in number. The upper one is made parallel with the supra-orbital margin and 5 mm. above it, and extends from the temporal end of the incision to the median line of the nasal bones. The second periosteal incision begins internal to the attachment of the pulley of the superior oblique muscle (Fig. 159, p^1), passes inward and then curves downward and outward, following the direction of the incision of the skin around the inner canthus of the eye. This incision passes over the processus frontalis of the maxillary bone.

FIG. 160



The Killian frontal sinus operation completed: *P*, the periosteal incision 5 mm. above the superciliary skin incision; *S*, the superciliary skin incision 5 mm. below the periosteal incision; P^1, the periosteum elevated and everted along the side of the nose.

The soft parts, including the periosteum, are lifted from the bone, thus forming the skin and periosteal flaps, with the exception of the periosteum covering the superciliary ridge, where it is left intact to prevent the dislodgement of the pulley of the superior oblique muscle.

The frontonasal process and a portion of the nasal bone are chiselled away, thus exposing the anterior ethmoidal cells, which are removed through the opening. The entire anterior wall of the frontal sinus is completely removed with a chisel and rongeur forceps (Fig. 160).

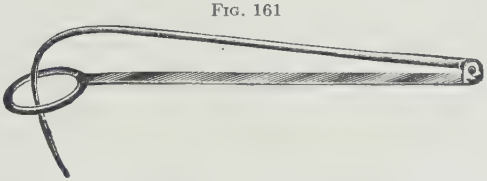
The cavity of the sinus thus exposed should be thoroughly inspected and curetted in all its ramifications. Killian insists that when the anterior bony wall is removed the mucous membrane should not at once be disturbed, but that it should be left intact as long as possible, so as to

avoid unnecessary infection of the external wound. He makes a small preliminary opening through the bone, and then with a probe, introduced between the bone and mucoperiosteum, determines the limitations of the frontal sinus. A skiagraph, previously taken, would obviate the necessity of this procedure. Having done this, he proceeds to remove all the bone necessary for its complete exposure. He then opens the membranous sinus and proceeds to inspect and curette it according to the conditions present. All septa are removed.

The next step in the operation consists in the removal of the floor of the sinus with a curette. As this operation is one wherein there is some danger of injuring the pulley of the superior oblique muscle, great care should be exercised to avoid it. As the pulley is variously located, this is not an easy matter. Dr. Ostrum has devised a pulley marker (Fig. 161) which may be applied to the tissues marking the location of the pulley, so that in the event of its detachment it may be sutured to the marked point, and thus prevent strabismus.

The opening around the processus frontalis may be enlarged upward and backward, to afford a better field for the curettement of the other sinuses, especially the ethmoidal and sphenoidal. Indeed, this opening

FIG. 161



Ostrum's localizer for the pulley of the superior oblique muscle.

should be united with the one in the floor of the frontal sinus, as shown in Fig. 160. Still exercising great care not to injure the nasal mucous membrane, the surgeon should introduce the curette through the opening made by the removal of the processus frontalis, and perform the curettement of the ethmoidal and sphenoidal cells. The limits of the ethmoidal cells are not difficult to determine with the curette, as the septa between them are usually very thin and easily broken down. The bone of the os planum and of the cranial plate is of greater density and resistance, and need not be mistaken for the septa between the cells. Personally, I prefer to remove the middle turbinate and posterior ethmoidal cells by the intranasal route. I also open the sphenoid by the intranasal route.

As the hemorrhage is considerable, the operator must depend upon his knowledge of the anatomical relations, the conditions of the diseased parts, and his sense of touch, rather than upon sight in exenterating the ethmoidal and sphenoidal cells. The wound should be thoroughly cleansed by irrigation with normal salt or boracic acid solution, then dusted with bismuth powder or bismuth paste, and the skin and periosteal incisions closed with sutures.

A point in the after-treatment insisted upon by Killian is, that the patient should be placed upon his healthy side and forbidden to blow

his nose. He must aspirate the secretions from the nose, and the nasal cavity should be inspected daily, carefully dressed, and exuberant granulations touched with nitrate of silver. If a double operation is performed the patient should lie upon his back and snuff the secretions from his nose.

A few days after the operation, if pus still comes from the sinus, gentle pressure over the skin should be made to force it into the nasal cavity. The patient should not be allowed to blow his nose, as to do so might force infected matter from the nose into the frontal cavity. The deformity following the operation is usually of moderate degree, and often becomes less conspicuous after a few months. The frontal sinus becomes more and more filled with granulation tissue, and the orbital fat pushes upward through the open floor of the sinus. In this way the depression becomes fairly well filled, except when the sinus is very large and deep, in which case the disfigurement may be very great.

This radical method of procedure is less likely to injure the pulley of the superior oblique muscle than the Kuhnt-Luc operation, or the Kuhnt operation, on account of the manner in which the periosteal incision is made, the periosteum over the superciliary ridge serving to hold the pulley in its place.

Taking all the facts into consideration, if the case is complicated by ethmoidal and sphenoidal disease and an external operation is deemed necessary, the Killian operation is the most effective and least disfiguring of the external operations.

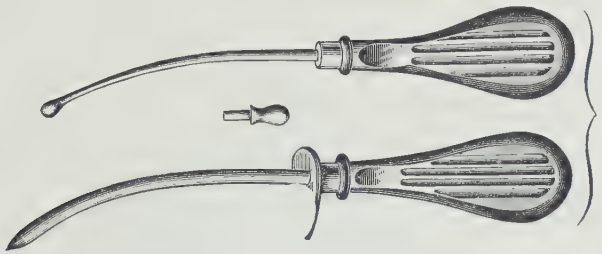
Of one hundred and twenty-five cases of frontal sinuitis in which the clinical diagnosis was confirmed by skiagraphy, in only twelve (10 per cent.) did I find it necessary to perform the Killian operation, the others being cured by giving surgical attention to the structures within the "vicious circle" of the nose. Of the twelve Killian operations performed by me, ten resulted in cure, two did not, as I failed to remove all of the anterior ethmoidal cells at the primary operation. The deformity was almost nil except in one case.

SURGERY OF THE MAXILLARY SINUS

Intranasal Operations (for Indications see How to Determine Which Operation Should be Elected).—The intranasal surgery of the antrum may include (*a*) the structures within the "key," or "vicious circle," (*b*) the interior turbinated body and the naso-antral wall, and (*c*) the removal of the uncinate process. If the infundibulum is blocked by morbid tissue or by anatomical peculiarities, they should be removed. In exceptional cases this will be sufficient to establish a healthy condition of the mucous membrane of the sinus. If, however, the mucous membrane has undergone marked degenerative changes, it is usually necessary to perform an extranasal operation, as the Caldwell-Luc or the Denker operation, or, better still, the Canfield-Ballenger operation, which is equivalent to the Denker and is performed intranasally.

Removal of the Naso-antral Wall.—This operation was first performed by Myles, and has had many advocates since then. Clinical experience has shown that a small opening in the naso-antral wall quickly closes, whereas a large one remains open permanently. Puncture and irrigation through a Krause cannula (Fig. 162) are often sufficient to effect a cure in

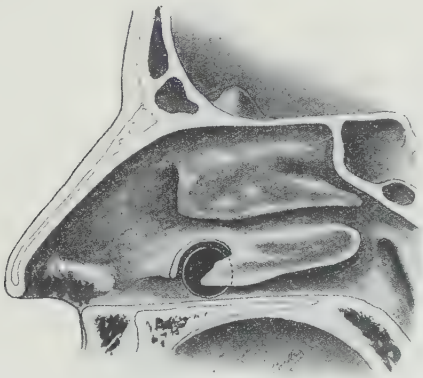
FIG. 162



Krause's antrum trocar with obturator.

acute and subacute inflammation of the sinus. The puncture should be made beneath the inferior turbinated body. The canunla may be introduced daily under cocaine anesthesia, with little discomfort to the patient. The irrigating solution may range all the way from normal salt and boric acid solutions to the more irritating solutions of zinc and

FIG. 163



Vail's operation on the maxillary antrum. The fragment of the turbinate extending over the naso-antral opening should be removed with biting forceps. Vail prefers his method, whereby a portion of the inferior turbinate is removed with the saw.

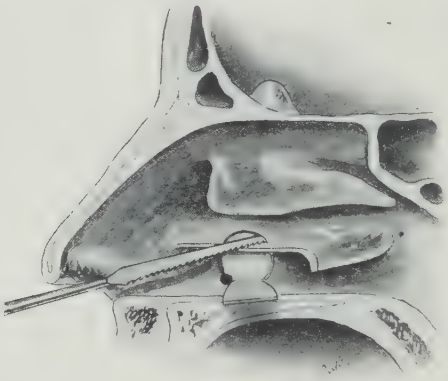
iodine. The usefulness of this procedure is largely limited to diagnosis, though it has some therapeutic value.

Many instruments have been devised for the removal of the naso-antral wall, some of which enable the operator to do the work with ease and precision. The instruments which have given the best satisfaction

are Vail's saw, Ostrum's forward cutting forceps, Wells' trocar and cannula rasp, Corwin's chisel, and Bishop's trephines.

Vail's Operation.—Vail's is perhaps the most ingenious and practical method for the removal of the naso-antral wall. His saw is slightly curved upon the flat, and when introduced obliquely through the naso-antral wall, makes a circular or oval incision, thus removing a large portion of the wall (Figs. 163 and 164), separating the nasal chamber from the antrum.

FIG. 164



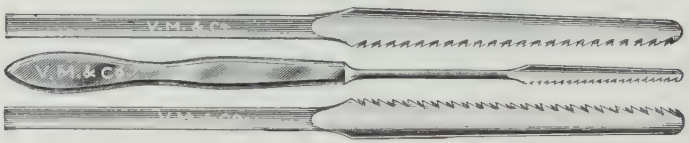
The removal of the naso-antral wall with Vail's convex saw. A mucous membrane flap is dissected from the naso-antral wall to be turned on to the floor of the antrum.

Technique.—(*a*) Induce local anesthesia of the inferior turbinal and of the inferior and middle meatuses.

(*b*) Remove the anterior half of the inferior turbinated body with the swivel knife or with scissors, or with the saw as it removes the naso-antral wall (Fig. 163).

(*c*) Puncture the naso-antral wall near the floor of the nose with Vail's perforator.

FIG. 165



Vail's antrum saw.

(*d*) Introduce the saw (Fig. 165) through the puncture and then make the circular or oval incision shown in Figs. 163 and 164. While the saw has a tendency to describe a circle, the size of the opening may be regulated by the operator, as the bone is thin. The opening should be made as large as possible, to overcome the tendency to close.

(*e*) If a flap of mucous membrane is to be turned into the antrum to cover its floor, its anterior and posterior boundaries should be incised

with a right-angle knife. The upper boundary of the flap is made when the inferior turbinate is removed (Fig. 164). The mucoperiosteal flap should be separated from the bone with a small periosteal elevator. Having separated the flap, the saw is introduced and the button of bone removed as described in the preceding paragraph, after which the flap is turned on to the floor of the antrum, which has been previously curetted. The flap hastens the process of regeneration and epidermization.

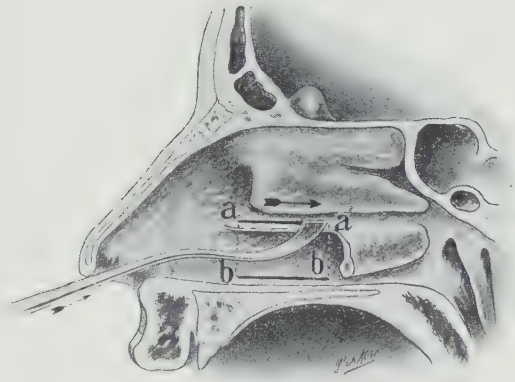
(*f*) The first dressing consists of iodoform gauze loosely packed in the maxillary sinus. It should be removed in from twenty-four to forty-eight hours.

(*g*) In the after-treatment gauze dressings should not be used. The cavity should be left open for drainage and ventilation. Every time the patient blows his nose he blows through the antrum. The case should be watched, and if exuberant granulations form, they should be promptly reduced by the application of dehydrated chromic acid crystals or with some other caustic.

FIG. 166

Corwin's antrum chisels.

FIG. 167

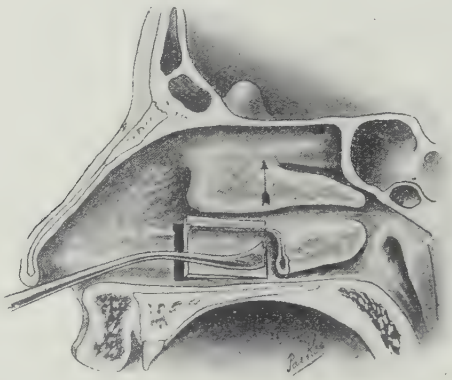


Corwin's operation upon the antrum: *a*, *a*, chisel making upper horizontal cut; *b*, *b*, lower horizontal cut.

Corwin's Operation.—Corwin's chisels (Fig. 166) are admirable instruments for removing the wall. The projecting points enable the operator to engage them at an acute angle in the body wall. Chisels without

these points are not easily engaged, as they would glide over the surface of the mucous membrane (Figs. 167 and 168).

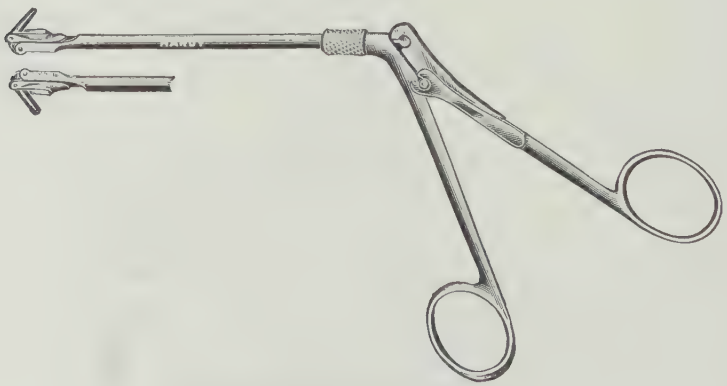
FIG. 168



Corwin's operation, second step, showing the chisel making the posterior perpendicular incision, the anterior one being already made.

Ostrum's forward cutting forceps (Fig. 169) may be used after puncturing the naso-antral wall at its posterior portion. It possesses the advantage of the forward cut, a point of no inconsiderable importance

FIG. 169



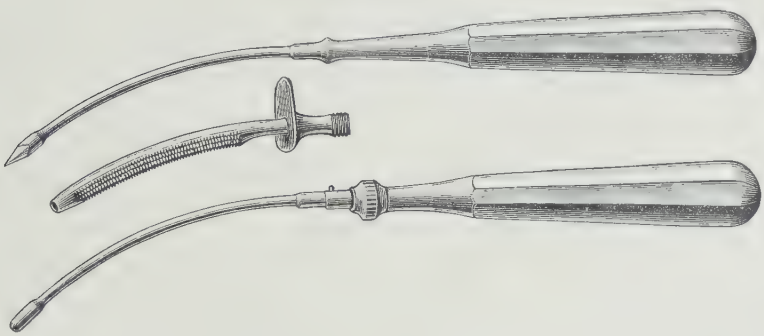
Ostrum's forward cutting antrum punch.

in view of the fact that the anterior angle of the antrum is usually the seat of the greatest morbid lesion. Hajek's sphenoidal forceps may also be used for this purpose.

Wells' combination antrum perforator and rasp file (Fig. 170) answers admirably for the purpose of making an opening in the naso-antral wall. After perforating the wall the sharp obturator is removed and the rasp is used to remove the remaining portion of the wall, which it does com-

pletely. The fragments of mucous membrane which remain are removed with sharp, biting forceps.

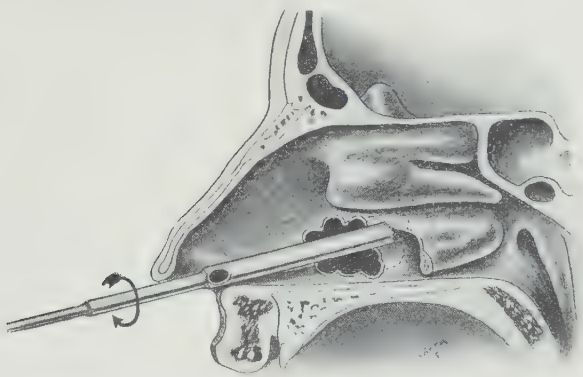
Fig. 170



Wells' trocar cannula rasp for removing the naso-antral wall.

Bishop's trephine (Fig. 171), the Nobel-Cordes forceps (Fig. 172) and Stein's hand gouge or chisel (Fig. 173) are also admirable instruments for removing the naso-antral wall.

Fig. 171



The removal of the naso-antral wall with a trephine.

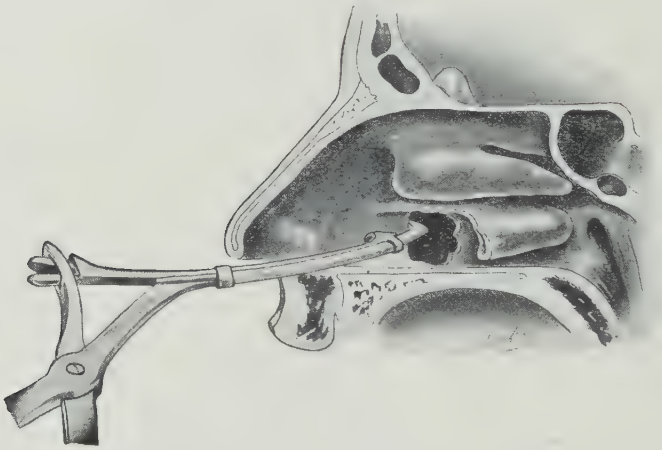
With Stein's gouge two cuts are made: one beginning just posterior to the anterior attachment of the inferior turbinate and extending above its attachment to the posterior wall of the antrum, the other from the same point and extending backward along the floor of the nose to the posterior wall of the antrum. The two incisions thus make a large tongue flap, including the anterior half or two-thirds of the inferior turbinate. This is then removed with heavy forceps. It my hands this method of operating leaves the largest possible opening in the naso-

antral wall. The only objection to it is that by it too much of the inferior turbinate is removed.

Extranasal Operations.—(1) Alveolar; (2) Kuster; (3) Caldwell-Luc; (4) Denker.

1. **The Alveolar or Cooper Operation.**—The alveolar operation was for a long time a popular procedure. Tilley, of London, reports that of 300 cases of antral disease seen during ten years, only one had sound teeth, and that of 27 cases drained by the alveolar route, 15 were obliged to use the tube and irrigation for from six months to ten years. Of these, 5 afterward elected the radical operation, which was followed by

FIG. 172



Removing the naso-antral wall with the Nobel-Cordes forceps.

FIG. 173

Stein's antrum chisel or gouge.

complete cure. Of 37 cases operated on by the radical method, 34 were successful. He also says that the alveolar route is indicated in recent cases (of a few months' standing) and in chronic cases as a preliminary measure.

Of the alveolar methods, the removal of a carious tooth, usually the second bicuspid or the first or second molar, is attended with the most happy results. It is obvious, however, that this method is only applicable when there is positive evidence that the tooth is diseased beyond hope of repair. The conditions are rare, indeed, that justify the removal of a tooth that could be successfully treated by a dentist. Even should

it be admitted that more perfect drainage can be obtained by the removal of a tooth, there are still other methods of establishing good drainage which do not require the interference with an important physiological organ, or other essential structure of the head. Drainage by the removal of a tooth should, therefore, be limited to those cases in which a competent dentist states that the tooth cannot be saved, or it can be demonstrated that there is a carious fistula extending from it to the antral cavity. In such cases the tooth may be removed, and the opening thus made enlarged and its walls rendered smooth. Daily irrigations with warm boric acid solution may be used until the discharge ceases. The alveolar opening should be closed with a strip of gauze, saturated with the compound tincture of benzoin, until healing occurs, or with a tube made for the purpose.

2. **The Kuster Operation.**—This operation has been in much favor, as the interior of the antrum of Highmore is thereby exposed, permitting inspection and curettement of its cavity. The operation consists of the removal of the anterior wall of the antrum, as shown in the Caldwell-Luc operation. The opening is usually limited to the area of thin bone of the canine fossa, and should be large enough to admit the introduction of the index finger. With the head mirror, light is reflected into the cavity and its walls examined. The portion of the cavity which cannot be inspected should be thoroughly explored with a curved probe.

If necrotic areas and granulation tissue are found, they should be removed by thorough curettement. The preliminary step of the operation consists in the elevation of the upper lip and an incision at the labiogingival junction (Fig. 174). The incision is carried through the periosteum, and should be one and one-half inches in length. The periosteum is then dissected upward over the canine fossa and the upper lip pulled toward the eye with a retractor, after which the anterior wall should be removed with a chisel and rongeur bone forceps. The cavity should then be explored with a probe and the diseased mucous membrane and necrotic bone removed with the curette. If the antrum is divided by septa, they should be broken down to convert it into one large cavity.

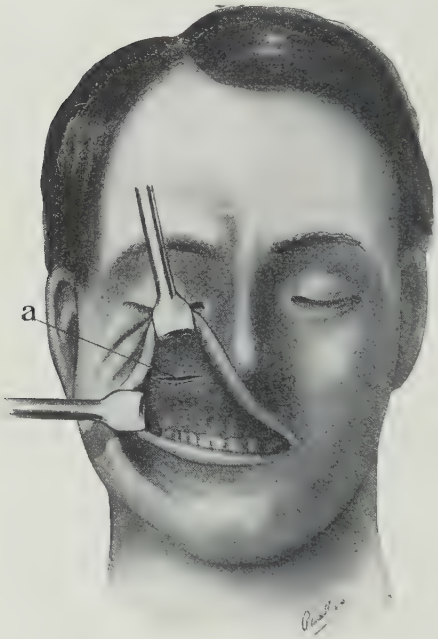
Having thoroughly removed the morbid tissue, the sinus should be loosely packed with gauze saturated with the compound tincture of benzoin. The end of the gauze should protrude through the labiogingival incision to prevent closure of the wound. If there is marked suppuration the cavity should be irrigated daily and a wick of gauze introduced to promote drainage. When complete healing has taken place the dressings are discontinued and the labiogingival opening allowed to close. This operation is not as good as the removal of the naso-antral wall, the Caldwell-Luc and the Denker operations.

3. **The Caldwell-Luc Operation.**—This operation is, in most cases, preferable to the Kuster operation. By it the antrum is exposed as in the Kuster operation, and a large opening made through the naso-antral wall. The opening may be made with forceps, Vail's saw, Corwin's chisels, or Myles' barbed cannulas through the nasal orifice. Preliminary to this, however, the anterior two-thirds of the inferior turbinal should

be removed. In making the naso-antral opening shown in Fig. 175, care should be exercised to avoid injuring the lacrymal canal which opens beneath and near the anterior end of the inferior turbinated body and passes forward and upward to the inner canthus of the eye (Fig. 177, 1).

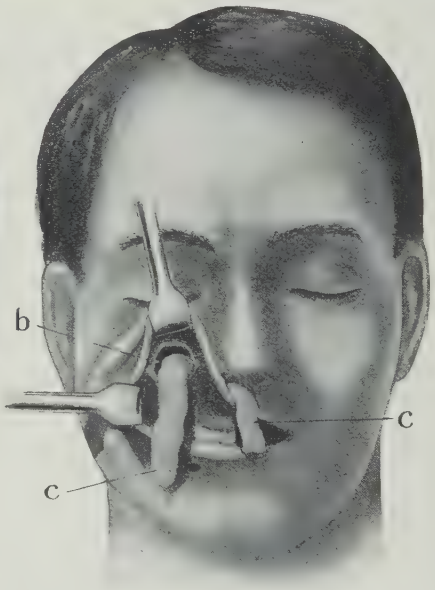
Having completed the removal of the canine and naso-antral walls, and having removed all diseased tissue from the antrum, the cavity should be lightly packed with a strip of gauze, the end of which is brought out through the nose. The labiogingival incision should be sutured (Fig. 176) and allowed to heal by first intention. After the first dressing is

FIG. 174



The labiogingival incision in the Kuster and Caldwell-Luc operations.

FIG. 175



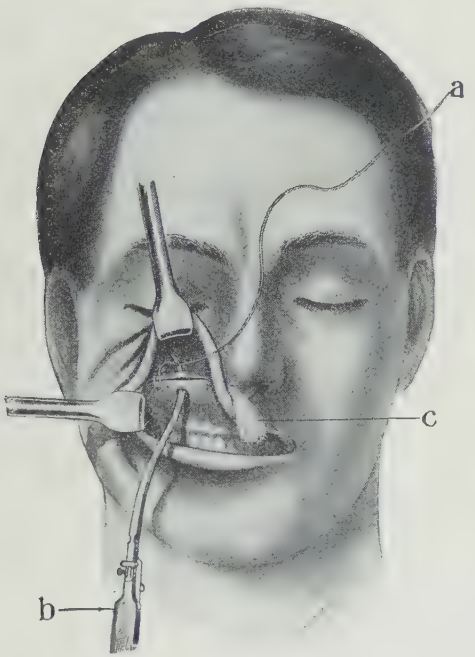
Applying the dressing after the Caldwell-Luc operation: *a*, the anterior or canine wall removed; *c*, *c*, the gauze wick in the antrum and extending through the naso-antral opening into the nasal chamber.

removed it is usually unnecessary to repack the antrum, drainage being very successfully accomplished through the naso-antral wound. At the end of the second day the gauze dressing should be removed through the nose. The secretions may be removed by forcibly blowing the nose and by irrigation.

It has been claimed that it is unnecessary to do either the Kuster or the Caldwell-Luc operation, the simple opening through the naso-antral wall being quite sufficient. That the naso-antral opening is sufficient

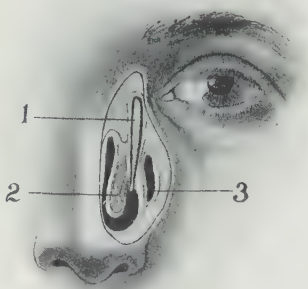
in a number of cases is true. In other cases, in which a pronounced degeneration of the mucous membrane and caries of the bony walls of the antrum are present, it is necessary to do the Kuster operation first, and to explore the antrum by ocular inspection and curettement, a procedure which cannot be successfully done through the nose. The Caldwell-Luc operation should, therefore, be elected in those cases in which there is pronounced suppuration with granulation tissue or polypi in the middle meatus of the nose. If these procedures are properly carried out and the suppuration continues, it is probable that the

FIG. 176



Closing the labiogingival incision in the Caldwell-Luc operation: *a*, the suture; *b*, the Revidan needle.

FIG. 177



Showing the relation of the ductus lacrymalis to the inferior turbinated body: 1, the ductus lacrymalis; 2, the inferior turbinated body; 3, the maxillary sinus. (After Bardeleben.)

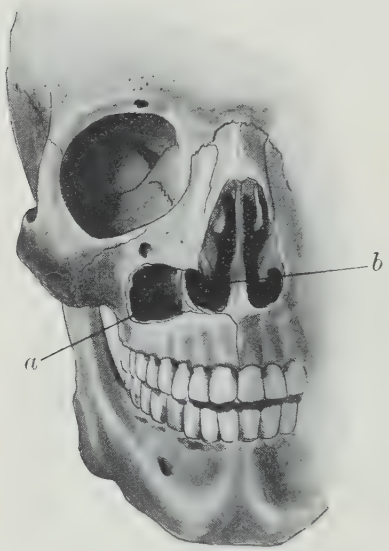
ethmoidal and possibly the frontal sinuses are also involved, and that some of the secretions from them drain into the antrum. In that event proper attention should be given to the other sinuses. A skiagraph would prevent this mistake being made.

4. **The Denker Operation.**—*Indications.*—This operation is indicated in obstinate inflammatory disease of the maxillary sinus, which does not yield to either the intranasal or to the Caldwell-Luc operation. In such a case the mucous membrane of the sinus may be very edematous and the seat of extensive granulations.

The anterior angle of the sinus adjacent to the nose is often inacces-

sible to the curette, either through the nasal or the canine fossa wound, hence the failure of the intranasal and the Caldwell-Luc operations. As the edematous membrane and the granulations must be thoroughly removed to effect a cure, an operation should be adopted that will thoroughly expose the entire cavity to curettement. The Denker operation does it, and it accordingly has a place in the treatment of selected obstinate cases.

Fig. 178



The Denker antrum operation: *a*, the area of bone removed in the Kuster and the Caldwell-Luc operations. In the Denker operation additional bone is removed from *b* to the pyriform aperture.

Technique:—(*a*) A general anesthetic should be given.

(*b*) The patient should be placed in Rose's position, with the head hanging over the end of the table.

(*c*) Postnasal tampons should be introduced to keep the blood from the throat and trachea.

(*d*) The labiogingival incision should be made as in the Caldwell-Luc operation, but should extend to the median line.

(*e*) Elevate the soft tissues and periosteum over the canine fossa.

(*f*) Remove the anterior wall (canine fossa) of the maxillary sinus as in the Kuster and Caldwell-Luc operations, and then remove the bridge of bone between the canine fossa and the lower portion of the pyriform opening of the nose, as shown in Fig. 178. By thus extending the bony wound the anterior angle of the sinus is exposed to operative interference.

(*g*) Through the opening thus made remove the edematous membrane and granulation tissue.

(*h*) Elevate the mucoperiosteum of the inferior meatus of the nose, and of the inferior turbinated body, with a small flat elevator so curved as to adapt it to the anatomical configuration of the part.

(*i*) Incise the mucoperiosteum thus elevated and convert it into a rectangular flap to be turned outward on the floor of the sinus.

(*j*) Remove the bony wall and the anterior portion of the denuded inferior turbinated bone with bone-cutting forceps, the mucoperiosteal flap being turned into the nasal chamber to prevent injuring it with the bone forceps. The opening through the naso-antral wall should be quite large, as in the Caldwell-Luc operation. Otherwise it will soon become closed and defeat the purpose of the operation.

(*k*) Turn the mucoperiosteal flap on to the floor of the sinus and

hold it in position for twenty-four to forty-eight hours with a bismuth gauze dressing.

(*l*) The after-treatment, as in the Caldwell-Luc operation, consists in watching the case and reducing exuberant granulations with caustics as soon as they appear.

5. **Canfield-Ballenger Antrum Operation.**—This is a radical, yet conservative, operation upon the maxillary sinus, wherein the inferior turbinated body is preserved intact. The operative technique upon the maxillary sinus has undergone so many changes during recent years that it is difficult to ascertain to whom the various points of technique are due. I shall not endeavor to review the development of antral operations, but shall limit my credit to those authors whose technique or principles are somewhat allied to those in this operation.

In general terms this operation consists in the removal of that portion of the naso-antral wall lying between the attachment of the inferior turbinated body and the floor of the nose, plus that portion lying anteriorly to the anterior end of the inferior turbinated body (Fig. 183, *c*). This is an elaboration of the old and well-known Mikulicz operation, in which but a small portion of the wall was removed. As a small opening soon closes by granulations extending from its borders, it is only suited to the relief of acute suppuration of the antrum. In order to adapt the operation to chronic suppurative processes, various operators have modified the operative technique, so as to remove a larger portion of the naso-antral wall. Most of them have sacrificed the anterior half or two-thirds of the inferior turbinal in order to render the naso-antral wall accessible to instrumentation. While this technique gave the desired access to the antrum by the nasal route, it possessed two vital defects, namely: (*a*) The inferior turbinal, a vital functionating respiratory organ, was largely destroyed, and (*b*) it failed to afford access to the anterior angle of the antrum, a region often most prominently affected by the disease. Hence, in those cases in which the whole mucous membrane of the antrum had undergone polypoid degeneration, or was the seat of edematous granulations, and required curettement or other treatment, this method of operation was inadequate.

The Caldwell-Luc operation, which consists of the removal of a portion of the anterior wall of the antrum, and a portion of the naso-antral wall and inferior turbinal, was for many years the most radical procedure in vogue for the cure of chronic maxillary sinuitis. Radical as it was, it still failed to give adequate access to the anterior angle of the antrum, and the inferior turbinal was sacrificed. Denker's operation was more satisfactory, as by it the entire anterior wall of the antrum was removed, thus exposing the anterior angle of the antrum to curettage. The operation is, however, objectionable in two respects, namely: (*a*) The major portion of the inferior turbinated body is destroyed and the approach to the canine fossa (anterior wall) is *via* a labiogingival incision, thus necessitating the use of a general anesthetic. These objections hold equally well against the Caldwell-Luc operation.

Hirsch, of Vienna, overcame one of the objections to the older forms of

operation by preserving the inferior turbinated body. He does an intranasal operation under cocaine anesthesia. He resects the anterior half of the inferior turbinated body at its attachment to the naso-antral wall and lifts the resected portion upward in the nasal chamber by means of a thread in the hands of an assistant. With the naso-antral wall thus exposed, he proceeds to resect it from the floor of the nose to the line of attachment of the inferior turbinated body above. He does not, however, remove that portion of the naso-antral wall anterior to the anterior end of the inferior turbinated body, hence the anterior angle of the antrum is not exposed. Having removed a portion of the naso-antral wall he replaces the inferior turbinated body and retains it in position with a single stitch at its anterior end.

Canfield, of Ann Arbor, first conceived the idea of entering the anterior angle of the antrum *via* the anterior naris under local anesthesia. His operation is in most essentials like the Denker; that is, he removes the anterior wall of the antrum, and after elevating the mucous membrane on the median side to the attachments of the inferior turbinal, resects the naso-antral wall, leaving the elevated mucous membrane, which he uses later as a covering for the floor of the antrum. He also elevates the mucous membrane on the outer aspect of the inferior turbinal to increase the size of the flap, which is to be reflected on to the antral floor. The only objection to this operation is the destruction of the anterior half of the inferior turbinated body. Many others have devised modifications of the foregoing operations, but none have succeeded in overcoming all the objections involved in the technique.

I shall endeavor in the following paragraphs to direct attention to a method of operating which is at once radical enough to meet the requirements of the most severe chronic involvement, and conservative enough to satisfy the most critical, in that it preserves the inferior turbinal intact at all times during and after the operation. By it the anterior wall of the antrum may be removed as in the Denker operation, the anterior angle of the antrum and all other portions of the antral walls exposed to inspection, curettement, and other treatment without impairing the integrity of the inferior turbinated body. The operation is a slight modification of the Canfield operation as originally described by him.

Technique.—*Anesthesia.*—(*a*) Induce anesthesia of the nasal mucous membrane by the local application of cocaine or any other drug preferred; (*b*) induce anesthesia of the vestibular skin of the naris by the injection of Schleich's solution. This solution should also be injected beneath the periosteum of the canine fossa *via* the vestibule of the nose.

Incision.—Distend the wing of the nose with a nasal speculum as shown in Fig. 179, *a*, to bring the anterior angle of the naso-antral wall into prominence. Then with a small scalpel make an incision the whole length of the exposed portion of the noso-antral angle (margin of the pyriform aperture (Fig. 180, *b*). Then elevate the membrane, including the periosteum over the canine fossa (Fig. 181).

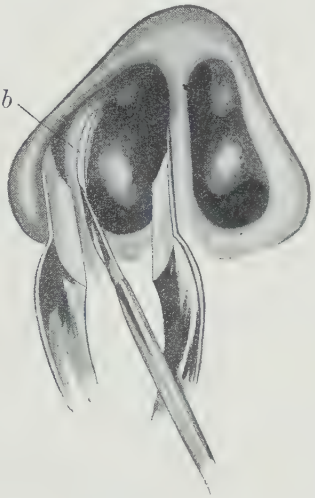
Opening the Naso-antral Angle.—The antrum should be opened *via* the naso-antral angle (margopyriformis) with rongeur bone forceps, as

shown in Fig. 181, *c*, or with a gouge and mallet. In some subjects the bone at this angle is dense, requiring considerable force to bite through it, while in others it is extremely thin and easily removed. While the

FIG. 179

Canfield-Ballenger operation: *a*, the margin of the pyriform aperture, the point of incision for the Canfield-Ballenger antrum operation.

FIG. 180

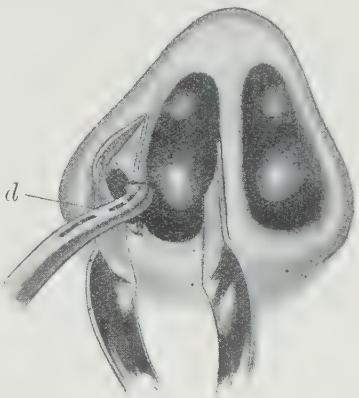


Canfield-Ballenger operation: *b*, the incision.

FIG. 181

Canfield-Ballenger operation: *c*, the naso-antral angle removed, thereby exposing the cavity of the antrum.

FIG. 182

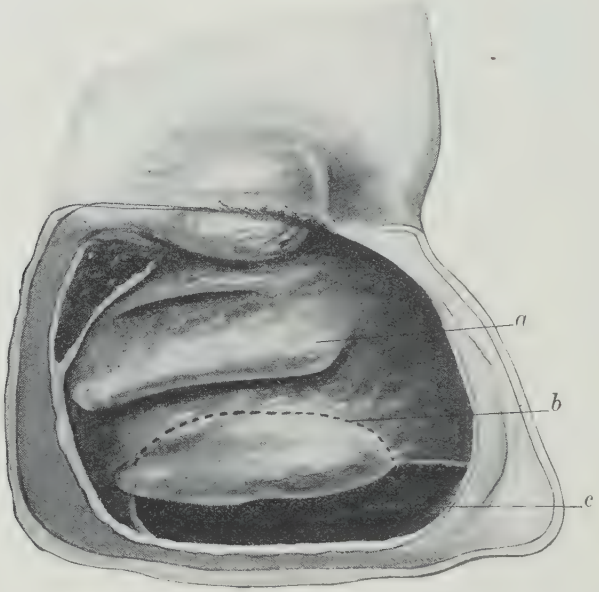


Canfield-Ballenger operation: *d*, the naso-antral wall being severed with the Wagener forceps.

incision extends higher than the attachment of the inferior turbinal (to allow retraction) the bone at the angles should only be removed below the line of attachment of the inferior turbinated body. In removing the

bone constituting the canine fossa, it is usually only necessary to remove enough to admit of the introduction of the Wagener antrum forceps as shown in Fig. 182, *d*. If, however, after making the opening through the naso-antral angle it is determined that the whole of the mucous membrane is not accessible to the curette, as much of the canine wall may be removed as will fully expose it.

FIG. 183



Interior view of the Canfield-Ballenger antrum operation: *a*, middle turbinal; *b*, line of attachment of the inferior turbinal, which is left intact; *c*, the naso-antral wall removed, extending from the floor of the nose to the attachment of the inferior turbinal and from the anterior to the posterior limits of the antrum.

Removal of the Naso-antral Wall.—The biting jaws of the Wagener forceps (Fig. 182) are placed astride the naso-antral wall and the wall bitten away from the attachment of the inferior turbinated body down to the floor (Fig. 183). This makes an opening about $1\frac{1}{2}$ by $\frac{5}{8}$ inch in size. An opening of this size will never close by granulation. It was formerly thought necessary to remove the anterior half of the inferior turbinated body to expose the naso-antral wall to surgical interference. By this method it is rendered unnecessary, hence the inferior turbinal is unmolested and is preserved and continues to perform its respiratory functions.

IMMEDIATE AND AFTER-TREATMENT.—If the mucous membrane of the antrum is edematous and has not undergone polypoid or granulation degeneration it is not necessary to curette it away (Myles). When such a pathological condition is present, packing the antrum with gauze moistened with the compound tincture of benzoin daily for three or four days

will relieve the edema, and the mucous membrane will resume its normal structure and function. When, however, the mucous membrane is converted into large granulation masses or polypi, it should be thoroughly removed with a sharp curette, thereby denuding the bony walls. If this is done a new mucous membrane will not form, but the walls will become covered with thick fibrous tissue, which partially obliterates the antral cavity. Such tissue does not develop columnar epithelium, but continues to secrete a semipurulent fluid. For this reason, curettage should be avoided unless the pathological condition warrants it.

After three or four days the gauze dressings should be discontinued. The cavity may then be swabbed or sprayed daily with a 10 per cent. solution of ichthyol to stimulate local hyperemia and leukocytosis (raise the resistance; raise the opsonic index), thereby hastening the reparative process.

In conclusion, I wish to say that this method of operating is (*a*) radical, inasmuch as it fully exposes the cavity of the antrum to inspection and treatment; (*b*) it is conservative, as it is attended by the least possible destruction of physiological structures, particularly the inferior turbinal, which is neither temporarily nor permanently resected; (*c*) furthermore, the operation may be done under local anesthesia, whereas other operations equally radical (and more destructive) must be done under general anesthesia; (*d*) the time required for this operation is much less than that for other radical operations.

PARTIAL REMOVAL OF THE ETHMOIDAL CELLS

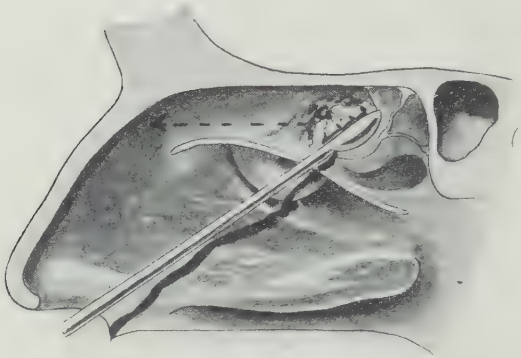
In some cases a single ethmoidal cell may be the seat of infection and inflammation, and it alone may require surgical interference. The bulla ethmoidalis is sometimes affected while all the other cells are apparently healthy. Less frequently one of the other ethmoidal cells is involved, or the anterior cells may be the seat of infection while the posterior cells are free from it, or the posterior cells may be affected and the anterior cells be normal.

When the location of the infection has been determined, the middle turbinated body (middle concha), or a portion of it, may be removed and the exposed wall of the diseased cells broken down with a curette or a Grünwald biting forceps. The cells thus opened may close by granulation in the process of repair and thus necessitate repeated curettements before a cure is established.

If after repeated attempts a cure is not effected, it may become necessary to perform a more complete operation.

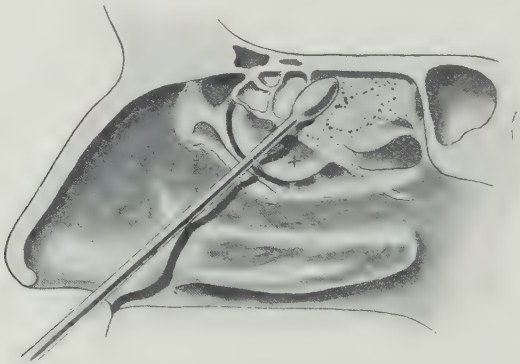
Turbinectomy with the Author's Knife.—Inasmuch as the partial or complete removal of the middle turbinated body is frequently necessary to relieve muscular asthenopia (lack of balance of the extra-ocular or intra-ocular muscles), and to establish drainage and ventilation of the

FIG. 184



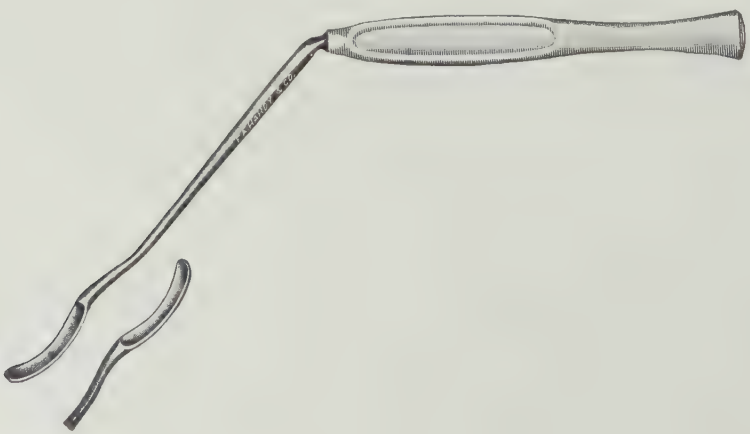
Curettage of the ethmoidal cells after the removal of the middle turbinated body. The cutting edge of the curette is directed upward and removes the cells from the cranial plate as far forward as the dotted line.

FIG. 185



Curettage of the ethmoidal sinuses. Second step. The curette is turned outward against the orbital plate and breaks down the intercellular walls of the ethmoid cells, including the bulla ethmoidalis x and the line of attachment of the middle turbinated body.

FIG. 186



The author's right and left middle turbinal knives.

nasal accessory sinuses, I have endeavored to devise some simple means to accomplish it. The turbinotome (Fig. 186), herewith presented, in a measure solves the problem.

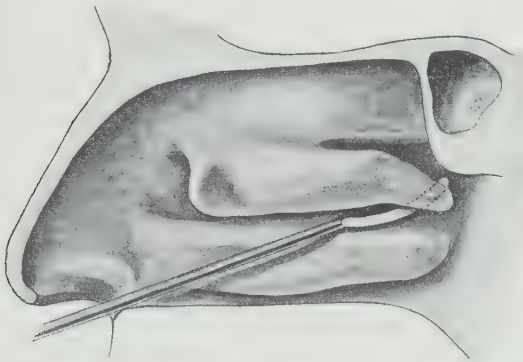
Technique of Turbinectomy.—(*a*) Cocaine anesthesia.

(*b*) Introduce the curved blade of the knife beneath the middle turbinate at the posterior extremity of the turbinated body (Fig. 187).

(*c*) Then draw it forward along the line of attachment to the anterior end of the middle turbinate, thus removing it in its entirety (Fig. 188).

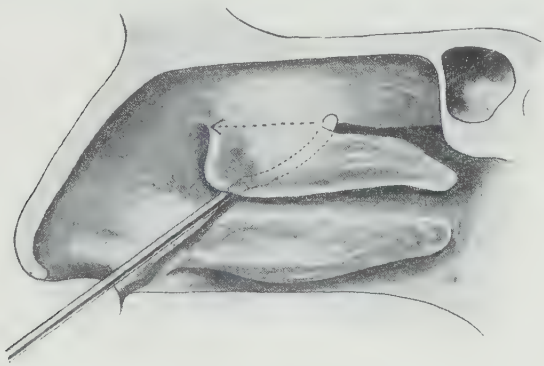
(*d*) Remove the severed turbinate with dressing forceps.

FIG. 187



The first step of the removal of the middle turbinate with the author's knife.

FIG. 188



The removal of the middle turbinate with the author's knife.

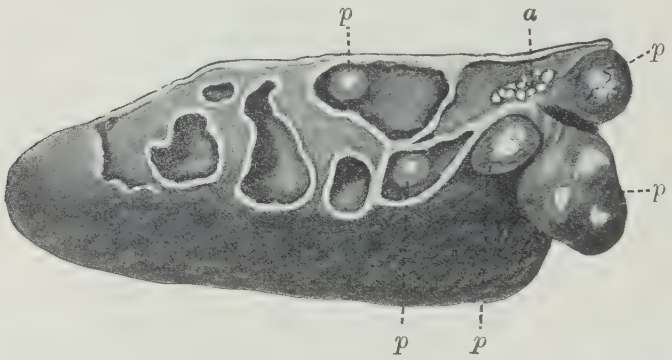
(*e*) As the anterior and posterior ethmoidal arteries supply the middle turbinate, hemorrhage may be free and persistent. If the patient is in a hospital, no dressing other than a dusting powder of bismuth or bismuth-iodine need be applied. If, however, the patient is at home, and is not easily accessible to the operating surgeon or his assistant, the space between the line of attachment of the turbinate and the septum should

be firmly packed with a strip of sterile gauze dusted with bismuth. This may be left in position for twenty-four hours. The nasal chamber should subsequently be kept free from secretions by frequent irrigations with sterile normal salt solution or by packing the nose lightly with a 10 per cent. aqueous solution of ichthyol, which should be removed after twenty or thirty minutes.

Meningitis has occasionally occurred after turbinotomy, probably on account of the tampon introduced.

The Author's Method of Removing the Ethmoidal Cells and Middle Turbinal En Masse.—The operation for the complete exenteration of the ethmoidal cells *en masse* was devised by the author five years ago for the purpose of obtaining specimens for examination. He has long believed that a better understanding of the local pathology can be had where the diseased conditions are thus exposed than where the tissues are removed piecemeal or with a curette. He holds, also, that while

FIG. 189



Lateral view of the middle turbinate and ethmoidal cells removed *en masse* by the author's operation: *p*, *p*, *p*, *p*, *p*, polypi; *a*, beginning polypoid degeneration.

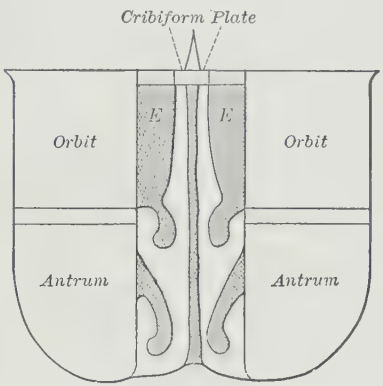
postmortem observations are valuable and instructive, those made upon specimens removed *en masse* from living subjects are much more so. With these motives in mind, he has endeavored to obtain material upon which to base conclusions concerning *sinuitis complicated with polypoid growths* in the ethmoid region.

A Specimen.—The specimen shown in Fig. 189 consists of the right middle turbinated body, five posterior ethmoidal cells, the bulla ethmoidalis, and five polypi. Three of the polypi grew from beneath the anterior end of the middle turbinated body, above the hiatus semilunaris, just anterior to the upper anterior border of the bulla ethmoidalis. The other and smaller polypi were within the ethmoid cells. The fact that some of the polypi were concealed within the posterior ethmoid cells, illustrates the futility of removing the visible tumors only, and explains why the removal of the exposed growths is so frequently followed by the appearance of others in the same or in a closely related region.

The Author's Operation.—The general method of procedure is based upon the anatomical observation that the ethmoidal cells have but three planes of attachment (Fig. 190), namely: (*a*) To the anterior wall of the sphenoid bone, (*b*) to the cranial plate, and (*c*) to the outer or orbital wall of the nose. If, therefore, these three planes of attachment are incised, a large portion of the lateral half of the ethmoid body (including the posterior ethmoidal and one or more of the anterior ethmoidal cells, and the middle turbinated body) is detached within the nasal chambers from which it may be readily removed.

The instrumentarium (Fig. 191) required for this operation consists of one instrument, supplemented by two others, which are only occasionally required. The important one consists of a short blade set at a right angle to a longer blade which is parallel with the shank of the instrument. The short blade makes the incision along the anterior wall of the sphenoid, and is then drawn forward and makes the incision along the cranial plate; when instrument is drawn forward the long blade makes the incision along the orbital wall and thus completes the excision of the ethmoid cells and middle turbinated body.

FIG. 190



Scheme showing the chief attachments of the ethmoidal cells (*E*, *E*) to the cranial plate of the frontal above and to the inner orbital walls on the outer aspect. It is obvious that if these planes of attachment are severed that the ethmoidal cells and the middle turbinates will be entirely detached.

Technique.—(1) Anesthesia is induced by massage of the mucous membrane of the middle and superior meatuses and the corresponding portion of the septum with a small cotton-wound applicator, the cotton being slightly moistened and dipped in powdered cocaine. The applications should be made at intervals of from five to ten minutes to the areas previously named until complete anesthesia is induced. If preferred, the operation may be done under general anesthesia.

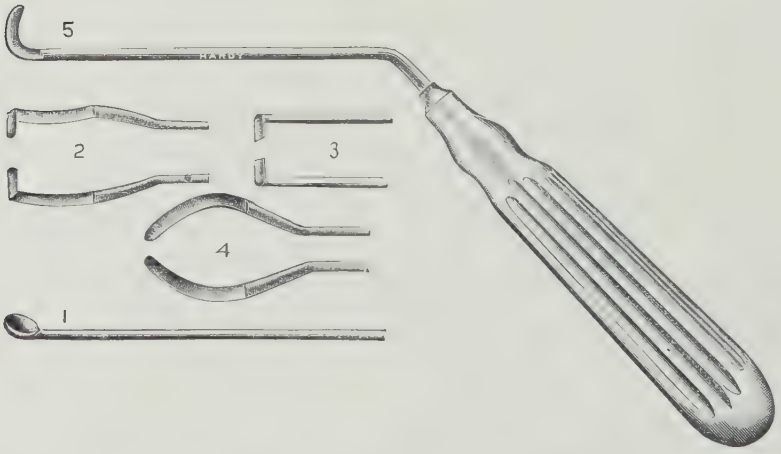
(2) The exenteration is accomplished by the following procedures: (*a*) Introduce the author's ethmoid knife (Fig. 191) into the middle meatus, with the short blade turned upward until it impinges against the lower portion of the anterior wall of the sphenoid bone, or until it engages the posterior end of the middle turbinated body (Fig. 193). During this procedure the handle of the instrument is turned horizontally across the opposite side of the face (Fig. 192, position *a*). The short blade is then forced outward into the tissues in front of the sphenoid.

This procedure is facilitated by moving the instrument backward and forward over a distance of about one-fourth of an inch, as these movements cause the short blade to penetrate the tissues to the depth of the

orbital wall and thus cut the ethmoid cells from their attachment to the sphenoid body. These movements also engage the short blade behind the posterior end of the middle turbinated body.

(*b*) The handle of the instrument is then rotated 45 degrees, to position *b*, Fig. 195. The short blade is then forced upward to the junction of the anterior wall of the sphenoid with the cranial plate, care being taken to have the long blade pass between the middle turbinated body and the outer wall of the nose. When the operator is assured that the blades of the knife are in their respective positions, he should work them upward parallel with the anterior wall of the sphenoid until the cranial plate is reached. The short right-angle blade should be forced upward in front of the anterior wall of the sphenoid until it strikes against the cranial plate, the long perpendicular blade resting against the orbital wall of the nose. The blades are not drawn forward as in

FIG. 191



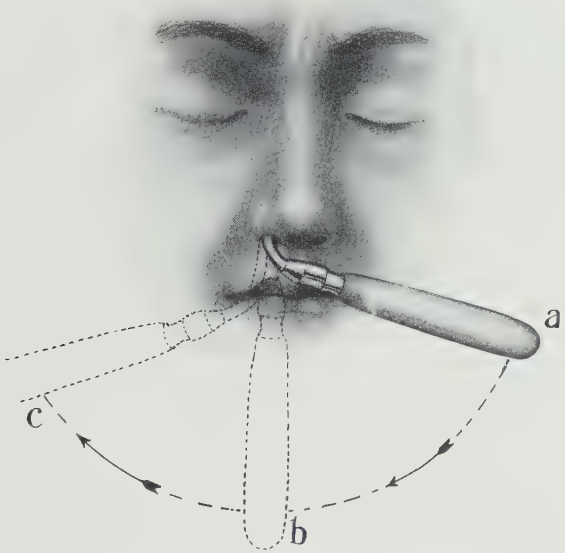
The author's new ethmoid knives and curette.

making a clean cut, but are wiggled or rotated slightly in their respective axes. This is done in order to fracture the cell walls in front of the blades, which then readily cut the mucous membrane. The instrument is thus brought forward to the anterior attachment of the middle turbinated body (Figs. 193, 194, 195).

(*c*) As the nasal chamber is quite narrow in its anterior portion, the handle of the instrument should be rotated another 45 degrees, to position *c* (Figs. 192 to 195). This turns the short right-angle blade downward into the nasal chamber and away from the septum. The knife should then be drawn forward and downward to complete the severance of the tissues. This being accomplished, the instrument is withdrawn through the vestibule of the nose. This movement of the instrument usually delivers the severed ethmoid mass from the nose; otherwise, it should be gently seized with forceps and withdrawn.

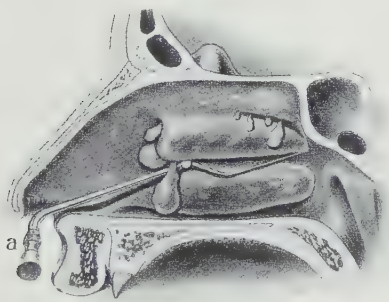
If it is found that the specimen is still attached to the nasal walls by some fibers, the blunt hook knife (Fig. 191) should be introduced between the specimen and the outer wall of the nose and the attachments severed with it.

FIG. 192



Showing the three positions (*a*, *b*, *c*) of the ethmoid knife, in the successive steps of the author's exenteration of the middle turbinate and ethmoid cells *en masse*.

FIG. 193



The first step of the author's exenteration of the middle turbinate and ethmoid cells and polypi *en masse*. The instrument in position *a*, Fig. 192.

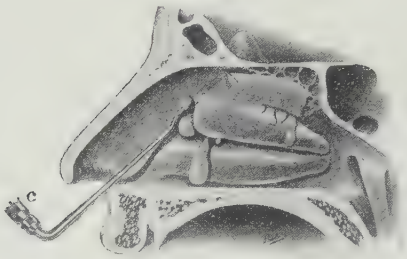
FIG. 194

The second step of the author's ethmoidal operation. The instrument in position *b*, Fig. 192.

(*d*) The blood should be mopped from the nasal chambers, and the remaining fragments of cells should be broken down with the curette. This completes the operation.

The Dressing.—If there is serious hemorrhage the upper or ethmoidal region of the nasal chamber should be packed with a one and one-half inch strip of gauze impregnated with the subnitrate of bismuth powder. The bismuth prevents decomposition and infection, and thus wards off the dangers of septic absorption. The gauze should be introduced against the anterior wall of the sphenoid, and folded and packed until the upper half of the nasal cavity is completely filled with it. Stout dressing forceps should then be introduced beneath the dressing, and the whole lifted in order to compress it into the area which has been operated on. The dressing should be removed in from one to twenty-four hours. The subsequent treatments consist in lightly packing the nose with cotton tampons saturated with a 10 per cent. aqueous solution of ichthyol or of argyrol. The applications should be repeated daily and left in place twenty minutes. This mode of treatment is more effective in removing the secretions and sterilizing the wounded surface than irrigations.

Fig. 195



The third and final step of the author's exenteration of the middle turbinate and the ethmoid cells *en masse*. The instrument in position c, Fig. 192.

Never introduce nasal tampons unless forced to do so on account of profuse hemorrhage, as they may cause infection and meningitis. Firmly packed dressings are dangerous. Personally, I rarely pack the nose, as I find severe hemorrhage rare.

The Complications.—Hemorrhage.—(*a*) Hemorrhage nearly always attends the operation, and it may either persist, or appear later as a secondary hemorrhage, though the latter is comparatively rare. When we remember that the ethmoidal region receives its blood supply from the anterior and posterior ethmoidal and the sphenopalatine arteries the possibility of a severe hemorrhage is apparent. By packing the nose as described, this complication may be controlled. A slight serosanguineous oozing may continue for twenty-four to forty-eight hours in spite of the gauze packing, though it is of no serious consequence. If the patient is operated on in a hospital and remains there for three days, it will rarely be necessary to pack the nose. The activity incident to leaving the physician's office and going home increases the blood pressure, and, as a consequence, the chances of hemorrhage are greatly increased, whereas if the patient remains quiet in a hospital the danger is greatly diminished.

(*b*) *Emphysema of the Orbital Tissues.*—The lamina papyracea of the orbital wall may be fractured in the operation, and upon blowing the nose may admit air into the cellular tissue of the orbit. This occurred twice in my practice, but in neither instance did it inconvenience the patient, as it disappeared in one or two days.

(*c*) *Orbital Infection, Cellulitis.*—It is within the range of possibility for infection of the orbital tissues to occur subsequent to an ethmoid operation, though I have never observed it in an experience embracing four hundred operations. The orbital plate while thin is very resilient in the living, and is not easily fractured.

(*d*) *Meningitis.*—Meningitis following the ethmoid operation is rare. The cribriform plate of the ethmoid and the cranial plate of the frontal bone are not easily fractured and in my experience have never been fractured. The chief point to be mentioned concerning them is that the operation should not be performed if a latent chronic meningitis is already present, as it may cause an acute exacerbation and extension which may prove fatal. The chief subjective symptom of latent meningitis is a severe headache. When this is present the operation should be postponed until it has been proved that it is not due to meningitis. If there is any doubt Quincke's spinal puncture should be made, some of the spinal fluid is withdrawn and subjected to the proper examinations. In one case of this description meningitis was demonstrated to be present.

(*e*) *Nasal Stenosis from Swelling of the Nasal Mucous Membrane.*—This complication has occurred several times in my practice and has always been due to a partially severed fragment of the middle turbinated body which has been left in the nasal chamber. This was especially true of my earlier operations, in which I had not perfected the technique in its present form. Since performing the operation as described in this section, this complication has not occurred.

I wish to say in conclusion, that the operation has given me greater satisfaction, and in properly selected cases has given better results than I have been able to obtain by any other method of operating.

Mosher's Frontal-ethmoid Operation.—This operation may be performed under either local or general anesthesia. In my experience it has proved a most excellent and satisfactory procedure, and appears to be the simplest and safest operation devised for its purpose. While it will not replace the Killian frontal sinus operation, it will very greatly reduce the necessity for that operation.

Technique.—1. Cocainize the interior of the nose or administer a general anesthetic.

2. Introduce a curette into the nasal chamber, until the cutting edge of the instrument facing the orbit is above the anterior attachment of the middle turbinate, as shown in Fig. 196. This area covers the frontonasal canal and the anterior ethmoidal cells draining into it. The bone at this point is usually very thin and easily broken down. In some cases the bone at this point is very dense, thus making it necessary to break through it more posteriorily. Having located the instrument, make gentle but firm pressure toward the orbit, and at the same time withdraw it downward and forward one-fourth to one-half inch. A few such procedures with the curette will give the result shown in Fig. 197. The anterior ethmoidal cells are thereby completely opened. By continuing the curettage in a forward and upward direction the frontonasal opening in the floor of the frontal sinus is enlarged and free

drainage of this sinus established. The frontal sinus may now be entered with a blunt pointed frontal sinus probe, as shown in Fig. 197. Indeed, in most instances, a suitably bent, cotton-wound applicator may be easily introduced.

FIG. 196

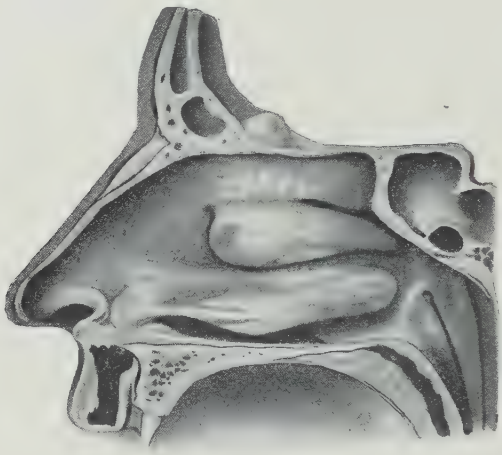
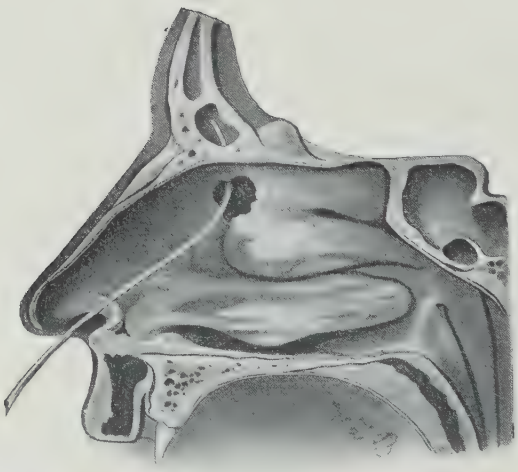


FIG. 197



3. The next step of the operation is the removal of the posterior ethmoidal cells. This is done with the same curette introduced through the opening already made, as shown in Fig. 198. The curette is introduced beneath the cranial plate and then brought downward between

the orbital and turbinal plates. This procedure is repeated several times until the anterior wall of the sphenoid is reached.

FIG. 198

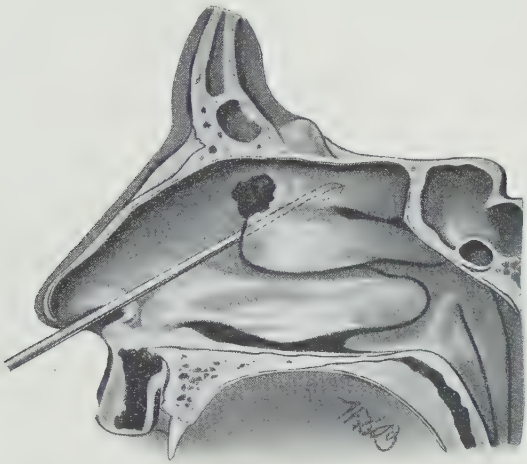
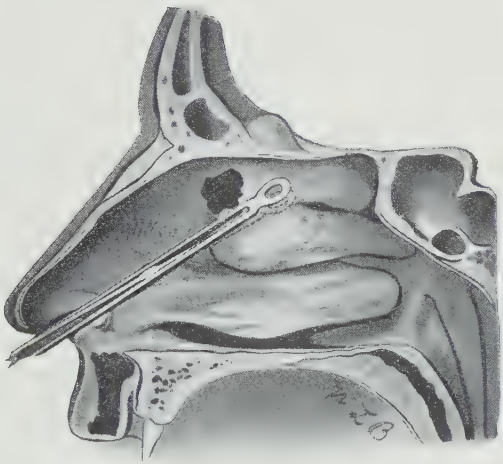


FIG. 199

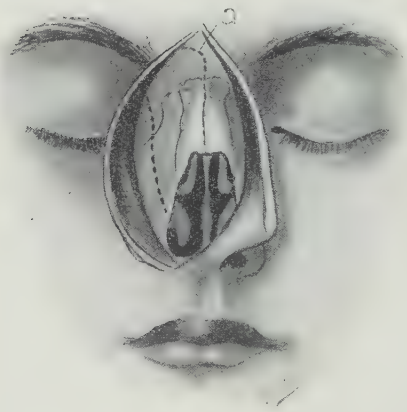


4. The turbinal plate, consisting of the superior and middle turbinate bodies, is then seized with suitable grasping forceps, and by gentle traction combined with twisting motions is detached from the cranial plate and removed from the nose.

5. The entire posterior and anterior ethmoidal regions are again examined by ocular and probe inspection, and all portions of cells remaining are removed.

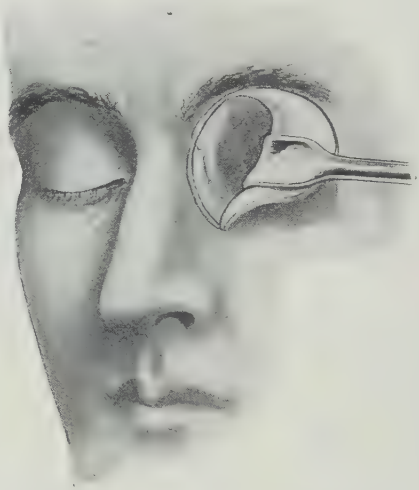
The after-treatment is the same as for any other method of operation, my own preference being packing the nose daily with pledgets of cotton saturated with a 10 per cent. ichthyol or argyrol solution. The packing should be left in place about fifteen minutes.

Fig. 200



Moure's operation upon the anterior ethmoidal cells. The dotted line *a* indicates the area of bone removed from the lateral wall of the nose to expose the cells.

Fig. 201



Exposure of the anterior ethmoidal cells through the inner wall of the orbit. This method of procedure is adapted to those cases complicated by orbital cellulitis.

EXTERNAL OPERATIONS UPON THE ETHMOID SINUSES

Moure's External Ethmoid Operation.—This operation may be performed in those cases in which extensive necrosis and polypi are present in the ethmoidal region upon both sides, as it exposes the field of operation better than any other method. It may also be used to expose large tumors in this region.

Technique.—(*a*) The operation should be performed under general anesthesia, though it may be done under local injections of Schleich's mixture combined with local cocaine anesthesia of the nasal mucous membrane.

(*b*) Insert postnasal tampons, one in either nostril, to prevent the blood entering the trachea.

(*c*) Make an incision along the ridge of the nose from a point midway between the eyebrows, extending downward to the nasal opening on the side to be operated on, at the junction of the cutaneous septum with the ala or wing of the nose.

(*d*) Elevate the soft tissues, including the periosteum, as shown in Fig. 200.

(*e*) Resect the nasal bone and the frontal processes of the maxilla, as shown in the area encircled by the dotted line (*a*) in Fig. 200.

(*f*) When the ethmoidal labyrinth has been thus exposed, the entire ethmoid region may be thoroughly exenterated with a curette.

If the disease is well advanced, that is to say, if there are polypi and granulations, every vestige of the cells should be removed. The cranial plate, the os planum (paper plate of ethmoid) or orbital wall, and the lacrymal bone which is adjacent to the anterior cells should be gently but thoroughly curetted until they are smooth. In addition to these surfaces the ethmosphenoidal wall (posterior limit of the ethmoidal cells) should also be thoroughly curetted. If all these surfaces are cleared with the curette and the anterior and posterior ethmoidal labyrinths are separated from their attachments, the cells and the middle turbinated body may be removed through the nasal wound or through the anterior naris.

(*g*) The space from which the cells have been exenterated may be packed with a strip of gauze in front of the postnasal tampon on the side operated upon, and the postnasal tampon removed from the other side.

(*h*) The skin and periosteal incision should be closed with fine silkworm sutures.

(*i*) Watch the case, and should granulations form at any point, touch them lightly with carbolic or chromic acid. Should points of suppuration be found, probing should be done, with a view to tracing them to their sources. If the cause is found to be a cell which, through error, was not removed, or which was inaccessible, as an anterior ethmoidal cell extending over the orbital cavity or a posterior ethmoidal extending to the lateral side of or behind the sphenoidal sinus, steps should be taken to maintain a patulous opening for drainage purposes. All granulations should be removed from the point of suppuration as rapidly as they appear. Persistent after-treatment as described above will often be rewarded by a cure of the case.

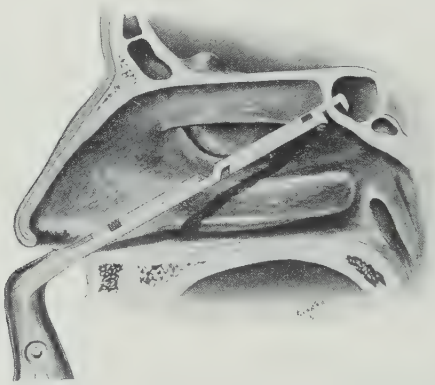
Orbito-ethmoid Operation.—(*a*) Make the Killian incision and elevate the tissues and periosteum at the inner aspect of the orbit, as shown in Fig. 201. (*b*) Remove the nasoörbital plate of bone and curette the ethmoidal cells through the opening. The orbital tissues should also be explored and the pus evacuated if present. Maintain external drainage until the discharge ceases, and allow the wound to heal by granulation from the bottom.

THE SPHENOIDAL OPERATION

The preliminary operative procedure for reaching the sphenoidal sinus consists of the complete removal of the middle turbinated body, thus exposing the ostium sphenoidale to view.

I use the Hajek or the Fletcher sphenoidal forceps because they are strong and remove the anterior wall completely. One of Hajek's forceps cuts upward and the other downward. Fletcher's forceps cuts in all directions, as its biting end is a circular disk. The disk is conical on its distal aspect to enable the operator to force it through the ostium sphenoidale without first enlarging it, as is necessary in using Hajek's forceps. Fletcher's instrument is very powerful, and as it cuts in all directions is most admirably adapted to the surgery of the sphenoidal sinus; furthermore, but one instrument is required. If the ostium sphenoidale is

FIG. 202



Removing the anterior wall of the sphenoidal sinus with the Hajek forceps. The distal blade of the forceps is introduced through the ostium sphenoidale and the bony wall removed by successive bites.

small it should first be enlarged with a curette. The upward cutting forceps should then be introduced and the upper portion of the wall removed. By turning the forceps to either side the lateral portion of the wall may be removed. Next introduce the downward cutting forceps (Fig. 202) and remove the lower portion of the wall. The wall near the floor of the sinus is quite thick, but is readily removed with Hajek's forceps. When the wall is entirely removed, the opening is often one-half by three-fourths of an inch in area, and the interior of the sinus may be inspected by reflected illumination. When the mucous membrane is normal it is pale, and by contrast with the nasal mucous membrane appears almost white. Under probe pressure, it is thin, firm, and slightly resilient. When diseased, it is more red, edematous, and thickened. In some cases the sinus is filled with granulation tissue or polypi.

When the anterior wall is removed and the mucous membrane is diseased, it should be thoroughly curetted.

The after-treatment consists of irrigations and the topical application of a 10 per cent. aqueous solution of ichthyol. As there is a marked tendency for the mucous membrane to reform over the opening in the sinus, it may be necessary to remove it from time to time to maintain ventilation and drainage. This is easily accomplished, as the middle turbinate has been previously removed and the tissue to be removed is membranous. The after-treatment may extend over many weeks.

CHAPTER XII

NASAL NEUROSES. NASAL HYDRORRHEA. CEREBROSPINAL RHINORRHEA

NEUROSES OF OLFACTION

The neuroses of olfaction are characterized by either (*a*) a perverted sense of smell (parosmia), (*b*) oversensitiveness to olfactory stimuli (hyperosmia), (*c*) a partial loss of the sense of smell (hyposmia), or (*d*) a total loss of the sense of smell (anosmia).

Parosmia.—Parosmia is characterized by a perception of imaginary odors, and may be due to pathological changes in the olfactory brain-centre. Inflammatory disease of the mucous membrane in the attic of the nose may also produce parosmia by overstimulating the nerve endings. It usually accompanies lesions of the central brain, although it occasionally occurs in hysteria, hypochondria, epilepsy, insanity, and syphilis.

Hyperosmia.—Hyperosmia is characterized by an oversensitiveness to olfactory stimuli—that is, the perception of odors is exaggerated. The most delicate perfumes or odors not ordinarily perceived are recognized even to the point of unpleasantness. In some cases the perception of odors persists after the source of the odor is removed, and in this respect the condition approaches parosmia.

It may be due to an irritation of the olfactory lobes, hysteria, neurasthenia, hypochondria, sexual disorders in women (especially at the menstrual period), and to the lowered nervous forces accompanying wasting diseases.

Hyposmia.—Hyposmia is characterized by a partial loss of smell, either from an impairment of the mucous membrane of the attic of the nose, the nerve endings, the bulb, or the brain centre. The impairment is only great enough to obtund the perception of odors without totally destroying it.

Anosmia.—Anosmia is characterized by a total loss of the sense of smell, the pathological lesion being more extensive than that found in hyposmia.

I have often seen cases in which the total loss of smell was due to a blocking of the olfactory fissure by an enlargement of the middle turbinate, which was relieved by its removal. These cases were also complicated by ethmoiditis and sphenoiditis, but the loss of the sense of smell was not due to the inflammatory disease, as the ability to perceive odors was immediately restored by the removal of the middle turbinate. If it had been due to disease of the mucous membrane, considerable

time would have elapsed before regeneration could have taken place. A cold in the head is a frequent cause of transient anosmia.

Odors reach the attic of the nose by either the anterior or the posterior nares, hence any condition of the septum or of the tissues of the outer wall of the nose which blocks the anterior or posterior nares may produce anosmia. The lesion may be in the nerve endings, as in atrophic rhinitis, in the nerve, or in the olfactory brain centre. Anosmia of intranasal origin may be unilateral or bilateral according to the location of the obstructive lesion. In such cases the sense of smell may be restored by the proper surgical procedure within the nose. If, however, the lesion is in the olfactory nerve or brain centre a cure is scarcely possible.

SENSORY, VASOMOTOR, AND REFLEX NEUROSES

Hyperesthetic Rhinitis; Hay Fever.—Hyperesthetic rhinitis, or hay fever, is characterized by annual paroxysms of sneezing accompanied by a severe and prolonged coryza and asthma.

Etiology.—The Predisposing Causes.—The predisposing causes of hyperesthetic rhinitis are constitutional, local, climatic, geographical, racial, and altitudinal.

(*a*) The *constitutional causes* are a neurotic temperament, chemical changes in the glands which secrete mucus (D. Braden Kyle), and gout and rheumatism.

The *neurotic temperament* is difficult to define, but seems to be an unstable condition of the nervous system, wherein there is either an excess or a decrease in the nervous energy. Some physicians claim that the nervous disturbance is due to a faulty metabolism whereby certain toxic substances are liberated in the blood current. Thus a gouty or a rheumatic diathesis is held to be the basic cause. It is obvious, however, that there must be a reason for the gouty or rheumatic expression. It appears impossible in the present state of our knowledge to define clearly the conditions which cause a nervous temperament. That hay fever subjects are neurotic is generally accepted. Why they are neurotic is a much mooted question, concerning which many ingenious theories have been advanced, but none of which are convincing.

(*b*) The *local causes* of hyperesthetic rhinitis are various. A perfectly healthy nasal mucous membrane on a normally placed bony framework is not often affected by hay fever. On the other hand, an apparently healthy mucous membrane on a normally placed bony framework may be affected. I have seen cases in which there was no obstructive septal deformity and no absolute occlusion of the olfactory fissure by turbinal enlargement. The only noticeable morbid lesion was a slight redness of the mucous membrane over the anterior end of the middle turbinated bone. These cases were also subject to occasional attacks of severe coryza, with copious purulent discharge. During the interim between the attacks of coryza no symptoms were complained of, but an examination of the nose showed the reddened and slightly boggy edematous con-

dition of the anterior portion of the middle turbinal. While I do not care to promulgate a new theory as to the etiology of hay fever, I have been impressed with the possible relationship of catarrhal sinuitis, particularly ethmoidal and frontal, to hay fever. In some cases the surgical treatment of the sinuitis was followed by a relief of the hay fever. It is possible that the catarrhal discharge so irritates the nasal mucous membrane as to make it susceptible to the irritation of the pollen of certain plants and grasses. The difficulties in the way of diagnosticating catarrhal sinuitis have been so great that it has usually been unrecognized. With our present knowledge its detection should be more often made. It is now possible to study the relationship existing between sinuitis and hay fever, and I have some confidence that such a relationship will be satisfactorily established. Indeed, since the publication of the foregoing statements in the third edition, I received a communication from Dr. P. M. Farrington, of Memphis, in which he refers to two cases of hay fever cured by autogenous vaccine. He used about 50,000,000 bacteria at the first injection (Case 1), and at the third injection used 100,000,000 without reaction. After the first injection there was great improvement, and after the second all symptoms, except a slight stuffiness of the nose, disappeared. The injections were given every fourth day. The second case was treated in the same manner and with the same excellent results.

The late Dr. Schadle recently called attention to the possibility of relationship between maxillary sinuitis and hay fever. Whether or not such a relation exists, we must recognize the fact that the local hyperesthesia probably has an anatomical or inflammatory origin. The hypersensitiveness does not "happen," but has a definite cause. Inasmuch as sinuitis, either catarrhal or suppurative, is often associated with hay fever, it seems plausible to conclude that the irritation attending the discharge of the secretions over the nasal mucous membrane may be the cause. The hypothesis is still further supported by the clinical fact that some cases of hay fever are cured by successful treatment of the sinuitis.

While the above hypothesis is based upon clinical observations, they are too meagre to warrant final conclusions. They are sufficient, however, to justify the closest scrutiny of the sinuses in every case of hyperesthetic rhinitis (hay fever). Such a scrutiny should include the examination of the middle turbinal, the olfactory fissure, and the septum; transillumination, and a skiagraph of the sinuses. In addition the patient should be closely questioned concerning the presence of headache (chiefly frontal), dizziness, especially upon stooping forward, and unilateral disturbances of the ocular apparatus. The ocular disturbances may include errors of refraction, ulcer of the cornea, or lesions of the retina or other portions of the optic tract, and of any other of the structures of the eyeball. The composite picture thus elicited should show conclusively either the presence or absence of an associated disease of the sinuses.

Deflection of the septum, especially in the region of the middle turbinate

or enlargement of the middle turbinate, causing contact between the two, is another local factor in hyperesthetic rhinitis.

The "sneezing area" of the nose is at the points of contact between the middle turbinate and the septum, hence the sneezing which is so characteristic of this disease. As a rule, the sneezing ceases as soon as the pressure is relieved.

Sensitive areas on the nasal mucous membrane of the septum and the outer walls of the nose, which are reddened and slightly elevated above the surface of the mucous membrane, predispose to the hyperesthetic paroxysms. Whether they are due to some concurrent inflammation of the accessory sinuses, or to some change in the sensitive nasal branches of the sphenopalatine ganglion, is not established. It is reasonable to suppose that an inflammatory disease of the nose, attended with an irritating secretion, which is characteristic of catarrhal sinuitis, might affect the filaments of the terminal sensitive nerve and render them extremely hypersensitive. The local vasomotor disturbance in the same areas would cause their elevation above the surface of the mucous membrane.

Polypi have long been considered a local predisposing cause of hay fever. As these morbid growths are often secondary expressions of sinuitis, the possibility of the causative relationship of this disease is thereby strengthened. The polypi are usually found in the region of the hiatus semilunaris, the border of the middle turbinate, or the posterior ethmoidal cells. In the latter case they protrude through the olfactory fissure into the middle meatus or are lodged above the middle turbinate in the superior meatus. It is evident that the mere removal of the polypi may not suffice to eradicate the irritation. The diseased sinuses should also receive appropriate treatment.

(*c*) The *climatic influence* upon hay fever is well recognized as being confined to the neighborhood of the forty-fifth parallel of the northern hemisphere. The territory a few degrees either north or south of this latitude is comparatively free from this disease. This is probably due to the absence of the flora, the pollen of which is the chief exciting cause. If a map of the United States were divided into four belts by lines drawn through it from east to west, the majority of the cases of hay fever would be included within the third belt from the bottom, although many cases would be found in the other belts.

(*d*) The *geographical distribution* of hay fever is instructive. It is more prevalent in the United States than in any other country; England ranks second. It is also present in Germany and France, though in less degree.

(*e*) The *racial influence* in the predisposition to hay fever is marked. It is more common in the English-speaking races of the northern hemisphere than among the French or Germans, though it is more or less prevalent among them.

(*f*) *Altitude* has considerable influence in the causation of hay fever. The disease is more prevalent in the low portions of the country than in the higher altitudes, which are comparatively free from it. The annual

pilgrimages which are made into the mountains in the northern portion of the Eastern States and into the cold, bracing atmosphere along the shores of Lake Superior and the northern shores of Lake Michigan are indicative of the benefits derived from altitudinal and climatic change.

(*g*) *Age* is an important factor in the causation of hay fever; it is most common in persons between the twentieth and fortieth years of life.

Exciting Causes.—It is generally believed that the exciting causes of hay fever or hyperesthetic rhinitis are the emanations from certain plants and animals. It was at one time thought that all cases were of vegetable origin in the haying season, hence the name. Subsequent observations have shown that the exciting cause may emanate from various plants and animals, chiefly the following: Graminaceæ, Solidago virgaurea (goldenrod), Ambrosia artemisiæfolia (rag-weed), cats, dogs, horses, and cows. The emanations from grasses and other plants, which cause the paroxysmal symptoms, is probably their pollen. In 1873 Blackley conducted a series of experiments with glycerin-covered glass plates and observed the rise and fall of the intensity of the symptoms with the increase and decrease in the quantity of pollen within a given area on the plates. From these observations he proved that the pollen of certain plants was an exciting cause of the disease. Since then many observers have reported that the emanations from animals are also exciting causes.

The season has a characteristic influence upon the occurrence of the paroxysmal attacks of hyperesthesia. This is due to the fact that there are no emanations from plants except during the time they throw off their pollen. The disease occurs most frequently in August and September and less frequently in June, when the roses are in bloom.

An analysis of the causes of hyperesthetic rhinitis resolves the etiology into three groups, as follows: (1) A constitutional or neurotic habit. (2) Local morbid lesions of the nose and accessory sinuses. (3) The pollen of certain plants and emanations from certain animals.

Pathology.—The structural changes in the affected nasal mucous membrane consist of hyperemia, edema, and (after repeated attacks) hyperplasia of the turbinated bodies. The presence of nasal polypi in a hay fever case is scarcely to be considered a pathological lesion of this disease, but rather a result of inflammation of the sinuses. The elevated hypersensitive areas are chiefly found at the terminal endings of the sensitive branches of the sphenopalatine ganglion, and are due to the increased hyperemia in these areas, while the hypersensitiveness is due to the irritation of the sensitive endings of the nerve fibers.

If the disease were a pure neurosis there would be other nervous phenomena somewhat proportional to the intense paroxysms of the hay fever, whereas if it were a true inflammatory disease there would be greater structural changes. The disease is probably a combination of a moderately severe neurosis, with local morbid changes which give rise to the local irritation of the nerve endings of the sensitive branches of the sphenopalatine ganglion, upon which, at favorable seasons of the

year, the pollen of certain plants and the emanations from certain animals lodge, and give rise to the phenomena characteristic of hyperesthetic rhinitis.

Symptoms.—The symptoms of hay fever are those of an acute coryza, as malaise, elevation of temperature, sneezing, serous discharge, headache, etc., to which are added an itching in the region of the soft palate and the median palpebral commissures (inner canthi) of the eyes, and asthma. The sneezing is paroxysmal and may be excited by slight draughts of air, bright sunlight, particles of dust, and psychical impressions, such as the consciousness of being observed by another person, or the thought of his own condition. The sneezing is accompanied by profuse lacrymation and serous nasal secretion and by suffusion of the conjunctiva. The profuse serous discharge from the nasal mucosa is followed by a contraction of the swollen mucous membrane, which brings temporary relief.

The serous secretion from the nose is acrid, and excoriates the alæ of the nose and the upper lip. (I have observed the same phenomena in some cases of inflammation of the ethmoidal cells when pus was absent.) The secretions become seromucous and in some cases purulent in character.

Intermittent and alternate stenosis of the nose is present. During the continuance of the nasal stenosis the patient suffers from the paroxysmal sneezing and asthma, and from headache, lacrymation, and diffidence. The diffidence is extreme, and the patient dreads the approach of another person, especially if he is a stranger or someone with whom he is ill at ease.

The pharynx is often dry and painful upon deglutition. The tonsils are not usually inflamed, although they may be.

Tinnitus aurium is frequently present, and is due to a swelling of the mucous membrane of the Eustachian tubes.

The appetite is impaired, and there is a corresponding loss of weight.

Prognosis. A conservative prognosis should always be given. So many methods of treatment have been promulgated, with the assurance of success, and have proved wholly inadequate, that I doubt the value of nearly all of them. Upon theoretical grounds it appears that if either one of the three major causes of the disease is removed a cure must follow. If, for instance, the local morbid lesions of the nose are overcome, the patient should be freed from the hay fever; if the neurotic habit is overcome, the hay fever should be cured; and if the patient is removed from the influence of the pollen, or is rendered immune by serums or antitoxins, he should be cured. Many a patient has been treated and operated upon with a view to the total removal of the local morbid lesions, but the hay fever paroxysms continued from year to year without abatement. Many a hay fever sufferer has been persistently treated for neurosis, and the various dyscrasias causing it, without effect upon the hay fever; and many a patient has been sent year after year to the mountains or to the northern lakes without preventing the recurrence of the paroxysms the following year. On the contrary, a few patients have been cured

permanently by recourse to one or more of the foregoing methods of treatment. The same is true of other methods; a few are cured, though many are not benefited at all. A remedy that is efficacious for one subject is totally ineffective when applied to another.

Either the existing ideas concerning the etiology or our methods of diagnosis of the local morbid lesions are wrong—probably both. Nevertheless, we can only act upon present knowledge. We must, therefore, continue to remove the local morbid lesions from the nose and accessory sinuses, for this is the most hopeful method of treatment, unless the patient is removed to a place where the pollen or other irritant peculiar to his case is absent; or we must administer a serum that is an antidote to the pollen in question. In the meantime our knowledge of the morbid processes in the nose and accessory sinuses is rapidly advancing, and it may be that we shall soon be able to cure this elusive and distressing disease.

Treatment.—The treatment may be divided into five groups, namely: (*a*) The treatment of the dyscrasias; (*b*) the removal of the local morbid processes in the nose and the accessory sinuses; (*c*) the removal of the patient from the influence of the pollen or other emanations which act as the exciting cause of the disease; (*d*) the immunization of the patient; and (*e*) the relief of acute symptoms.

Treatment of the Neuroses and Dyscrasias.—The treatment of the neuroses and dyscrasias due to modern civilization is a very difficult undertaking. We are in a domain of pathological entities the forms of which are shadowy and the definitions obscure. We are dealing with unknown quantities upon hypotheses not yet proved. Failure is the almost inevitable result. While all this is true, something may still be done to improve rheumatic and gouty diatheses and the ill-defined neurotic manifestations. The intestines and stomach can be flushed by lavage and by saline cathartics. The kidneys and skin can be made to eliminate more freely, and the hemoglobin of the blood can be raised so as to attract more oxygen. These and other processes may be stimulated or modified so that the neurotic state of the nervous system and the various constitutional disorders are in a degree improved. Indeed, the treatment should include some of these measures, although a cure may never be effected by them. According to Major Woodruff, excessive exposure to sunshine is a cause of neurasthenia, and this may in a measure account for the greater prevalence of hay fever in America.

Treatment of the Local Morbid Lesions.—(*a*) The circumscribed sensitive areas should be cauterized with a flat electrode at white heat, without the use of a local anesthetic. The use of an anesthetic would make it impossible to find the sensitive areas, and, furthermore, the cauterization is superficial and lasts only a fraction of a second. The current should be turned on until the point of the electrode is almost instantly brought to a white heat. It should then be introduced cold into the nose, a sensitive area found with it, and the current turned on by pressing the button on the handle. The moment the white heat is seen in the nose the button should be released and the electrode

removed. Another sensitive area should be found and cauterized in like manner. From four to five sensitive areas may be cauterized at a sitting. The treatment may be repeated in from five to seven days.

(*b*) Nasal catarrh, if present, should be treated during the period of quiescence; that is, when the hyperesthetic rhinitis is not active. (See various forms of Chronic Rhinitis.)

(*c*) Nasal polypi should be removed during the period of quiescence, although they may be removed during the acute paroxysms. (See Nasal Polypi or Myxoma.)

(*d*) Deviations of the septum which cause any type of rhinitis, or which contribute to the causation of sinuitis, should be corrected during the period of quiescence, according to the methods described under Deviations of the Septum.

(*e*) Sinuitis, either catarrhal or suppurative, should be treated during the period of quiescence, according to the methods described under the Inflammatory Diseases of the Nasal Accessory Sinuses.

The late Dr. Schadle has reported that irrigation of the maxillary sinus results very favorably. At first a saponaceous substance is washed away, but the fluid finally comes away perfectly clear. Dr. Schadle believed that the ostium maxillare is so large that it admits the irritating substance which excite the paroxysmal attacks, and that when washed from the antrum the symptoms are relieved. I doubt this explanation, and am inclined to believe the relief is due to the lessened irritation of the nasal mucosa by the discharge from the antrum.

I have known equally good results following the total exenteration of the ethmoidal labyrinth *via* the nose. One patient was compelled for three months each year to sleep in a sitting posture with her head upon a table. Since the radical removal of her ethmoidal sinuses the only manifestation of the old trouble is a mild asthma, which is present for short intervals at any season of the year. I have since performed a double Killian operation upon the frontal sinuses of this patient with complete success. This operation has apparently had no influence on the slight asthma.

In view of my theory of the etiology of hay fever, in selected cases, being sinuitis, and of Dr. P. M. Farrington's successful treatment of such cases by autogenous vaccines, it is proper to suggest the autogenous vaccine therapy in this disease. Inject 50,000,000 bacteria at the first treatment, gradually increasing the dose to 100,000,000 at the third treatment. The injections should be made every third or fourth day.

Protection of the Patient from the Pollen or Other Emanations which Excite the Acute Paroxysms.—(*a*) Small, soft sponges may be worn in the vestibules of the nose to filter the pollen and other irritating substances from the inspired air. They are sometimes effective, but, on the whole, are unsatisfactory. A moistened handkerchief may also be utilized for the same purpose by holding it close to the nasal openings. At best, these devices afford temporary relief, and cannot be depended upon throughout the paroxysmal period.

(*b*) The geographical treatment consists in the removal of the patient to a place where the exciting emanations are absent. Lake Superior or the Muskoka region in Canada and the Adirondack Mountains are favorite resorts for many patients in the United States and Canada. An extended ocean or lake trip is also a satisfactory method of escaping from the emanations of the irritating pollen, etc.

While the geographical treatment is not always effective, it is nearly always so if protracted over the entire period of the acute exacerbations. Some patients may return before the expiration of this period without experiencing a recrudescence of the acute symptoms, although this is rarely so. Others are not relieved by a change of geographical location; at least, all cases are not relieved by a change to the same locality. Each patient must learn by experience the place best suited for him. On the other hand, he may find relief for a number of seasons in one locality, and upon returning the following year may experience but little or no relief. Under these circumstances he should try another locality. If, for instance, he has been going to the Lake Superior region or the Muskoka Lake region, he should be sent to a higher altitude, as the Adirondacks or the Rocky Mountains.

Palliative Treatment.—Various local and internal remedies have been advocated, but none of them are of universal value. They may be tried in series in individual cases until one is found that gives relief.

(*a*) The extract of the suprarenal gland is often successfully used. It should be prepared, according to Dr. H. L. Swain, by adding 10 to 20 grains of the powdered gland to one-half dram of cold, sterile water. After stirring thoroughly, it should be filtered and a few drops of alcohol added to prevent early decomposition. Boric acid, cinnamon water, and camphor water may also be used to prevent decomposition. When thus prepared it should be applied to the nasal mucous membrane with a spray tube, or with thin pledgets of cotton pasted over the surface of the mucous membrane. It is harmless, except in those occasional cases in which it excites irritation and sneezing. S. Solis Cohen has used it internally with success.

(*b*) Insufflation of the powdered sulphate of quinine into the nose has been recommended. I have used it in a few cases with complete success, and in many others without result. When it is effective the nasal mucous membrane becomes dry and the turgescence disappears. The absorption of the drug causes tinnitus. In one case two insufflations of 5 grains each were followed by complete relief which lasted throughout the paroxysmal season. This case was a mild one, beginning the latter part of August.

(*c*) Alkaline and oleaginous solutions may be sprayed into the nose, with temporary relief. In some cases a postnasal douche of boric acid solution is soothing. Oil with menthol in 0.5 per cent. solution, or with 0.1 per cent. of formaldehyde, sometimes gives relief to the inflamed membrane. The formaldehyde burns for a few seconds and is followed by a grateful sense of relief.

(*d*) The itching at the inner canthi of the eyes may be relieved by irrigating with boric acid or normal salt solution.

(*e*) The rays of the 500 candle-power incandescent lamp (Fig. 100) applied for ten to twenty-five minutes over the face, with the eyes closed, at a distance of from twelve to eighteen inches, increase the speed of the arterial and venous currents. The passive congestion and edema are thereby reduced and the relief is considerable. (See Leukodescent Light and the Technique of Application.) The light should be applied from one to four times daily. In those cases in which its use is attended by marked relief a lamp may be installed in the patient's home. A lower power than 500-candle-power is not recommended, nor is a cluster of 50 candle-power lamps as efficacious as a single 500-candle-power lamp. The therapeutic value of the light is chiefly determined by the candle-power of a single lamp, no matter how many are connected in a series or in a group.

(*f*) Powdered diphtheria antitoxin has been used locally with gratifying results (Pierce). Numerous other local remedies have been recommended from time to time, but have proved of little value after more extensive trial.

(*g*) Antilithemic remedies, as the salicylate of soda, have been extensively used to counteract the uric acidemia with indifferent success except in occasional cases.

Serum Treatment.—The serum treatment recently introduced by Dunbar, while not perfected, affords relief in some cases. Sir Felix Semon, Liebreich, and Lobe indorse Dunbar's serum treatment, with the proviso that all the conditions recommended by him be observed. The serum is prepared in liquid and powdered form, the powder being the more stable and reliable. The solution may be applied to the conjunctiva or the nasal mucous membrane. The object of the serum is to afford immediate relief and ultimately to establish immunity. The conditions attending its use are so complex that it is at present a rather unsatisfactory remedy.

In my opinion, serum treatment will not solve the problem of the management of hay fever or its kindred types of hyperesthetic rhinitis. The predisposing factors are ignored in this method of treatment. There are conditions which render the mucous membrane of the nose susceptible to irritation by the toxin of pollen and other substances which excite hay fever. Heretofore we have regarded the neuroses and constitutional dyscrasias, the various obstructive lesions of the septum, and the catarrhal affections of the nasal mucous membrane as the predisposing causes. The treatment applied in accordance with these ideas has generally been disappointing. In my opinion we must look beyond the nasal chambers to the accessory sinuses for the real conditions which predispose the mucous membrane of the nose to the irritation by the pollen of certain grasses, flowers, etc. The irritation caused by the more or less constant discharge from the sinuses is, I think, a rather common cause of hay fever. Schadle has called attention to the relief afforded by the irrigation of the maxillary sinuses. According to my observations the exenteration of the ethmoidal sinuses (including the removal of the middle turbinate) has apparently resulted in a cure extending over three years. The sinuitis may or may not be purulent.

Indeed, the catarrhal type is often more irritating than the purulent as shown by the excoriations and fissures at the margin of the vestibules of the nose.

In view of these facts I believe that the ultimate cure of hyperesthetic rhinitis and asthma will not be found in the serum treatment, but in the proper comprehension and treatment of catarrhal and suppurative sinuitis. This will include the obstructive lesions of the septum and the structures within the "vicious circle" of the nose. The neurotic element is often so marked in these cases that any method of treatment may fail.

According to O. J. Stein the injection of a few drops of alcohol into the mucous membrane of the nose at the point where the sensitive branches of the sphenopalatine ganglion enter the nasal chambers (Fig. 2) controls the acute symptoms in hay fever subjects. Three to four injections at intervals of a few days suffice to control the attack throughout the season.

According to O. J. Stein, but two factors are necessary for the causation of hay fever, namely: (*a*) An internal irritant, which affects the sensitive nerve filaments, and (*b*) an external irritant, as dust, cold, light, the pollen of certain plants, etc., which affects the fifth nerve supplying the nasal chambers.

The internal irritant is the result of faulty metabolism, which causes what may be called the susceptibility of the individual, *i. e.*, a disturbance of the normal functional equilibrium.

The external irritant may be dust, pollen, a draught, light, cold, heat, pungent odors, the discharges from infected sinuses, etc. It need not enter the nose to produce irritation, as any area supplied by the fifth nerve may be the origin of the reflex symptoms. Hence a bright ray of light entering the eye may irritate the hyperesthetic ciliary nerve filaments and cause reflex symptoms in the nose, or a draught of air striking the side or back of the head may produce nasal reflex phenomena.

Technique.—(*a*) First correct any disturbance of metabolism and nutrition that may be present.

(*b*) Remove the local and external causative irritating factors, such as spurs and ridges of the septum, secretions from the sinuses and sensitive areas, and protect the patient from the particular pollen that poisons him, by instructing him to wear sponges in the vestibules of the nose, or by sending him to some place where this pollen is absent. If the eyes are the source of irritation, the patient should wear dark glasses.

(*c*) Reduce or temporarily abolish the sensibility of the nasal portion of the fifth nerve. This may be accomplished in some measure by the administration of certain drugs, as morphine, the bromides, atropine, cocaine, etc. The action of these drugs is transient, and they may have deleterious effects, and are not recommended, but on the contrary their use for this purpose is condemned.

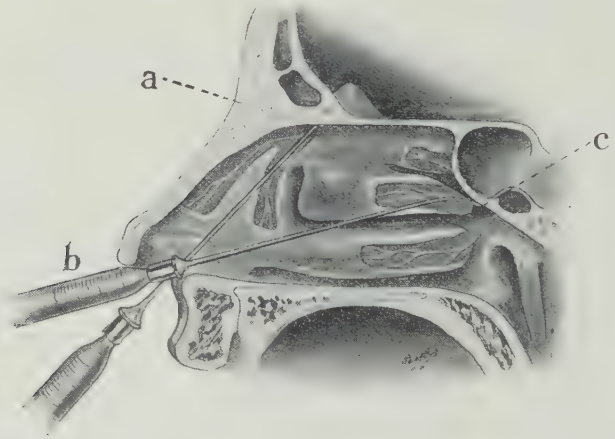
Stein's Treatment.—Dr. Stein recommends that the nasal branches of the fifth nerve be desensitized by the injection of alcohol into the nasal chambers. The nerve enters through the most anterior perforation in the cribriform plate (Fig. 203), and the needle should puncture

this point and be made to penetrate the nerve sheath. The method of procedure is as follows:

(*a*) Apply a 10 per cent. solution of cocaine to the cribriform and spheno-ethmoidal region of the nasal chambers.

(*b*) The straight needle, previously sterilized, is attracted to the glass syringe which contains the alcohol. It is then carefully inserted into the tissues just posterior to the nasal bone, *i. e.*, the anterior extremity of the cribriform plate (Fig. 203, *a*). Five drops of alcohol are then injected and the needle is withdrawn. The other nostril is then similarly treated. The posterior group of nerves is seldom treated at the first sitting, because in the majority of cases Dr. Stein has found that the injection of alcohol into the anterior group will control the symptoms. If, however, after a few days no relief is experienced the posterior group of nerves may be given an injection. For this purpose a longer needle with a curved tip is used, as shown in Fig. 203, *c*. The posterior nerves may be reached by directing the curved needle tip outward, upward, and slightly backward at the posterior extremity and lower border of the middle turbinate. After one

FIG. 203



Stein's method of treating hay fever: *a*, the anterior point where the needle is inserted; (*b*) the hypodermic syringe filled with alcohol; (*c*) the posterior point where the needle is inserted.

to four treatments, the patient should have relief through the hay fever season. No ill effects have occurred other than a slight hemorrhage, and pain and dizziness of short duration. This treatment does not protect against the recurrence of the symptoms the following season.

Killian has suggested and successfully practised the injection of cocaine into these nerves to produce anesthesia preliminary to intranasal operations.

ACUTE CIRCUMSCRIBED EDEMA OF THE NOSE; CORYZA EDEMATOSA; ACUTE CIRCUMSCRIBED EDEMA

This affection may involve both the pharynx and larynx in the same case. It is not an inflammatory affection, but is an edema of neurotic

origin, and probably results from some disturbance of the digestive tract. It is quite like urticaria, though it involves the mucous membrane. It is usually associated with other symptoms or diseases, as hay fever, urticaria of the skin, headache, gastro-intestinal disturbances (as watery vomiting and colicky pains), and itching. In Matas' case a distinct periodicity was present, the edema recurring regularly between 11 and 12 A.M. daily. In this case the toxin was probably the malarial plasmodium.

I reported a case in 1896 in which the angioneurotic edema came on during an attack of hay fever. Gastro-intestinal disturbance was also present. The edema involved the nose, soft palate, and hypopharynx. The mucous membrane was swollen, gray, and semitranslucent. The suffocative symptoms were severe, although at no time was there imminent danger from this source.

Numerous punctures of the edematous membrane were made and cocaine applied, after which the edema gradually disappeared. Free saline catharsis should be induced.

NASAL HYDRORRHEA; RHINAL HYDRORRHEA

Nasal hydrorrhea is a symptom of some other nasal lesion rather than a disease, and is characterized by thick, viscid, and slightly opalescent secretion more or less rich in mucin. The amount of discharge varies from a few ounces to a pint or more in twenty-four hours. According to St. Clair Thompson, the secretion contains amorphous matter and mucous corpuscles. The secretion when tested with alcohol or acetic acid throws down a stringy precipitate like mucin. When the precipitate is boiled with dilute sulphuric acid, a sugar-like material is formed; this is probably due to the presence of mucin. The proteid is coagulated by heat; it does not reduce Fehling's solution. Peptones and proteoses are absent. The alcohol extract of the secretions contains no reducing substance. The secretion may be distinguished from normal cerebrospinal fluid by the presence of mucin and the absence of a reducing substance.

Symptoms.—The clinical picture of nasal hydrorrhea shades off in one direction into cases of what are generally called hay fever, with symptoms of intense local irritation, while in the other direction it may consist of a passive and almost painless watery discharge from the nose. It is apparently a disease of adult life, which affects males and females equally. Although it may be more marked on one side of the nose than on the other, the flow usually comes from both nostrils. When handkerchiefs are soaked with it they generally become stiff when dry. In cerebrospinal rhinorrhea, on the other hand, the discharge is so watery that handkerchiefs dry quite soft; and in this affection the discharge is limited entirely to one nostril, unless there happens to be some obstruction on the affected side, when it may make its way to the opposite nasal fossa. When the fluid is of arachnoid origin, headache or other mental symptoms are frequent, but are relieved by the discharge.

The disease is not accompanied by lacrymation or suffusion of the conjunctiva, and photophobia, and it may occasionally give rise to sneezing, especially in the morning.

In nasal hydrorrhea the feeling of malaise begins with the discharge and only stops with its cessation. It is frequently ushered in with sneezing, photophobia, and lacrymation. It rarely continues during sleep, while cerebrospinal rhinorrhea continues day and night. It is very erratic in its onset and in its intermissions, and is very dependent on external influences and on conditions of health. Moritz Schmidt states that some cases have been observed which were dependent on ulcer of the stomach or biliary lithiasis. He defines the disease as a vasomotor rhinitis. McBride recognizes the diversity of the conditions of which nasal hydrorrhea may be but a symptom. I have seen cases in which the reactions given by St. Clair Thompson were present, thus differentiating the condition from cerebrospinal rhinorrhea.

Treatment.—The treatment should be addressed to the morbid nasal lesions, such as are found in hay fever or other forms of hyperesthetic rhinitis, or to any other pathological condition present in the nose and accessory sinuses.

CEREBROSPINAL RHINORRHEA

St. Clair Thompson, in 1899, made a notable contribution to rhinological literaturewhen he described for the first time the escape of cerebrospinal fluid from the nose. Such cases had been previously regarded as nasal hydrorrhea. Thompson's analysis of his and other cases, recorded in the literature under various names, made the differential diagnosis between cerebrospinal rhinorrhea and nasal hydrorrhea quite clear. The subarachnoid fluid may, under conditions not yet clearly demonstrated, escape from the cranial cavity through the nose without apparent harm to the patient. The fluid is clear and watery in contrast to the slightly opalescent and viscid fluid of nasal hydrorrhea. The dripping is constant and is free from taste, sediment, odor, albumin, and mucin. It reduces Fehling's solution.

Etiology.—The etiology is as yet but little understood, although Thompson is inclined to believe that there is some pathological change in the contents of the skull leading to increased intracranial pressure. In 17 out of 21 cases recorded there were cerebral symptoms, and 8 showed retinal changes. The following table prepared by St. Clair Thompson gives the essential tests for cerebrospinal fluid:

1. The fluid is perfectly transparent like water, and contains no sediment.
2. It is faintly alkaline in reaction, and either tasteless or slightly salt.
3. The specific gravity is between 1005 and 1010.
4. It is not viscid, and gives no precipitate (mucin) on adding acetic acid.

5. On boiling there is not more than a trace of coagulum of serum globulin and serum albumin.

6. Cold nitric acid gives a precipitate which disappears on heating, and separates again on cooling.

7. Saturation with magnesium sulphate should give a precipitate. Saturation with sodium chloride should also produce a precipitate. Ammonium sulphate should be tried if the above salts fail.

8. The liquid should give a pink or rosebud color with a trace of copper sulphate and excess of caustic potash.

9. When boiled with Fehling's solution there should be a reduction of the copper (due to pyrocatechin or some similar body).

10. The reducing substance may be obtained by evaporating to dryness an alcoholic extract of the fluid. It is then found in the form of needle-like crystals.

11. The aqueous solution of this residue does not ferment with yeast.

If applied to suspected cases, these tests will in future avoid any question as to the true nature of cerebrospinal fluid when it escapes from the nose.

Treatment.—The successful treatment of cerebrospinal rhinorrhea is obviously almost impossible. Whatever may be done, extreme care should be exercised to avoid infection of the nose, which might be communicated to the meninges or to the cerebrospinal fluid of the brain and spinal cord.

ASTHMA

Asthma may or may not be of nasal origin. The bulbar nuclei of the fifth nerve has an anatomical connection with the vagus, hence it is possible for an irritation in the nose to excite reflex phenomena in the lower respiratory tract. The most common cause of asthma of nasal origin is ethmoiditis accompanied by nasal polypi. In other cases hypertrophy, hyperplasia, and other morbid lesions appear to cause it. On the other hand, these conditions are often present without exciting asthma.

Treatment.—The treatment of asthma of nasal origin consists in the correction of the nasal morbid lesions, especially ethmoiditis, polypi, or hypertrophy of the turbinated bodies. (See Ethmoid Operations.)

A useful test as to the curability of the case is to apply a solution of cocaine to the mucous membrane of the nose, and if the asthma is greatly relieved or altogether checked, it is probable that the removal of the morbid lesions will result in a cure, though this cannot be positively promised, nor can it be stated how long the relief will continue.

EPILEPSY OF NASAL ORIGIN

Epilepsy of nasal origin has been reported by various authors. Watson Williams refers to a case which was brought on by cauterizing the nose

for nasal polypi. He also cites two cases reported by Baron: one case had nasal polypi, the removal of which was followed by marked alleviation of the epileptic seizures; the other case was a young unmarried woman who had had epileptic fits at her menstrual periods from the time menstruation began. Her inferior turbinated bodies were greatly hypertrophied, and she was always troubled with nasal stenosis during the menstrual periods, and it was at these times only that the fits occurred. Removal of the hypertrophied tissue was followed by a cessation of the fits for seven or eight months, and when they began again the turbinal hypertrophy was found to have returned.

I have a patient who has sarcoma of the nose, upon which I operated in April, 1903, and who has had repeated epileptic fits since the operation. Following each fit I have found a sequestrum of bone in the ethmoid region near the cribriform plate, after the removal of which the fits did not return for several weeks or a few months.

Nasal Tachycardia.—Watson Williams in his treatise on *Diseases of the Upper Respiratory Tract* cites the experiments of Gruber and the reports of several cases as follows:

Of the 43 subjects tested by Gruber, 13 with normal noses and 30 with nasal disease, the irritation of the nasal mucosa was negative. Watson Williams has never seen a case of reflex effect on the heart from nasal disease, though Spencer Watson reports one apparently due to polypi. Charsley observed, after cauterization of the inferior turbinate, temporary exophthalmos with tachycardia, the pulse ranging as high as 110 per minute, lasting for a period of three months. B. Fränkel and Hack report cases simulating Graves' disease, with goitre and tachycardia, which disappeared after curing the existing nasal disease.

CHAPTER XIII

NEOPLASMS OF THE NOSE

MYXOMA, OR NASAL POLYPUS; HYPERPLASTIC RHINITIS

Myxoma, or nasal polypus, is usually a pedunculated tumor of hyperplastic tissue, which most often grows from the middle turbinated body, the uncinate process of the ethmoid bone, or the ethmoidal cells, though it is not infrequently present in the maxillary frontal and sphenoidal sinuses. It is usually significant of a preëxisting catarrhal or suppurative inflammation of the sinuses, and is classified by Uffenorde as hyperplastic rhinitis (Skilleren). Some believe the tumor is primary and the inflammation of the sinuses secondary. Such a belief probably results from an indefinite conception of the symptoms of catarrhal sinuitis. Fortunately, catarrhal inflammation of the sinuses is now well understood, and I believe that clinical experience will show that the inflammation exists prior to the formation of the myxomatous tumors.

Etiology.—While it has not been definitely proved that nasal polypi are directly due to sinuitis, they nevertheless often appear to be secondary to such an inflammation. If the cases are carefully studied, it will often be found that the patients complain of a vague frontal headache, pressure between the eyes, dizziness, especially upon stooping forward, irritability of the eyes upon prolonged reading, or difficulty in securing a proper refraction of the eyes. Some or all of these and other symptoms are present in catarrhal as well as in suppurative sinuitis. It is claimed that repeated attacks of coryza may cause polypi. This is practically equivalent to saying that they are due to sinuitis, as the distressing symptoms of coryza are usually due to the associated inflammation of the accessory sinuses. Clinically we know that polypi are often associated with suppurative sinuitis and with caries of the bone in the immediate neighborhood of the tumors. Some writers state that polypi are found in the less obstructed nasal cavity, and use this as an argument against the previous existence of sinuitis. I believe that a careful examination of the nose will show that the polypi are usually present on the side of the nose in which there is the *greatest obstruction in the region of the middle turbinated body*, or "vicious circle" of the nose. A casual examination of these cases often shows a concavity on the side of the polypus, but the concavity is in the lower portion of the nasal chamber, while there is a convexity high up on the same side. It is easy to understand how the examination might show an open nostril on one side in this instance, if the lower portion of the nose only were taken into consideration. If, however, the upper portion is considered, the obstructive lesion is readily discovered on the side where polypi are present.

One of the most frequent causes of nasal polypi is a preëxisting inflammation of the membrane of the nasal sinuses and of the nasal mucosa in the region of the cell openings. The irritation and pressure give rise to a passive congestion and a proliferation of cells. A serous or edematous infiltration is a later manifestation. The connective-tissue cells subsequently become filled with the serum, thus leading to a hydropic degenerative change in some cells, and a myxomatous or gelatinous change in others (D. Braden Kyle).

The tissue thus degenerated becomes pendulous and in most instances pedunculated. Such a tumor is known as a polypus.

Hyperplasia of the nasal mucous membrane, due to other causes, may develop into nasal polypi. If, for instance, a foreign body is lodged in the nasal chamber for a long time, or any other continued source of irritation is present, it may result in nasal polypi. Some writers claim that the suction of the inspiratory current of air produces the tumors. D. Braden Kyle has pointed out that the ingoing current of air exerts as much pressure as it does suction. As a matter of fact, the presence or absence of suction depends largely upon the location of the obstructive lesion of the septum in relation to the polypi. If the polypus is posterior to the obstructive lesion, it is subject to suction from the rarefied or negative air pressure posterior to the obstruction. If there is no anterior nasal obstruction, the polypi are subjected to pressure rather than to suction. Suction may have something to do with the formation of polypi in some cases, but it is not probable that it is often if ever the sole cause.

Pathology.—While polypi are usually called myxomata, they are, as a rule, fibromyxomata. Pure myxoma is rare, and when found consists of an epithelium-covered connective-tissue sac, which contains a mucoid fluid, some bipolar spindle cells, and a fine network of connective tissue. The fibromyxoma, the usual type, is much richer in connective tissue, and less so in mucoid fluid. The tumors are supplied with bloodvessels and nerve filaments, which do not penetrate the substance of the tumor, but are limited to the mucous membrane covering it. They contain plasma cells, which stain with polydrome, methylene blue, and eosin. Robert Levy reports a case of multiple cystic polypus richly supplied with bloodvessels, as shown in Fig. 205.

Symptoms.—The symptoms of nasal polypi are often complex on account of the nasal obstruction (middle turbinal region) and the associated inflammation of the nose and sinuses, which usually co-exist.

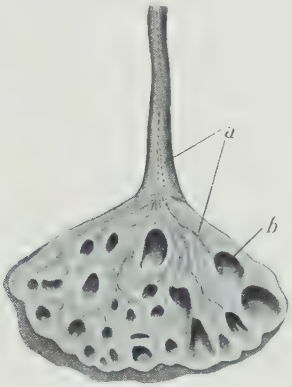
The symptoms caused by the polypi are largely dependent upon their location, size, and the amount of obstruction produced. If pedunculated, and hanging into the lower portion of the nose, they give rise to the sensation of a movable foreign body. The patient can sniff and blow them back and forth in the nose at will. If sessile, they cannot be thus moved, but cause a feeling of tightness or of fulness across the bridge of the nose. The voice has the nasal twang in proportion to the obstruction produced. The voice is often muffled, owing to the almost total loss of nasal resonance.

Upon examination a grayish semitranslucent tumor is seen hanging in the middle meatus of the nose. If pedunculated, it may move with the inspiratory and expiratory currents of air. Pressure with a probe shows a soft and yielding mass freely movable in the nasal chamber. There may be single or multiple tumors, but the latter are the more frequent. H. W. Loeb reported a case from which he removed 308 polypi at one sitting. They varied in size from that of a pinhead to such proportions as to protrude from the nose.

Fig. 204

The apparently open nostril, only open in its inferior portion. The obstruction in the upper portion interfering with drainage and ventilation of the sinuses, hence it gives rise to sinuitis, or hyperplastic ethmoiditis and later to polypi. Nasal passage obstructed in its lower portion. Open in the upper portion, hence drainage and ventilation of the sinuses are good; sinuitis and polypi absent. Polypus likely to form on the apparently open side, but in reality on the side where there is an obstruction in upper or sinus portion of the nose.

Fig. 205



A polypus of the cyst adenoma type removed from the nose: 4 cm. long, 2.5 cm. wide, 1.25 cm. thick, weight 8 grams, color pinkish white, solid and elastic. The section shows numerous cavities filled with colloid and caseous material. Some of the cysts are lined with ciliated epithelium; others have a degenerated columnar cubical or flattened epithelium, and in some the epithelium is entirely lost. Some areas are infiltrated with inflammatory round cells: *a*, bloodvessel; *b*, cyst. (Robert Levy's specimen.)

Various reflex symptoms, as cough and asthma, may be caused by polypi. I have seen a case in which the cough and asthma were so persistent as to compel the patient to sleep every night for three months at a time with the head on a table. This and other similar cases were relieved by the removal of the polypi and the total exenteration of the ethmoidal cells. External signs of nasal polypi are not always present, excepting the inclination to keep the lips parted, to supplement the nasal breathing. In rare cases the tumors are of such aggregate magnitude as to broaden the bridge of the nose.

The sense of smell may be impaired or lost, owing to the closure of the olfactory fissure. The pharynx may be dry on account of the loss of the nasal respiratory functions, or from the thick, tenacious mucopus which is discharged into it.

Caries and necrosis of the bone of the middle turbinal and of the ethmoidal cells may be found in some cases by the use of a heavy blunt-pointed probe. A small probe should not be used, because it might readily pass through the degenerated mucosa and lead to a mistaken conclusion as to the condition present. The probe should be gently passed over the mucous membrane of the middle turbinal, the ethmoid space above, and along the lip of the hiatus semilunaris (uncinate process), as these are the most frequent sites of nasal polypi.

The symptoms of the associated disease of the sinuses are headache, dizziness, especially upon stooping or sudden jarring, irritability of the eyes upon prolonged reading, or occasionally unilateral blindness. (See Diseases of the Sinuses.)

Prognosis.—The prognosis of nasal polypi is good if they are removed, and the preëxisting disease of the nose and sinuses which caused them is also remedied. In those cases in which the cause is a slight nasal inflammation the removal of the polypi followed by cauterization of their points of attachment will effect a cure. If the polypi are removed and the cauterization is neglected they are likely to recur. In those cases which are due to hyperplastic rhinitis or suppurative inflammation of the sinuses, it may be necessary not only to remove the polypi, but to exenterate the ethmoidal sinuses also. If caries of the bone is present, the operative procedure should include it as well as the polypi.

Treatment.—In view of the tendency of the polypi to recur, the treatment is not as simple as is ordinarily supposed. The average practitioner regards his duty as being performed when he removes the growth, or growths, and establishes a fair degree of nasal respiration. The aim should be, however, to not only remove the growth, but to remove the tissue from which it springs, and to remove the disease process (sinuitis), which is often the cause. Whether or not bony necrosis is always present, clinical experience teaches us that polypi are much less likely to return if the ethmoidal cells from which they often spring is removed. The use of the galvanocautery or fused chromic acid upon the stump of a solitary polypus effectually prevents its recurrence.

The surgeon should ascertain as nearly as possible the points from which the polypi spring, so that he may determine the difficulties likely to be encountered in the operation, and formulate a correct prognosis if the operation is refused by the patient.

Surgical Classification.—I. If polypi spring from the free border of the middle turbinated body their removal and after-treatment are comparatively simple. In this location it is not difficult to engage the snare around the growths in such a way as to include also a portion of the middle turbinate from which they spring, or the turbinal tissue may be removed with Holmes' scissors. Thus in a single operation it is sometimes possible to eradicate both the growths and their points of attachment.

II. If they have their origin above the middle turbinated body there is a strong probability that they come from the posterior ethmoidal cells. Here the treatment is much more complicated. It may become necessary to remove all, or a large part, of the middle turbinated body,

and to exenterate the ethmoidal cells. After this is done, the case may require attention for several weeks.

III. When they have their origin in and around the hiatus semilunaris, either the maxillary, anterior ethmoidal, or the frontal sinus may be the seat of infection, and it may be necessary to perform a radical operation upon the affected sinus to effect a cure.

IV. In other cases they spring from the anterior ethmoidal cells, in which case these cells and the frontal sinus may be seriously involved.

It is evident, therefore, that the simple removal of the polypi, or myxomatous growths, does not constitute the whole duty of the attending surgeon. Such treatment is usually only palliative and temporary. The presence of the polypi should be regarded as an indication that hyperplasia of the mucous membrane and bone and sinuitis are present. The principles of treatment for inflammation of the middle ear apply with equal force here. They are, briefly, (1) to establish free drainage; (2) to remove the morbid material; and (3) to maintain asepsis of the parts while healing is in progress.

Operative Technique.—I. *Polypi springing from the free border of the middle turbinated body* are perhaps the most easily and successfully treated of the types enumerated above. They are accessible and are attended with less involvement of the deeper tissues than those which are in either of the other locations. The method of procedure is as follows:

(*a*) Wash the nasal cavity with a warm antiseptic spray and apply adrenalin and a 4 per cent. solution of cocaine. This is most effectively applied on a thin pledget of cotton saturated with the solution and introduced with an applicator and adjusted over the operative field. The pledget should be left in position for about seven minutes.

(*b*) Carefully inspect the polypus by the aid of reflected light, and determine as nearly as possible its point of attachment. Having determined that it springs from the free border of the middle turbinated body, the next step is to examine for evidences of other diseased processes.

(*c*) With a large blunt probe the point of attachment and the neighboring parts should be examined for bare, rough bone. If a small probe is used, it may penetrate the unbroken tissue and thus come into contact with bony tissue. It is quite important, therefore, that a large one be used. It is not always possible to detect denuded bone, but if the examination is made in every case it may be found where it is not otherwise suspected.

(*d*) The wire loop of the snare should now be introduced, so as to encircle the pendent tumor. It should be held so that both sides of it are against the septum, the lower portion of the loop being on a level with or lower than the inferior portion of the polypus. It should then be turned so that its inferior part passes outward under the polypus, and then in an upward direction until the polypus is encircled. The procedure is often facilitated if the loop is also moved slightly in a forward and backward direction while engaging the polypus.

(*e*) Care should be exercised to carry the loop so as to include the point of attachment and a portion of the middle turbinated body it

possible. If the growth is on the anterior portion of the turbinate it is usually easy to include the anterior third of it. The loop passes backward, under, and on either side of the turbinate, while the cannula (Fig. 206) is firmly placed in the notch formed by the anterior attachment of the turbinate to the anterior wall of the nose.

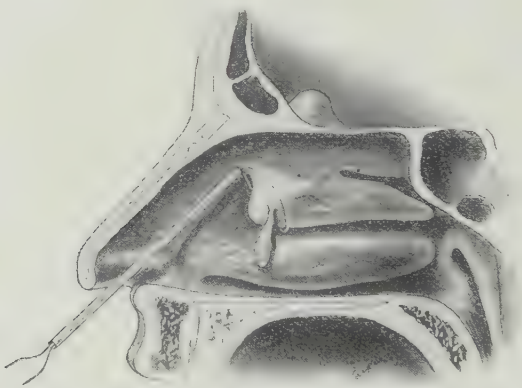
(*f*) Firm pressure of the cannula into the notch being maintained, the loop is tightened until the tissues are engaged. It is still further tightened until the anterior portion of the turbinate, to which the growth is attached, is severed.

(*g*) With a blunt probe the wounded surface is examined for evidences of carious or necrotic bone.

(*h*) If softened or necrotic bone is found it should be removed by curettement.

(*i*) If none of the middle turbinated body is removed the fibrous base of the polypus should be cauterized at the next sitting three or four days later.

FIG. 206



Removing a polypus and anterior end of the middle turbinate with a snare.

(*j*) The after-treatment should consist of the use of warm antiseptic douches or sprays and the insufflation of bismuth-iodine powder. If the douche is used, the Birmingham nasal douche is preferable to any of the pressure or fountain douches, as they are likely to force the solution into the middle ear, or the region may be packed with cotton saturated with a 10 per cent. solution of ichthyol.

II. When the polypi have their attachment *above the middle turbinated body* they usually spring from the posterior ethmoidal cells, and the treatment is correspondingly more difficult. One may be able to remove a portion of the growths, but it is difficult to reach their points of attachment. It therefore becomes necessary to remove the anterior half or all of the turbinated body. This is not objectionable, as the ethmoid cells contained therein and those in the body of the ethmoid bone are probably more or less filled with budding polypi. In cases of this class

my method of procedure is the same as for the removal of the ethmoid cells and middle turbinate *en masse.*

III. If the polypi *spring from the hiatus semilunaris* or *infundibulum* it may become necessary to open the maxillary antrum, which may also be the seat of similar growths.

These should be removed with the cold-wire snare and their *bases* cauterized. If upon further observation the antrum is found to be affected, the Caldwell-Luc or Denker operation should be performed.

IV. When the *polypi are attached to the border of the hiatus semilunaris,* mouth of the infundibulum, there is probably an involvement of the anterior ethmoidal and the frontal sinuses. The treatment is much like that described in I, in so far as the removal of the polypi is concerned. Subsequently it may become necessary to remove the anterior half of the middle turbinated body.

After this is done the diseased area is exposed to further examination, and, if necessary, to more extensive operation by curettement. In other words, the obstructions within the "vicious circle" should be obliterated.

No arbitrary rules can be laid down in a text-book for the guidance of the surgeon. He must study the facts in each case, and arrive at a conclusion as to the best course to pursue. The foregoing operations are sometimes advisable if it is hoped to effect a permanent cure of nasal polypi. These operations are usually only described in connection with the subject of empyema of the nasal accessory sinuses. I have described them in connection with polypi in order to emphasize the significance and importance of these growths, as pointing to conditions much more important than the polypi themselves. While in some cases it may not be shown that the polypi have much significance, nevertheless, in my experience, the more nearly I have treated polypi as though necrosis and suppuration were associated with them, the more satisfactory have been my results.

For timid patients non-surgical treatment may be recommended, as the injection of a saturated solution of the sulphate of zinc, or a solution of tannic acid into the substance of the polypi. I have occasionally used tannic acid with satisfactory results. A few minims should be injected with a hypodermic syringe into the body of the tumor. Within two or three days it shrinks and sloughs away. In the aged or the infirm it is usually inadvisable to recommend measures more radical than the simple removal of the polypi, as the danger from shock and acute infection is greater in these subjects.

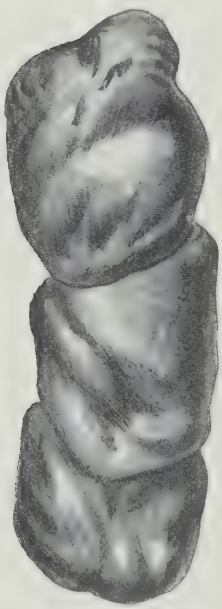
Papilloma.—Papilloma of the nose is rare, but when it occurs it appears as a corrugated red mass growing either from the inner or inferior surface of the inferior turbinated body, the septum, or the posterior end of the inferior turbinated body. The subjective symptoms are those of a partial nasal stenosis; the patient often consults the physician only on account of nasal "catarrh."

Treatment.—The treatment consists in the complete removal of the growth with a snare or nasal scissors. The surrounding tissue should be anesthetized by the local application of a 5 to 10 per cent. solution

of cocaine, after which the tumor is excised. After the bleeding has ceased the wounded surface should be mopped dry and cauterized with the galvanocautery. This is done to prevent a recurrence of the growth. When papilloma recurs in a patient forty or more years of age, the possibility of carcinoma should be suspected.

Fibroma.—Fibroma of the nose is characterized by the presence of a dense fibrous growth containing bloodvessels and no mucous glands, with slowly increasing nasal obstruction. The growths vary in size, are smooth and pale pink in color. They are firm to the touch or probe pressure, though not as dense as bone or cartilage. They may be sessile or pedunculated (Fig. 207). If pedunculated, they are movable like a polypus, though their consistency is quite different.

Fig. 207



Fibromyxoma removed from the epipharynx. Actual size. (Specimen kindly loaned by A. G. Wippern.)

They are usually attached to the septum, the floor of the nose, or to the turbinated bodies. They sometimes have multiple secondary attachments, owing to the inflammatory reaction excited by their presence.

Treatment.—The treatment consists in their complete removal, with a snare or cutting forceps. In those cases in which the tumor is pedunculated and comparatively small, the removal with the cold-wire snare or the author's turbinotome is the easiest and best method to pursue.

When the growth is sessile and large it may be removed piecemeal with cutting forceps, or at least so much of it that the snare can be passed over the remainder. This procedure may be done under cocaine anesthesia. When the growth is so large that it invades the surrounding structures of the nose, and extensive adhesions are present, it may become necessary to resort to a temporary resection of the superior maxilla to eradicate it.

The operation as given in *Surgical Technique*, by Drs. von Esmarch and E. Kowalzig, is as follows: Osteoplastic, or temporary, resection of the upper jaw (von Langenbeck, 1861) is performed for the removal of non-malignant fibrous or cavernous tumors which originate from the base of the skull, fill the nasal part of the pharynx (nasopharyngeal space), and force themselves into the maxillary sinus, or through the sphenomaxillary fossa into the temporal fossa (retromaxillary tumors).

By reflecting a portion of the upper jaw upward, which has been sawn through, but which remains in connection with the soft parts, the tumor is completely exposed, so that it can be cut off from the base of the skull with a knife or scissors; this portion of the upper jaw is then replaced and the skin is sutured over it.

Von Langenbeck proceeds as follows: 1. An external incision is made down to the bone in the form of a curve from the external angle of the nostril to the middle of the zygomatic arch (Fig. 208).

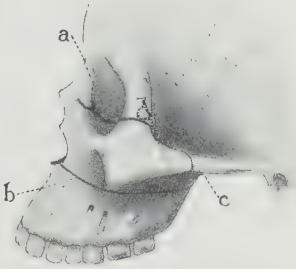
2. The insertion of the masseter muscle is separated from the lower margin of the malar bone portion of the buccal fascia.

3. After the lower jaw has been pressed downward by a gag inserted at the angle of the mouth on the healthy side, the right index finger is forced into the sphenomaxillary fossa between the tumor and the upper jaw and then through the distended sphenopalatine foramen as far as the nares; an elevator is carried along the finger, and on it a fine metacarpal saw is introduced into the pharynx. The left index finger, introduced from the mouth into the pharynx, catches the point of the saw.

Fig. 208

The incision for the temporary resection of the superior maxilla.

Fig. 209



Von Langenbeck's operation for the temporary excision of the superior maxilla: *a*, *b* (Fig. 208), the external skin incision; *c*, the zygomatic arch is first sawn through from within outward; *d*, next, the frontal process of the malar bone is severed with a metacarpal saw as far as and into the inferior orbital fissure, the orbital plate of the inferior maxilla as far as the lacrymal bone closely below the lacrymal fossa, and, finally, the middle of the nasal process of the superior maxilla as far as the nasal bones are divided. The contents of the lacrymal canal should be carefully guarded from injury; *b*, horizontal division, with a saw, of the superior maxilla above the alveolar process as far as and into the pyriform aperture.

4. Horizontal division is obtained by sawing the upper jaw above the alveolar process as far as and into the pyriform aperture (Fig. 209, *b*). In operations on the right upper jaw, the left index finger is forced into the maxillary fossa, and the operator saws toward it from the nasal passage.

5. Make the external incision down to the bone in the form of a curve from the root of the nose along the lower orbital margin, meeting the first skin incision at the zygomatic arch (Fig. 208).

6. After the external lower angle of the orbit and the angle between the temporal and the frontal process of the malar bone have been freed from the soft parts the zygomatic arch is sawn through in the middle from within outward; next, the frontal process of the malar bone as far

as and into the inferior orbital fissure, the orbital plate of the upper jaw as far as the lacrymal bone closely below the lacrymal fossa, and, finally, the middle of the nasal process of the upper jaw as far as the nasal bone divided with a metacarpal saw. The organs which constitute the lacrymal duct should be protected.

7. By means of an elevator inserted under the malar bone the excised piece of the upper jaw is lifted up toward the median line, like the lid of a box. The sutural connection between the nasal bone and the upper jaw, in most cases, breaks during this maneuver.

8. With a broad elevator the tumor, now laid bare, is lifted out of the sphenomaxillary fossa, and the base is detached from the under surface of the skull with a knife, scissors, or thermocautery. Finally, the resected portion of the upper jaw is replaced in its former position and the skin is closed by sutures.

Adenoma.—Adenoma bleeds so readily upon examination with a probe that sarcoma is at once suggested. A microscopic examination, however, reveals the true character of the growth. This type of tumor grows from the septum or the ethmoidal region and produces rapidly increasing nasal stenosis. Adenoma, like polypi and papillomata, has a strong tendency to recur unless completely removed. It consists of a simple hyperplasia of gland structure having its type in the acinous or tubular glands. It also has a tendency to maligant degeneration.

Treatment.—The treatment should consist in the total removal of the tumor. In order to insure this, its base should be cauterized or curetted. The bleeding which attends the removal of adenomata is considerable, but may be readily controlled by a nasal tampon of bismuth gauze.

Lymphoma.—Lymphoma of the nose is characterized by a smooth mass, pinkish red in color, and less dense in consistency than fibroma. It is not common and a microscopic examination is necessary for a positive diagnosis. The treatment is the same as for polypus and fibroma.

Angioma.—Angioma of the nose is rare (Harry Kahn), and consists of a distention of existing bloodvessels rather than of new-formed ones. According to D. Braden Kyle, the distention is due to changes in the walls of the bloodvessel from deficient nutrition rather than to mere congestion.

Symptoms.—The symptoms are those of more or less nasal obstruction, epistaxis, and a reducible and pulsating tumor. The nasal obstruction is proportionate to the size of the growth. Pressure upon the growth materially reduces its size. The pulsation is greater when the tumor is attached to a large artery than if it is attached to a vein, when the pulsation is much less and the color is blue, whereas if it is connected with both vein and artery the color will be a dark red.

Treatment.—The treatment consists in strangulation at the base of the tumor. The object of the strangulation is to cause closure of the bloodvessels which supply the tumor. If the strangulation is performed too quickly the vessels will not close and hemorrhage from their severed ends results; by gradually tightening the wire loop the vessels close and bleeding does not follow.

The galvanocautery loop is also adapted to the removal of these growths, when easily accessible and pedunculated, as it sears over the ends of the vessels and prevents subsequent hemorrhage. When the growth is sessile, silk ligatures may be passed through it and tied, thus strangulating a portion with each ligature. Cocaine anesthesia by injection is all that is necessary for either of these procedures.

Osteoma.—Osteoma[1] of the nose and the accessory sinuses is rare. It may occur in any of the accessory sinuses, but is more common in the frontal. It may invade the nasal and orbital cavities when growing from the sinuses. It sometimes springs from the inferior turbinated bone and occludes the nasal chambers. Cases have been reported in which the tumor had its origin in the nasal process of the superior maxilla.

Pathology.—Osteoma is usually composed of dense, compact, cancellous, horny tissue on a congenital or postnatal matrix of osteoclasts, and usually has its growth from the periosteum, though it may grow from the medullary portion of the bone. Some osteomata are soft and spongy, with a dense capsule of bone, while others are dense throughout their substance. The spongy type occurs most frequently. They are in some instances pedunculated, the pedicle being composed of either spongy bone or soft connective tissue and mucous membrane. They vary from the size of a small walnut to that of a goose egg.

Symptoms.—As the nasal chambers are usually invaded, nasal obstruction is a prominent symptom. The growth of the tumor externally produces more or less marked deformity, and in some instances the resemblance to horns is so great that the cases are referred to as "horned men." In some instances they present the "frog-face" type of countenance, especially when both sides of the nose are involved in the region of the infra-orbital ridge, as in O. J. Stein's case. Palpation of the tumor, whether intranasal or extranasal, yields a sense of bony hardness. The lacrymal duct may be occluded. The mucous membrane covering the tumor is usually pale, thin, and not eroded. Transillumination of the maxillary sinus may show obstruction to the rays of light. If constant mouth breathing is present it gives rise to epipharyngeal catarrh. In Stein's case there was inability to rotate the left eye inward. There was external divergence of two lines, the pupil was widely dilated and fixed, and did not respond to either light or accommodation. The fundus was normal.

Diagnosis.—The diagnosis is largely based upon the microscopic examination of the tissue.

Treatment.—In cases of syphilitic origin the iodides are of value. The removal of the bony growth is usually the best treatment. The technique of the operation varies with each case. Boenhaupt reported 23 cases in which the tumor grew from the frontal sinus, in 11 of which it communicated with the cranial cavity. It is obvious, therefore, that

[1] I am indebted to Dr. Otto Stein's paper on Symmetrical Osteoma of the Nose for most of the data on this subject.

osteoma of this region is the most serious from a clinical and surgical point of view.

In the removal of osteoma, if there is no pedicle, it is better to enucleate the tumor rather than to attempt to chisel or drill into its substance, as it is often so dense as to resist the instruments.

In one of my cases of osteoma of the epipharynx, the posterior choanæ were completely blocked. The bone was so dense that it could not be removed with a chisel. The only instrument that would penetrate it

FIG. 210

Lipoma of the tip of the nose. (Pynchon's case.)

was a trephine. With this a large portion of the tumor was removed through the nose, and nasal respiration was successfully reëstablished. One year later the nasal occlusion returned. This case should have been treated by temporary resection of the superior maxilla.

Lipoma.—Lipoma of the nose may be external or internal, and is usually pendulous. When external it generally affects the alæ of the nose. The case illustrated involves the tip of the nose (Fig. 210). The treatment consists of the excision of the growth.

MALIGNANT NEOPLASMS OF THE NOSE

Carcinoma.—Carcinoma of the nose is more rare than sarcoma, and usually begins in the anterior portion of the nasal structures, at which point there is the greatest physiological irritation.

Diagnosis.—The diagnosis is based upon (*a*) the presence of an intense irregular lancinating pain; (*b*) a mucopurulent secretion, which if ulceration is present is admixed with blood; (*c*) the characteristic ozena or stench of cancer; (*d*) nasal stenosis more or less marked according to the stage in which the disease is observed; (*e*) impairment of vision if the ethmoid cells are involved; (*f*) ulceration of the growth if in an advanced stage; and (*g*) cachexia. (*h*) In addition to the foregoing clinical symptoms it is usually necessary to remove a portion of the growth for microscopic examination. D. Braden Kyle properly calls attention to the necessity of observing two precautions in securing the specimen, namely: (1) That there should be as little laceration and irritation on the parts as possible; (2) that the portion removed should not involve directly the ulcerated area, which will contain inflammatory embryonic connective tissue, and, as pointed out by J. Bland Sutton, this cannot be differentiated from sarcoma or from a simple inflammatory process with ulceration. If, however, the specimen is taken early, before ulceration has occurred, this source of error may be obviated.

Prognosis.—The prognosis is always grave.

Treatment.—The surgical treatment of carcinoma of the nose, except in the very early stage, is contra-indicated.

The palliative treatment consists in the local application of orthoform powder to ease the pain, and local applications of dilute hydrochloric acid and formalin to the ulcerated areas.

Sarcoma.—Sarcoma of the nose is of slow growth, and is less malignant that sarcoma in other parts of the body. Unlike carcinoma it occurs most often before the fortieth year of life, and is not uncommon in infancy and childhood.

Diagnosis.—The diagnosis is based upon (*a*) progressive nasal stenosis; (*b*) a mucopurulent nasal secretion, which, in the advanced stage, becomes sanguinolent; (*c*) more or less slight pain in strong contrast to the intense pain in carcinoma; (*d*) the age of the patient, if below forty years, is also of diagnostic significance, though carcinoma occasionally occurs before this age; (*e*) finally, the diagnosis must be made by submitting a specimen of the growth to microscopic examination.

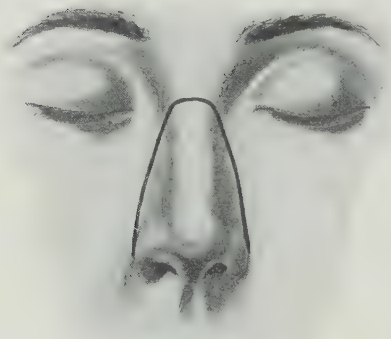
Prognosis.—The prognosis is grave, though not as grave as carcinoma. When operated upon early there is a fair chance of recovery. In one of my cases operated on by Ollier's method (Fig. 211), there has been no recurrence of the sarcoma after six years.

Treatment.—The treatment in the early stage is surgical, especially in view of the slighter malignancy of nasal sarcoma. The growth may be removed with a curette, or galvanocautery through the nasal orifices, or, if extensive, an external operation may be required.

Ollier's Operation.—This operation is performed under general anesthesia, with the head of the patient hanging over the end of the table in Rose's position. Postnasal tampons should be introduced to prevent entrance of blood into the epipharynx and larynx. An incision extending from the left ala of the nose, upward over the bridge, and thence downward to the right ala, should be made through the cutaneous tissue (Fig. 211). A Gigli saw should then be placed at the bridge of the nose and all the bony structures along the cutaneous incision severed.

The nose, thus temporarily resected, is then turned downward over the mouth. This having been done, the growth should be enucleated by blunt dissection, if possible, or if this cannot be done it should be

FIG. 211



Ollier's incision for exposing the nasal cavities for operative purposes.

FIG. 212

Lateral view of the Ollier incision.

removed by dull curettage. A sharp curette should not be used, as it leaves the lymphatic vessels open and may cause septic infection and extension by metastasis. Hemorrhage may be considerable, hence the postnasal tampons introduced before beginning the operation serve as bases against which strips of gauze may be packed to check it.

In my case, illustrated in Figs. 211 and 212, the hemorrhage was very profuse and necessitated the use of normal salt enemata. The transfusion of normal salt solution would have been better, but as arrangements had not been made for it the enemata were substituted. This patient was thirteen years old when I first saw her, and was fourteen when I per-

formed the Ollier operation. She is now twenty-two years of age, and is free from the growth. Bony sequestra have been removed from time to time, and but little ozena is present.

Having removed the tumor, the incision should be closed by sutures, and the tip of the nose raised into position and fixed with adhesive strips. The stitches should be removed on the fifth day. The nasal wound should be packed with gauze impregnated with bismuth or the compound tincture of benzoin, to prevent decomposition and saprophytic infection. The intranasal dressing should be removed and renewed daily.

CHAPTER XIV

EPISTAXIS (NASAL HEMORRHAGE). RHINOSCLEROMA. FURUNCULOSIS. SCREW-WORMS

EPISTAXIS (NASAL HEMORRHAGE)

Epistaxis is a nasal hemorrhage, that is, a bleeding from the interior of the nose. While the hemorrhage is usually from the anterior portion of the septum (90 per cent. of the cases, according to Casselberry), it may occur from any portion of the nasal mucosa. The bleeding is not often serious in character, though several deaths have occurred therefrom. It is most serious in bleeders, or hemophiliacs, and in arteriosclerosis, valvular heart lesion (right side), sarcoma, and pressure on the veins of the neck by aneurysm, bronchocele, and intrathoracic tumors.

Etiology.—(*a*) Anterior deflection of the septum is the predisposing cause of hemorrhage in a large majority of the cases. This portion of the septum is richly supplied with blood from the septal artery, a branch of the superior coronary, and is exposed to the ingoing current of air, which is often loaded with foreign particles. The air, furthermore, dries the secretions on the anterior portion of the septum, especially if it is deflected in this location. The membrane is quite thin in this area. Slight erosion of the mucosa readily gives rise, therefore, to nasal hemorrhage.

(*b*) Catarrhal inflammation causes chronic hyperemia of the mucous membrane, hence the increased supply of blood in the parts contributes to the epistaxis.

(*c*) A number of febrile diseases are often attended by epistaxis. The diseases most commonly thus characterized are typhoid and diphtheria. "Black diphtheria," or hemorrhagic nasal diphtheria, is attended with a destructive degeneration of the nasal mucosa, submucous hemorrhage, and epistaxis.

(*d*) The veins on the anterior portion of the septum are sometimes varicosed and give rise to hemorrhage.

(*e*) Obstruction to the portal circulation may be attended by nasal hemorrhage.

(*f*) Suppression of the menstrual flow and a severe hemorrhoidal hemorrhage is sometimes attended by a vicarious nasal hemorrhage.

(*g*) Traumatic epistaxis may result from picking the nose with the finger nail or violently blowing it. Intranasal surgery is frequently followed by severe nasal hemorrhage. This is especially true after operations upon the middle turbinate, the ethmoidal cells, and the "swell bodies" or erectile tissue of the inferior turbinated body. The

middle turbinated and the ethmoidal cells receive a generous blood supply from the anterior and posterior ethmoid arteries (Plate I, Fig. 1). External violence to the nose is often followed by epistaxis or the so-called "bloody nose."

(*h*) A perforating ulcer of the septum frequently gives rise to epistaxis. The vessel walls are broken down in the destructive process, and the granulation tissue upon the border of the perforation bleeds upon slight cause.

(*i*) Certain constitutional diseases, as hemophilia, Bright's disease, purpura, scorbutus, chloremia, leukemia, and arteriosclerosis, are characterized by nasal hemorrhage, for obvious reasons. Syphilis and tuberculosis of the nose also give rise to epistaxis.

(*j*) Sarcoma of the nose, like sarcoma elsewhere, is often attended with hemorrhage.

Treatment.—The treatment of nasal hemorrhage in most cases is very simple, as the local application of cocaine or of adrenalin readily stops it. In other cases, however, when the cause is a constitutional disease, a growth pressing on the veins of the neck, or when the trunk of one of the larger septal arteries, as the anterior ethmoidal, is severed in an intranasal operation, the bleeding is not so easily checked.

The hemorrhage may usually be checked by one of the following procedures:

1. Hot nasal irrigation is quite effective in many of the cases when the epistaxis is not due to some grave disease. The temperature of the water or normal salt solution should be as high as can be tolerated, or about 130°.

2. Ice-water may also be injected into the nose with advantage in operative hemorrhage while the patient is under an anesthetic. Only two or three injections of four ounces each should be used, as a greater quantity might produce serious shock to the brain by sudden or excessive chilling. I have frequently resorted to this method of treatment at the close of nasal operations when the hemorrhage was profuse, with the most gratifying results.

3. The local application of cocaine or adrenalin often checks the hemorrhage when it is of capillary origin. If blood clots are present, the nose should first be cleared. The adrenalin extract may be given internally for its hemostatic effect.

4. Blood clots are sometimes allowed to remain in the nose, with the idea that they will finally check the hemorrhage. This procedure is based upon an erroneous idea. The blood clots only serve to shield the bleeding area from such local medicaments as may be used, thus hiding the bleeding point from view. The bleeding usually continues beneath the clots, hence they should be thoroughly removed at once in order to expose the bleeding area to inspection and to make it possible to apply such local remedies as may be deemed necessary.

5. Astringent remedies, such as the nitrate of silver in 5 to 20 per cent. solutions, may be made from time to time when the oozing is persistent.

6. The application of the actual cautery has sometimes proved to be a

speedy and efficient means of controlling the bleeding; a flat-pointed electrode should be used at a cherry-red heat for this purpose.

7. Local pressure over the bleeding point for a few minutes will sometimes control the bleeding.

8. Tampons in the nose should only be resorted to in those cases in which the bleeding persists in spite of all other measures. Their use, as a general rule, should be avoided, as they are likely to give rise to conditions favorable to sepsis. The more completely the nasal chambers are packed with gauze the greater the danger. Hence a postnasal tampon, with one anterior to it, is the most dangerous of all. This method of packing the nose in epistaxis should be avoided except in an extreme emergency.

When bleeding occurs from the anterior portion of the septum, and it becomes necessary to introduce a tampon, I would advise the use of a Bernay tampon cut into the form of a nasal splint, as recommended by Simpson. It absorbs less of the secretions, and is easily introduced and removed without further injury to the diseased mucous membrane. The interior of the nose should first be covered with subnitrate of bismuth by insufflation to prevent decomposition of the secretions.

RHINOSCLEROMA

Synonyms.—It is probable that a rare lesion described as chorditis, chronic hypertrophica inferior, and what is known as Stoerk's blennorrhea are identical with rhinoscleroma.

Definition.—Rhinoscleroma is characterized by a cartilage-like hardness and nodular enlargement of the nose and other portions of the upper air passages. The affected tissues have no tendency to ulceration or to inflammatory reaction either in the growth or in the contiguous parts, although rhinoscleroma frequently affects the other divisions of the respiratory tract.

Etiology.—But little is known of the etiology of the disease beyond the fact that it is due to a specific microörganism, the bacillus of rhinoscleroma, and that it is chiefly confined to Austria and southwestern Europe. About 800 cases have been reported, and of these, about 20 occurred in America, but a large majority of these were born in Poland and Austria. It usually begins in youth, and most cases are observed between the ages of fourteen and forty-five. Sex seems to have no influence. Heredity seems to be a negative factor, though there is apparently a family predisposition to the disease. It is now generally regarded as a contagious disease.

Bacteriology.—The hard, cartilage-like nodules may affect the skin, and the mucous membrane of the nose, pharynx, larynx, and trachea. They spread with greater freedom in the mucosa than in the skin. The hard, nodular masses, or plaques, contain the encapsulated bacillus of rhinoscleroma, which is similar to Friedländer's bacillus, though the latter is not always encapsulated. The bacillus of rhinoscleroma is more rod-shaped

and stains by Gram's method, is motile, non-spore bearing, and aërobic. It always has a capsule in culture, as well as in the tissues. It occurs singly and in pairs. Gelatin plates show yellowish-white granular bodies in two or three days. In gelatin tubes the growth appears along the needle track as a whitish granular line, with an almost hemispherical elevation on the surface. The growth in the tube has the appearance of a round-headed nail. When grown upon agar it appears as a dirty whitish moist layer on either side of the needle track. On potato the growth is creamy white. It grows rather rapidly at a temperature of 37° C. It is pathogenic for mice, guinea-pigs, and rabbits.

Pathology.—The histological changes are inflammatory in character and usually begin on the nasal septum, trachea, or larynx. In rare instances the reverse course is pursued. The skin and mucous membrane of the nose assume a smooth nodular appearance of cartilage-like consistency, which pits little, if at all, upon probe pressure. The parts are sensitive to the touch, but are otherwise free from pain. Kaposi has likened the external appearance of the nose to keloid. According to Goodale the affected tissues consist histologically of certain typical elementary lesions. The substance of the swelling is composed of large plasma cells, irregularly distributed in all layers of the mucous membrane, and in the submucous tissue. They accompany the bloodvessels in the new portions of the growth. The plasma cells do not contribute directly to the hypertrophy, but it is possible that they become changed partly into spindle cells, and then give rise to the formation of new fibrillary tissue. Two forms of retrograde metamorphosis occur in the plasma cells. These may be transformed into swollen, hydropic, so-called Mikulicz cells, or into hyaline degenerated cells, probably identical with the so-called Russell's fuchsinophiles, described under Colloid Degeneration. The hydropic cells lie close together, have a distinct contour and spongy cytoplasm dilated into large masses, in which there is a smaller mass within a faceted nucleus. In this stage one often sees from six to eight bacilli in the cells near the nucleus which lie always at regular distances.

This stage appears, however, to be rapidly finished, and when the cell membrane breaks, the fluid contents, together with some of the bacilli, find an exit and fill some of the nearest lymph spaces. These cells are, however, intimately related to the direct action of the bacilli.

Symptoms.—The changes in the external appearance of the nose, while presenting many of the characteristics of keloid, are, nevertheless, rather easily differentiated from it by the whole symptom complex. The tissue at the tip of the nose becomes infiltrated, hard, and nodular. The nose broadens and becomes firmly fixed to the face. The tissues become more and more thickened, until the breathing is more or less occluded. The color of the skin varies from a red to a bluish or brownish red. The skin is traversed by small bloodvessels, and is usually slimy, though it may be finely wrinkled. The extension of the growth is rather slow, requiring several months to reach the epipharynx. The infiltration often interferes with the movements of the lips, the fauces,

and the larynx, and very rarely with that of the eyes and ears. There is no tendency to ulceration and discharge, or to edema and inflammation of contiguous parts.

Laryngeal stenosis may give rise to serious or even fatal dyspnea, otherwise the disease does not materially affect the general health.

Diagnosis.—Rhinoscleroma should be differentiated from syphilis, epithelioma, and keloid. The disease is exceedingly rare in this country, hence it is natural to infer that a suspected case in a native-born American is probably not rhinoscleroma, but that it is either syphilis, epithelioma, or keloid. Rhinoscleroma presents a hard, nodular growth, which usually begins at the anterior end of the nose and spreads gradually to the deeper recesses of the respiratory tract, without pain, but with some tenderness upon pressure, and without tendency to ulceration or inflammation of the surrounding tissues. In syphilis there is inflammation, while in epithelioma there is pain, ulceration, and discharge. In keloid the similarity is often so striking that it may be necessary to demonstrate the absence or presence of the germ of rhinoscleroma in order to make a differential diagnosis.

Treatment.—Thus far the extirpation of the diseased tissue has been tried with negative results as to the cure of the disease. The surgical extirpation of the diseased tissue has almost invariably been followed by recurrence. Tracheotomy should be performed when suffocation is imminent. Thiosinamin apparently softens the tissue (Glass), as it does in keloid; it may, therefore, be of some therapeutic value. A reliable method of treatment, however, has not been discovered. Freudenthal suggests the injection of Coley's fluid, as in sarcoma. The iodides and mercury have been tried with but little success. The *x*-rays have been used by Emil Mayer with apparent success, though it is probable that this mode of treatment will prove disappointing, as have all other methods.

FURUNCULOSIS OF THE NOSE

Definition.—Furunculosis of the nose is a superficial abscess formation which may occur in any part of the nose, and does not differ materially from the same process in the other parts of the body.

Etiology.—The abscess is usually located on the anterior portion of the septum, *i. e.*, that portion covered by the vestibular skin, and is usually due to an injury, as from picking the nose. One or more furuncles may be present at a time or they may occur in quick succession. The hair follicles of the vestibule offer favorable sites for the infection. If they recur frequently the cartilaginous septum becomes involved. Recurrence most commonly takes place in the young or the middle-aged, especially in those in whom an impoverished state of the blood exists. The infectious fevers are often attended with nasal furunculosis.

Symptoms.—There is more or less throbbing pain, swelling, redness, and tenderness. Elevated areas characteristic of boils may be seen upon inspection. When they are well advanced the centre of the elevation

is yellowish from the contained pus. The pain is often intense, on account of the closely attached and unyielding nature of the tissue composing the parts.

Treatment.—If seen early, before pus formation, the application of a 50 per cent. solution of ichthyol or a 10 per cent. glycerin solution of carbolic acid on a pledget of cotton will often abort the process. If pus has formed, they should be incised from within the nasal cavity with a sharp bistoury. After incision their cavities should be irrigated with warm boric acid solution and the tincture of iodine applied.

PHLEGMONOUS RHINITIS

This is somewhat different from furunculosis, in that it is an abscess formation affecting the nasal mucous membrane. The condition is rare except as the result of an operation or other traumatism. (See Abscess of the Septum.)

FOREIGN BODIES IN THE NOSE

Foreign bodies in the nose may be animate or inanimate.

SCREW-WORMS IN THE NOSE

Screw-worms in the nose have been reported by M. A. Goldstein, Hal Foster, and J. S. Steele in most interesting and instructive articles, wherein it is shown that their invasion of the human being is not as rare as might be supposed. (See Foreign Bodies in the Ear.)

The screw-worm fly is attracted by a foul-smelling discharge from the nose or the ear, and it need be in the nose but for a moment in order to deposit its eggs. Dr. Steel narrates a case illustrative of this point. A railway engineer, while walking across the plaza of a Mexican city, inhaled a fly into one nostril, which he immediately blew out through the other. Twenty-four hours later fulness and pain between the eyes was noted, which increased for three days, when he came under observation. He was affected with syphilitic rhinitis with necrosis of the nasal septum, which accounted for the fly being attracted to his nose. About one hundred worms were removed with the douche and forceps. Calomel fumes were inhaled, which seemed to exterminate all that remained, as they gave rise to no further symptoms.

Foster removed two hundred and seven worms from the nose of an old Irish woman who was subject to epileptic fits, during which she would fall to the gound. Following one of these seizures she noted an itching of the nasal mucosa, which was accompanied by headache and sneezing. She was told that she had hay fever, and large doses of quinine were administered. Two days later the nose began to bleed and to give forth

a very offensive discharge. The eyes were closed from swelling of the subcutaneous tissue of the face, and she was in such discomfort that she was unable to sleep.

Upon examination the nostrils were found to be entirely filled with worms. Inhalations of chloroform were administered, which rapidly rendered them lifeless, after which they were readily removed with forceps. The live worms clung with tenacity to the tissues when force was applied in their removal. There was great destruction of tissue, and the temperature reached 102°. There was a bulging of the anterior part of the nose as a result of the penetration of the worms at this point.

Goldstein's case was that of a farm laborer who slept outdoors in a hammock. He was affected with syphilitic rhinitis, which offered an ideal attraction to the Texas screw-worm fly. When examined, the nose was found to be filled with the eggs of the fly; five hundred were removed with the curette. The curettage was thoroughly done, considerable tissue being removed with the eggs. Forty-eight hours later the patient suffered excruciating pain in the nostrils, which were completely occluded. The skin over the frontal sinus was red and tightly drawn. On the sixth day there was swelling over the dorsum of the nose near its centre. This was incised and considerable pus evacuated. Several worms were subsequently removed through this opening.

Chloroform is the most effective remedy, and may be administered by inhalation or in diluted solution with a syringe. Calomel fumes are also of value, but do not act as quickly as chloroform. Steele's case shows that its effects were apparent after about four hours, whereas chloroform is effective within a few seconds or minutes.

Inanimate foreign bodies include almost every kind of inert substance small enough to be introduced into the nose, and some that are too large to be introduced into the nose, at least through the nasal opening. One such case was under my care and gave the history of having received a wound thirty years previously from the explosion of a musket. The left eye was destroyed at the time. Upon removal of the foreign body it proved to be the breech pin of the musket which exploded thirty years previously. The mass of iron, as large as the first joint of the thumb, still preserved its mechanical form, as the screw threads and the tubular space for the flash powder. The cap pin was also intact. In most instances the foreign body is voluntarily introduced by the patient. Young children have an inordinate desire to introduce such substances into their noses, hence most cases occur in young children. Idiots and the insane also delight in putting foreign substances into their noses.

The removal of the foreign body may be accomplished through the anterior nasal opening without the use of a general anesthetic, though in some cases this may be necessary. Forceps with good, grasping tips should be used to seize it and, after dislodging it, to remove it.

CHAPTER XV

THE SURGICAL CORRECTION OF EXTERNAL NASAL DEFORMITIES

THE surgery of external deformities of the nose is being more and more relegated to rhinologists, for various reasons, chief among which are: (*a*) The rhinologist has a more intimate knowledge of the structures of the nose and can therefore more intelligently conserve and utilize them in reconstructing it; and (*b*) the rhinologist of modern times is better trained and more skilled in surgical principles and practice than formerly. For these and other reasons a chapter on some of the simpler nasal deformities, especially those which can be corrected by intranasal and subcutaneous routes, is introduced in this chapter.

FIG. 213

Traumatic lateral displacement of the nose to the right: *a*, depressed left nasal bone.

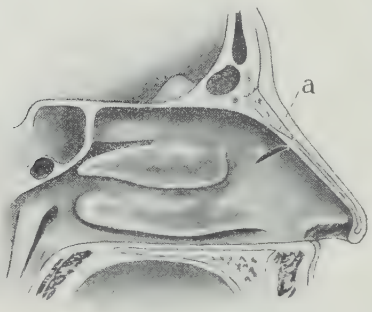
The Twisted or Crooked Nose.—This type of deformity may be due to the congenital maldevelopment of the structures of the nose and face, but it is generally caused by external violence to one side of the nose, which results in an irregular lateral displacement of the septum and tip of the nose. The nasal bone upon the side receiving the blow may also be dislocated laterally, or depressed (Fig. 213, *a*).

The Author's Operation.—*First Operation.*—To correct this deformity the septum should first be straightened by the submucous resection of

the deformed cartilage and perpendicular plate of the ethmoid bone. The cartilage forming the ridge of the nose should be left wide, as it will be needed in the third operation. If the vomer is deformed it should also be included in the submucous resection.

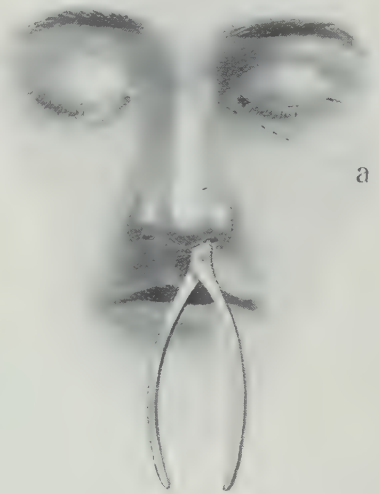
Second Operation.—The depressed nasal bone (Fig. 213, *a*) should be fractured from its attachment and reset in its normal position. This should be done two or more weeks after the submucous resection. The technique is as follows:

Fig. 214



The intranasal incision at the tip of the left nasal bone. One blade of the steel forceps is inserted through this between the skin and the nasal bone, the other grasps the tissue anterior to the middle turbinated body (*a*).

Fig. 215



The Steel septum forceps grasping the nasal bone (*a*) to fracture it preliminary to resetting in its normal position.

An intranasal incision should be made with a small scalpel through the mucous membrane of the outer and anterior wall of the nose at the inferior border of the nasal bone (Fig. 214, *a*). Hajek's semisharp septum periosteal elevator should then be introduced through the incision, and the skin and periosteum over the nasal bone stripped loose.

The Steel, Asch, or other stout septum forceps should be introduced into the nostril thus prepared, and one blade insinuated through the incision and between the skin and nasal bone, while the other remains free in the nose (Fig. 215).

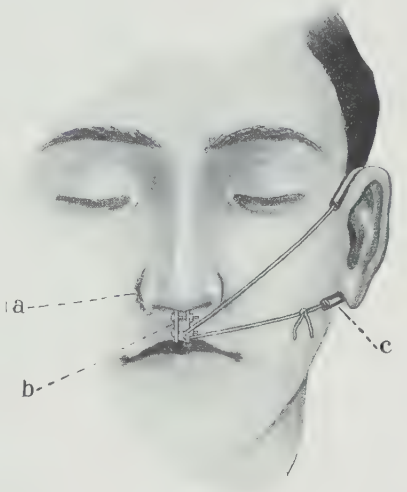
The nasal bone should then be firmly grasped between the blades of the forceps, and rotated upon the axis of the blades, and the nasal bone completely fractured from its attachments.

The nasal bone should be reset in its normal position and held there by means of Carter's nasal splint, or an intranasal cotton tampon impregnated with powdered bismuth until union takes place; this may be removed in three or four days. Carter's nasal splint, is, however, the best device for this purpose.

Third Operation.—At a subsequent time the union of the septal cartilage with the nasal bones should be overcome *via* the nasal route. The incision should be made through the mucous membrane and cartilage, beginning at the junction of the nasal bones and the cartilaginous septum just beneath the skin at the ridge of the nose. If the cartilage has previously been removed by submucous resection the lower end of the incision should extend to the area of the removed cartilage (Fig. 216). The mucous membrane on the opposite side of the cartilage need not be included in the incision unless greater mobility is to be thereby gained. The incision should extend entirely through the cartilage, which otherwise will not remain in the new position in which it is to be placed.

Push the tip of the nose forcibly beyond the median line, and note whether it tends to return to its former malposition. If it does, ascertain where the point or points of resiliency still exist. If at the floor of the nose, sever the attachment at this point and so continue until the whole portion of the nose below the nasal bones remains in the median line without support. If the vomer is still present it should be fractured from the premaxillary bone by twisting it with the Asch septum forceps until it is perfectly pliable. Having done this, the vomer should be reset and supported in such a position as to favor the correction of the external deformity.

FIG. 216



The nasal splint (*b*) held in position by the anchor cord (*c*) fixed behind the ear. *a*, the crescentic area left after the nose is reset in the median line.

If the skin and cartilage at the ala on the side toward which the tip of the nose formerly inclined interferes with the displacement toward the opposite side, an incision should be made at the junction of the ala and skin of the cheek, and the ala and cartilage elevated from the bone at the margin of the pyriform opening until they no longer interfere with the lateral displacement of the nose. When the tip of the nose is displaced laterally a crescentic wound is left (Fig. 216, *a*). This area may be allowed to heal by granulation or it may be covered by a Thiersch graft, after two or three days, when new granulation tissue has covered the denuded area.

The whole lower portion of the nose, being thus rendered perfectly mobile, should be fixed in the median line, or rather beyond it, as the tendency will be for it to return to its former position. To hold the nose in its new position the author's septum clamp (shown in Fig. 216, *b*) is placed astride the cartilage along the ridge of the nose, the blades approxi-

mated by tightening the milled screws, and a stout linen cord looped over the distal end of the clamp. The other end is then looped behind the ear and the knot drawn until the nose assumes the position desired by the surgeon. The portion of the thread which goes behind the auricle should be passed through a small rubber tube to prevent it from cutting the skin (*c*). This splint should be worn for one week or even longer to allow union of the tissues in the new position. The tension of the loop should be regulated daily. The splint may be removed and reinserted if it becomes necessary to cleanse the nasal chambers. A bandage should be placed around the head to hold the auricle in position.

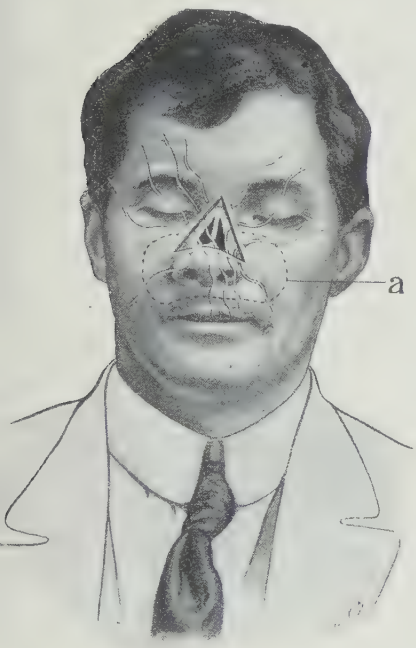
FIG. 217

Traumatic deformity of the face; the nose and upper lip dislocated downward in a cyclone disaster.

Dislocated Nose.—Violent force, as a cyclone, may cause the lower portion of the nose and the upper lip to be dislocated downward, as shown in Fig. 217. In this case the nose and upper lip were dislocated downward and had united to the tissues beneath. The openings of the nostrils were on a level with the gums, hence the nostrils were almost completely obstructed. The triangular space shown in Fig. 218 was filled with scar tissue, which is shown dissected away with the skin. The upper lip and cheeks were freely dissected loose and the sutures introduced

beginning at the lower angles of the triangular wound. When the sutures were tied the end of the nose and upper lip were drawn into their natural position. Large rubber drainage tubes were then placed in each nasal chamber for three or four days to prevent adhesions and to sustain the nose in its new position. Irrigation with warm normal salt solution was continued until crusts ceased to form. The final result is shown in Fig. 219, in which the nose is greatly shortened.

Fig 218



Operation for the correction of traumatic dislocation of the nose and upper lip: *a*, the area of tissue dissected loose to permit the displacement of the lip and nose. (Author's case.)

Fig. 219

The result of the plastic operation.

The Aquiline or Hump Nose.—Occasionally the possessor of an aquiline nose, especially if the "hump" is quite prominent, is anxious to have the "hump" removed or reduced. This may be done by external incision, or subcutaneously through the nose. Preference should be given to the intranasal route, because it does not produce a visible scar. I cannot conceive of a deformity of this kind that may not be removed *via* the nasal chambers.

External Operation.—If, however, an external operation is preferred, it should be made in the median line of the nose, over the area of deformity. The skin and the periosteum should then be raised on either side, exposing the prominent nasal bones (Fig. 220). The elevated flaps

should be pulled aside with retractors by an assistant. The surgeon should then carefully remove enough of the projecting nasal bones to reduce the deformity to the degree suggested by the patient. The cutanoperiosteal flaps should then be coapted with adhesive strips and allowed to heal by first intention. Stitches should be avoided if possible, as they add to the prominence of the linear scar in the median line of the nose. The adhesive strips may be removed at the end of from three to five days.

FIG. 220

External operation for the removal of the "hump" from the nose.

Intranasal Operation by the Author's Method.—This method of operating should usually be chosen, as it is not attended with an external scar.

Technique.—(*a*) Local or general anesthesia.

(*b*) Thoroughly irrigate the nasal chambers with warm salt or boric acid solution, or otherwise clear the nose of crusts, secretions, and bacteria.

(*c*) Introduce a scalpel into one nasal chamber until its point reaches the lower border of the nasal bone, then make an incision through the mucous membrane and pass the blade of the knife between the nasal bone and the skin covering it (Fig. 214, *a*).

(*d*) Withdraw the knife and introduce a small elevator of the Freer type and separate the skin from the anterior portions of both nasal bones.

(*e*) Withdraw the elevator and introduce the author's reverse chisel (Fig. 221), and with a downward and forward pull (parallel with the ridge of the nose) shave the anterior borders of the nasal bones until the hump is sufficiently reduced (Fig. 222).

(*f*) The skin over the operative field should be gently massaged every three hours to prevent the deposit and organization of a plastic exudate over the bones previously reduced. Heat, or the application of the leukodescent light over the nose, will also control the amount of inflammatory deposit.

(*g*) Compression with a nasal pad and a roller bandage may be used instead of massage, heat, etc., if these are not available.

The Long or Drooping Nose.—This type of nose is occasionally seen. I have twice corrected the deformity. The method pursued by me has been the resection of a wedge-shaped piece of the nasal septum through the nasal orifice.

Technique.—(*a*) Cocaine anesthesia as for the submucous resection of the septum.

(*b*) Make two incisions through the mucous membrane and cartilage to the opposite mucous membrane, as shown in Fig. 223. Connect the

divergent ends of the incisions at the ridge of the nose by an intersecting incision, which should separate the cartilage from the skin of the nasal ridge.

(*c*) Remove the triangular piece of cartilage with an elevator.

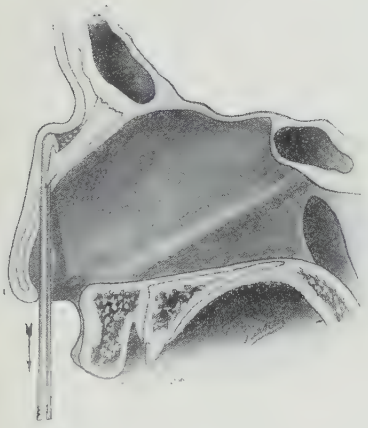
(*d*) Draw the whole end of the nose upward with a sling composed of strips of adhesive plaster.

(*e*) At the end of from four to eight days remove the adhesive strips.

FIG. 221

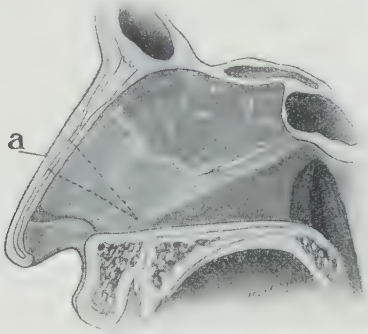
The author's reverse chisel for subcutaneous correction of nasal deformities.

FIG. 222



The author's method of removing the "hump" from an excessively aquiline nose.

FIG. 223



The author's method of shortening a long overhanging nose. The triangular piece of cartilage (*a*) is removed *via* the nostril and the gap closed by lifting the tip of the nose upward and securing it in place with adhesive straps applied externally. At the end of four to eight days the straps are removed, union being complete.

After-treatment.—To prevent local infection and assure firm union of the septal wound, introduce pledgets of cotton saturated with a 10 per cent. glycerin solution of ichthyol every four hours for three days. The ichthyol is antiseptic and the glycerin promotes osmosis of serum from the bloodvessels which washes away any bacteria that chance to invade the region of the wound.

Remarks.—When the nose is shortened in this way there is no redundancy of skin, as it contracts until the normal tension is established.

Paraffin Injection.—The use of paraffin has passed the stage of experimentation, and is, in fact, a well-established procedure in surgery, especially in nasal work. It is used principally in the correction of congenital

and acquired deficiencies. One of the most important locations for its use for cosmetic purposes is the bridge of the nose, in the characteristic saddle nose. The various locations and conditions where paraffin has been used about the ear, nose, and throat are as follows:

1. Saddle noses following trauma, syphilis, and cretinism. The case shown in Fig. 224 was due to cretinism. The patient is a graduate of a High School of Chicago, and is an intelligent young woman twenty-four years old.

FIG. 224

Congenital saddle nose due to cretinism.

2. Following operations on the frontal sinus to correct the frontal deformity.

3. To overcome the collapse of the alæ nasi.

4. Intranasal injections into the inferior turbinated body in rhinitis atrophica.

5. Following resection of the superior maxillæ to fill up the defect.

6. Partial reconstruction of the inferior maxillæ following necrosis and resection for malignancy.

7. Secondary repair of harelip, when there is great atrophy of the premaxillary bone.

8. In the region of the postnasal space when defect of speech (rhinolalia pata) results from the operation for cleft or immovable palate.

9. Following mastoid operations to fill up large retro-auricular deformities.

The paraffin may be injected either hot or cold, depending upon the firmness of the paraffin required. The hot becomes the firmer after cooling, hence for the correction of a saddle nose the hot paraffin may be used, although the cold is preferable and less dangerous. Cold paraffin should be used intranasally to build up the inferior turbinated body.

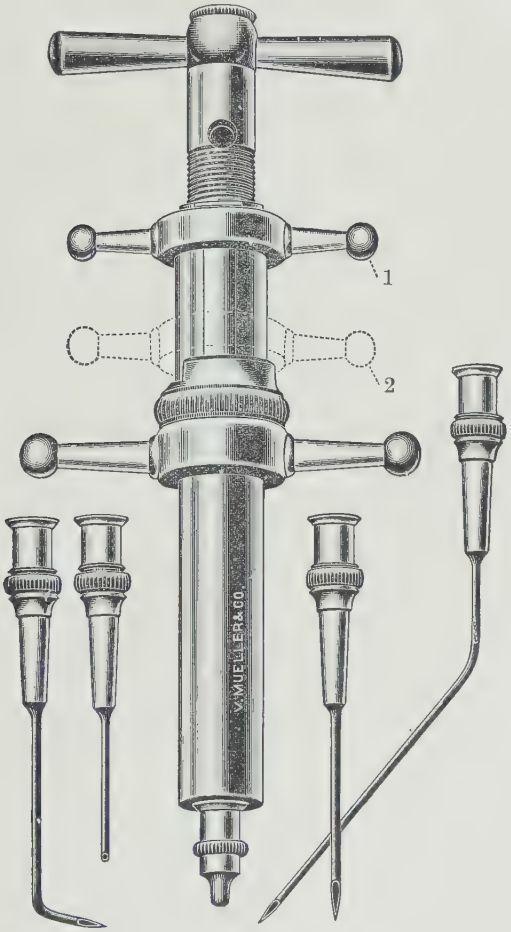
The instrument required for these procedures is the paraffin syringe (Fig. 225), which may be used for either the hot or cold paraffin.

The paraffin which is to be injected hot is kept in an ounce bottle, the cold in tubes, which are especially prepared for the syringe.

Technique.—If hot paraffin is to be used, place the bottle in boiling water until the contents liquefies, then fill the syringe with it by withdrawing the piston. Then turn the screw head from left to right until the paraffin comes out of the needle in the shape of a thread. Then introduce the needle into the cavity to be injected and continue to turn the piston slowly until the desired amount has been injected. If the cold paraffin is used, it is not necessary to heat it. Insert a cylinder of it

in the syringe and by turning the screw handle of the syringe force the paraffin through the needle into the subcutaneous tissue until the desired amount is deposited. An assistant should turn the screw handle while the surgeon molds the paraffin beneath the skin. The needle should be introduced one-half inch above the upper limit of the depression to avoid the subsequent extrusion of the paraffin.

Fig. 225



Beck's paraffin syringe.

The opening caused by the introduction of the needle is sealed up by a small pledget of cotton moistened with collodion. Considerable bleeding from this point sometimes occurs, and pressure should be applied for a few minutes or until bleeding ceases. It should then be sealed up.

In submucous injections an antiseptic gauze pad should be inserted for a few hours to control the slight oozing and prevent possible infection.

To prevent the spread of paraffin into the neighboring tissues, as the eyelid, in injecting the bridge of the nose, where a great deal of loose areolar tissue is present, it is good practice to have an assistant hold his fingers firmly against the underlying bone on each side of the area to be injected. Before complete hardening of the paraffin takes place, it should be molded to the desired form. The operation may be performed in one or more sittings according to the discretion of the surgeon. It is safer to inject paraffin at several sittings, because one can always add to the amount, but if too much is injected, it is very difficult to remove it.

The complications following injection are:

1. Infection.
2. Hematoma.
3. Embolism.

Each is comparatively rare. The first complication should be guarded against by observing the strictest antiseptic precautions in sterilizing the paraffin, the syringe, the field of operation, and the hands of the operator and assistants.

Hematoma is controlled by pressure, and if it is very large, it may require evacuation, followed by the application of ice and afterward warm applications to cause absorption.

Embolism has been reported twice in the literature, and in both cases ether was injected hypodermically in dram doses. The operation was successful.

The change that takes place in the injected mass is at first a reactive inflammation forming a fibrous capsule, which soon throws out trabeculæ, which ramify the paraffin mass in all directions, until the latter is held in a meshwork of fibrous tissue. It has been found that after a period of six months or a year considerable paraffin has been absorbed, connective tissue having taken its place. In cases injected several years ago the mass has remained about the same size as when first injected. Such a mass after organization is known as paraffinoma. Exposure to excessive heat, as in foundries, and during high and long-continued fevers, as typhoid and pneumonia, has very little effect on the injected mass; traumatism, however, such as a blow on the nose, has changed the contour and location of the paraffin mass.

Special Technique.—Saddle nose and other malformations of the nose.

1. To fill up a defect: Thoroughly prepare the field of operation and place the patient in a recumbent posture. Introduce the needle of the syringe beneath the skin from above and fill up the defect either at one or in several sittings. Do not dissect the skin loose from the underlying bone, as a hematoma will form and may become infected.

Stop oozing by compression, and after the paraffin is injected, close the puncture with collodion cotton. No after-treatment is required (Figs. 226, 227, and 228).

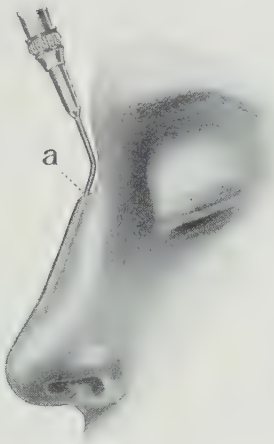
2. To stiffen collapsed alæ of the nose: The needle point is introduced between the cartilage and the skin along the whole alar area; inject a very small particle of paraffin to bring about the desired effect.

3. To reconstruct the inferior turbinated body following atrophic rhinitis: Thoroughly cleanse the mucous membrane of pus and crusts. Anesthetize with a 5 per cent. solution of cocaine that portion of the turbinated body which is to be penetrated by the needle. If a stronger solution is used, too much contraction will follow. Inject slowly by turning the screw head from left to right, and as the needle is withdrawn a track of paraffin is left along the course of the needle. Apply an intranasal tampon for a few hours. Keep the parts thoroughly clean. It is at times necessary to reinject the different areas. The mucous membrane may be too thin from atrophy to retain the paraffin.

Fig. 226

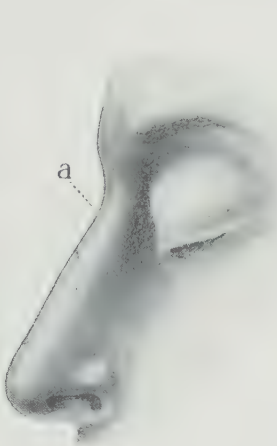
Traumatic saddle nose: *a*, point at which the needle should be introduced.

Fig. 227



a, showing the needle introduced one-half inch above the upper margin of the deformity.

Fig. 228



Showing the depression filled with paraffin.

4. To correct the deformity following the frontal sinus operation: Cleanse the skin, introduce the needle point in different directions, and insert the paraffin, as the scars are usually very firm and are not easily elevated. Extreme care must be taken not to pass the needle too deep, as the posterior table may be injured.

5. To correct the defects after the mastoid operation: Make a preliminary dissection of the skin, which is usually firmly adherent to the bone. This may be done by making a small incision through which a small elevator is introduced. Squeeze out all the blood and fill the cavity with paraffin. Close the incision by one or two horsehair sutures or adhesive plaster.

6. To correct defects caused by excision or disease of the upper or lower jaw: One must be guided by the disease present and apply the principles mentioned above. One of the most common defects is caused by necrosis following decayed teeth, and secondary periostitis.

COLLAPSE OF THE ALÆ NASI

Etiology.—Collapse of the wings of the nose is sometimes associated with prolonged nasal obstruction and mouth breathing. Lambert Lack suggests that the open mouth, with the resultant drag on the sides of the nose, and the atrophy of the dilator muscles of the alæ from prolonged disuse are the chief factors in producing this condition. The condition may also be due to senile changes.

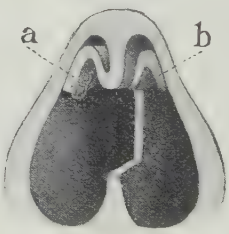
Symptoms.—The nasal orifices are greatly narrowed, often mere slits, and the alæ are flaccid and collapse upon inspiration. Under normal conditions the alæ dilate and are firm and resilient.

Treatment.—If the collapse is due to unilateral nasal obstruction, the cause of this obstruction should be removed. In some instances this is followed by a cessation of the collapse, especially if the condition is of comparatively recent occurrence. In older cases the collapse of the alæ persists.

Fig. 229

Walsham's operation: Collapse of the ala nasi corrected by a roll of mucous membrane from the septum.

Fig. 230



Schema showing Lambert Lack's method of overcoming collapse of the alæ nasi. The flaps *a* and *b* are made from the septum, and are about one-eighth of an inch wide. The upper surface of each flap is denuded of mucous membrane, and the nasal walls against which they are reflected are curetted to encourage adhesion. The flaps are held in position by a single suture in each flap.

Lack advises that the patient practise dilating the nostrils against resistance. He urges them to stand before a mirror for five or ten minutes twice a day and lightly compress the alæ with the thumb and finger, and dilate the nostrils to their fullest extent. This method gives results in recent cases, whereas in chronic ones, in which there is complete paralysis of the dilator muscles, it is ineffective. (See Paraffin Injections.)

Soft and hard rubber rings (Guye) have been worn to keep the nostrils patulous, but the discomfort attending their use is quite objectionable.

Walsham recommends elevating a narrow strip of mucous membrane from the anterior portion of the septum with an attachment above, and

then rolling it into a mass at the upper angle of the nostril (Fig. 229), stitching it in position where it mechanically prevents the collapse of the ala. Lambert Lack suggests the most ingenious and apparently the best method in obstinate and troublesome cases. "The operation consists in turning up a piece of cartilage as well as mucous membrane from the septum and stitching it across the top of the nostril at right angles to the septum, so as to push the ala forcibly outward. An L-shaped incision is made through the mucous membrane on one side of the nasal septum and the mucous membrane detached from the cartilage. A small piece of mucous membrane at the top, and extending a little on to the outer wall of the nostril, is then cut away so as to leave a bare surface, to which the cartilaginous flap becomes adherent. The knife is then passed completely through the septum, and a small quadrilateral piece of the septum, with the mucous membrane on the opposite side left intact, is cut. This flap should be about one-half inch long and one-eighth inch broad. It is fixed to the roof and outer wall of the nostril with a single stitch. A similar piece is then turned up on the other side (Fig. 230)."

CHAPTER XVI

CHRONIC GRANULOMATA OF THE NOSE, THROAT, AND EAR

LUPUS OF THE NOSE

Definition.—Lupus vulgaris is a chronic disease of the skin and mucous membrane, characterized by the formation of nodules of granulation tissue. It passes through a number of phases, and terminates by ulceration or atrophy with scar formation. The cause of the disease is the tubercle bacillus.

Etiology.—Lupus of the nose and upper air passages is practically always associated with, or is secondary to, a lupoid condition of the skin of the face. Rare instances of primary lupus of the pharynx and larynx have been reported by Emil Mayer, Rubenstein, and others.

Females are more often affected than males, and it is more common in the country than in the city. It is most common in middle life, though it occurs at all ages. An abraded or diseased mucous membrane predisposes to its development. While lupus is due to the tubercle bacillus, there is a clinical distinction between it and tuberculous ulceration. Lupus is slow and insidious in its development, and is not necessarily associated with pulmonary tuberculosis. It has a tendency to heal, cicatrize, and recur, and does not often result in death from pulmonary involvement.

Symptoms.—Lupus of the nose generally begins on the anterior portion of the cartilaginous septum or upon the skin around the nasal orifice. It may spread from the septum to the inner wall of the ala. It appears as small nodules which coalesce and ulcerate, and it may disappear by absorption. The reparative process takes place but feebly at the margins of the ulcer, thus forming a pale-bluish, smooth cicatrix. The ulcers reappear and then disappear. This process may continue for years without spreading to other regions. The nodules are firm and well marked. The disease rarely attacks the cartilage and never the bones. One or both nostrils may be affected, and there may or may not be stenosis. The discharge varies with the stage of ulceration. At the onset it is thin and watery, and later becomes thick and even fetid, especially after crusts appear. Pain and tenderness may be present, though I have seen cases in which they were absent. Itching is sometimes complained of.

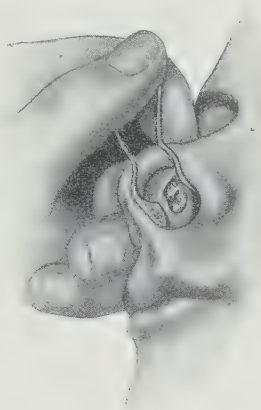
Deformity may be present if the alæ are involved; when limited to the septum it is rarely present.

I reported a case which was under the care of the late Dr. Max Thorner, of Cincinnati, for about four years. Subsequently it was

under my care for about the same time, and is now under the care of a *confrère*, who informs me that the ulcerous condition has yielded to applications of the high-frequency currents of electricity. It should be noted, however, that the patient spent the winter in the South, and that while under my care the ulcer disappeared spontaneously each summer. (The case has more recently been reported as cured with bismuth paste, thus conclusively proving the apparent cures to have been remissions rather than cures.)

The case has thus been under nearly constant observation for about twenty years. The patient is about forty-seven years of age, and is in robust health, never having had any pulmonary symptoms. She says her brother has a similar condition in his nose. I inoculated a guinea-pig with the tissue removed by curettage, and in six weeks the postmortem showed extensive tuberculous lesions in the neighboring glands and in the mesentery. The ulcer (Fig. 231) was superficial, irregular in outline, and had a somewhat nodular surface covered with crusts. It bled easily upon probing, was painless, and disappeared during the summer months, leaving a whitened, rather smooth, cicatricial surface. It reappeared in the autumn of each year, only to disappear the following summer. This case seems to be primary in the nose, and shows little or no tendency to spread. There is no lupous lesion of the skin.

Fig. 231



Author's case of lupus of the cartilaginous portion of the septum.

Treatment.—Spontaneous recovery may take place, though this is exceptional. It does not readily yield to treatment. Local escharotics, curettage, the galvanocautery, serumtherapy, surgical removal, and radiotherapy, have all been tried with varying success.

The escharotics which have been used are lactic acid, carbolic acid, chromic acid, the arsenic paste, and other destructive chemical agents. Curettage has also been tried, usually with little result. Curettage followed by the local application of an escharotic affords somewhat better results, though even this is far from satisfactory. Local cauterization with the galvanocautery is a procedure often resorted to, though usually with negative results. Serumtherapy has been attended with some success, but its limited use, thus far, does not afford a sufficient basis for a fair conclusion as to its efficacy. Surgical removal by excision of the diseased area is also as ineffectual as the measures just mentioned. Radiotherapy has proved of the greatest value in these cases.

Radiotherapy.—Radiotherapy consists in the local application of heat and light rays endowed with biochemical energy. Generally speaking,

the blue-violet rays are the most potent, though the ultra-violet and x-rays are also effective. The energy may be applied by the x-ray tube, the Finsen apparatus, the leukodescent lamp, and radium.

LUPUS OF THE PHARYNX AND LARYNX

Posey and Wright quote H. Myngid's report of 20 patients with lupus of the skin in which the larynx was affected in 10 to 20 per cent. of the cases. Fifteen of the cases were females and 5 were males. Hunt in 411 cases of external lupus found either the pharynx, larynx, or the nose involved in 20 per cent. of the number. In 173 cases of lupus of the mucous membranes in Doutrelpont's clinic, only 6 were free from cutaneous lesions. The nose was affected in 75 cases, the palate in 31 cases, and the larynx in 13 cases. The lesion often appears before puberty. (See Lupus of the Nose for a more general discussion of lupus.)

LUPUS OF THE AURICLE

Lupus of the auricle manifests itself in all the forms found in other parts of the body, namely, hypertrophic, macular, papillary, and ulcerous, and is usually an extension from the face.

It begins with tubercles the size of a pinhead or larger, which are brownish in color, and slightly scaly on their surface. They are arranged in groups, and are surrounded by a slight efflorescence. The skin is contracted around the diseased areas. The scarred appearance is due to the deep penetration of the tubercles. Keloid formations are quite common.

The ulcerous type is rare and is characterized by ulcerations covered with thick crusts beneath which there is a spongy base. The edges of the ulcers are undermined and pale, with an occasional typical nodule.

Treatment.—The treatment of lupus has been so uniformly successful under the Finsen phototherapy, the Röntgen-ray, and the leukodescent light that the older methods of treatment have become almost obsolete.

Hollander reports excellent results following the application of hot air to the diseased surfaces. The method is worthy of trial, especially if the Finsen, Röntgen-ray, and leukodescent light treatments are not available.

If simpler methods of treatment fail, the lupous areas may be excised and a subsequent plastic operation performed to overcome the deformity resulting from the primary operation. Another form of treatment, much in vogue in Europe, is first to curette the granulating areas and then apply a paste, the base of which is arsenic. This mode of treatment has been much vaunted in this country by charlatans as a means of curing cancer, most of the cancerous cases being, however, one or the other types of lupus heretofore mentioned.

TUBERCULOSIS OF THE NOSE

Tuberculous infection of the nose is characterized by either a low-grade, slightly depressed ulcer on the anterior portion of the septum or floor of the nose, or a sessile, wart-like tumor in which the tubercle bacilli are present.

Tuberculous lesions of the nose may be primary or secondary to a similar process in the lungs. It is generally secondary, though cases are not rare in which the processes are limited to the nose.

Varieties.—(*a*) Superficial ulceration; (*b*) wart-like or sessile tumors. The superficial ulcers are the most common.

The wart-like growths are hyperplastic, and, like the ulcerous variety, bleed easily. The removal of either variety is followed by rather slow healing and by subsequent recurrence.

The complications are perforation of the septum, with extension to the skin of the upper lip, and in extremely rare instances to the nasal accessory sinuses. Kyle suggests that the low resistance of the tissues affords a suitable soil for all forms of microörganisms of chronic granulomata. The treatment consists in curettage and the application of arsenical paste. The ulcer or tumor should be anesthetized with a 5 to 10 per cent. solution of cocaine, after which the diseased area should be thoroughly curetted. A light application of the arsenical paste may then be made to insure the destruction of remaining fragments of tuberculous tissue. The radiant energy of the leukodescent lamp, Finsen light, or some other source of radiant energy may be tried, although I am not informed as to their beneficial effects in this type of tuberculosis.

In spite of all forms of treatment, there is a strong tendency for the tuberculous lesion to persist, and if it disappears, to return.

TUBERCULOSIS OF THE PHARYNX AND THE FAUCES

Tuberculosis of the pharynx and fauces is rare and is probably always secondary to pulmonary or laryngeal tuberculosis. It is usually associated with, and is probably an extension from, tuberculous laryngitis. It has no point of attack, but may begin in the soft palate, uvula, tonsils, lingual tonsils, or the pharyngeal mucosa. Unlike nasal tuberculosis, it tends to spread to adjacent parts.

The part affected presents a worm-eaten appearance, the ulcers being surrounded by an area of congestion. The ulcers are superficial and covered with a dirty grayish secretion. They bleed easily upon probe pressure. There is little or no induration except at the borders of old chronic ulcers. When the lingual or faucial tonsils are the seat of ulceration the depth of the ulcer is great; even the whole tonsil may be destroyed. Cases are reported in which the faucial tonsils were the seat of primary infection and infiltration. It is, perhaps, impossible to estimate the proportion of cases that are primary in the tonsils, though it is perhaps

larger than is generally supposed. In other portions of the pharynx and fauces it is rarely primary. The infection occurs either through the lymph channels or by contact of the infected sputum with the mucous membrane.

Symptoms.—The symptoms vary with the anatomical location and extent of the lesion. If the soft palate is involved, the proper approximation of the palatal muscles to the posterior wall of the pharynx is interfered with, and fluids and solid food may enter the nose upon deglutition. The same condition allows the secretions to accumulate and dry in this portion of the pharynx, which leads to hawking and nausea in the effort to dislodge it. An infiltration of the uvula may cause pain and a tickling cough. As the secretions are thick and the parts often exceedingly painful upon movements, the secretions are often allowed to accumulate. The voice is muffled and hoarse, or aphonic.

Diagnosis.—Syphilis is about the only disease with which tuberculosis of the pharynx may be confounded. The following tables adapted from Lennox Browne will aid in the diagnosis:

Tuberculous ulcers.	Syphilitic ulcers.
1. Superficial moth-eaten surface.	1. Deep red and angry surface.
2. Mildly red areola.	2. Angry red areola.
3. Ragged, ill-defined edges.	3. Sharply cut edges.
4. Indistinct demarcations.	4. Distinct demarcations.
5. Grayish, ropy secretion.	5. Purulent yellow secretion.
6. Scanty secretion.	6. Profuse secretion.

Prognosis.—The prognosis is grave. In those cases in which it is primary in the tonsils it is not serious. When we remember that tuberculosis of the pharynx is nearly always secondary to pulmonary involvement the gravity of the disease is apparent. Kanasugi regards pharyngeal tuberculosis as being more grave than any other localized type, and the primary more than the secondary.

Treatment.—Curettage followed by the application of pure lactic acid is a common form of treatment. It is doubtful if climatic or outdoor treatment is as effective, as the pulmonary involvement is usually well advanced. Forced feeding on raw eggs and milk should be a part of the treatment of all tuberculous diseases when there is loss of weight and strength. The local application of a 2 to 10 per cent. solution of formaldehyde should be tried as in laryngeal tuberculosis. The pain should be controlled by the local application of cocaine, the administration of opiates, or the leukodescent light or other radiant energy. Painful deglutition is relieved by the application of cocaine immediately before meals.

TUBERCULOSIS OF THE LARYNX

Synonyms.—Consumption of the larynx; consumption of the throat; laryngeal phthisis; tuberculous laryngitis.

Definition.—Tuberculosis of the larynx may be primary or secondary, and is characterized by an infiltration of the glands and connective tissue of the larynx. It gives rise to dysphagia, aphonia, and dyspnea.

Etiology.—The view that laryngeal tuberculosis is always secondary is held by almost all observers, and is proved by the findings of autopsies, there being very few recorded cases of death by laryngeal tuberculosis in which either a healed or active pulmonary involvement has not been found. The opponents of this view are very few in number, the most prominent of them being Dr. Gleitsmann, whose researches have been extensive, and who reports two cases of primary laryngeal and pharyngeal tuberculosis in his own practice which were cured. In the report of his cases, he quotes Demme, E. Fraenkel, Prof. Rebinski, Orth, Coghill, J. S. Cohen, Dehio, and Lancereaux in support of his view.

Goodale has seen many cases of tuberculous laryngitis which he thought were primary, and which for a time seemed to yield to treatment; but the subsequent progress of the disease always proved fatal through the associated pulmonary tuberculosis. It is possible in a suspected instance of tuberculous laryngitis, where the pulmonary signs are negative, that a radiograph may disprove or substantiate the presence of pulmonary tuberculosis. Demme, in 1883, reported the case of a boy, aged four and one-half years, who died of tuberculous meningitis; the necropsy showed the presence of laryngeal ulceration with tubercle bacilli, the thorax and abdominal organs being at the same time free of tuberculous disease. He says many other cases in which such a condition was suspected have also been recorded; and it may now be considered as an accepted fact that tuberculous disease may not only attack the larynx primarily, but may cause death without the lungs being affected.

The disease is more common in men than women, and occurs especially between the ages of twenty and forty years.

Knight quotes Heinze's statistics, and adds that of the laryngeal lesions more than one-half were ulcerative, a proportion confirmed by the Brompton Consumption Hospital, nearly twice as large a percentage as that given by many other investigators. The mode of invasion of the larynx is either by direct infection through the inspired air or by the expectorated sputum, or indirectly by conveyance of bacilli from the tuberculous foci in the lungs through the blood current or lymph channels, which is doubtless the more frequent route. If the contrary were true, tuberculous laryngitis would be much less rare than it is. The apparent immunity of the larynx against primary infection is difficult to explain. There is no essential difference between the mucous membrane of the larynx and the nose and other portions of the upper respiratory tract, excepting the pharynx. The mucosa of the nose is more exposed to the irritating influence of the atmosphere, and to trauma from picking crusts from the vestibule, and in this respect the abrasions offer a favorable site for the infection; the larynx is also subject to abrasions in the course of chronic laryngitis and in excessive use of the voice, but it remains to be proved that under these conditions it becomes the seat of primary tuberculosis. Shurley contends that the ventricles of the larynx afford a sheltered, quiet place for the development of the tubercle bacilli, and that in spite of this fact they do not readily develop here. The hidden recesses of the crypts of the tonsils also afford an ideal place for the

growth of the bacilli, and, according to Mayo, 8 per cent. of all tonsils removed by him are tuberculous. Robertson's statistics support Mayo's. There is the necessary temperature, quiet, and protection from the currents of air to favor such a process. The tonsils are undoubtedly a common source of infection. Having gained entrance to the lymphatic circulation by this route, they travel downward to the lymphatic glands of the anterior triangle of the neck, thence to the lymphatic glands of bronchial tubes, and from there to the substance of the lung. I believe that the explanation of the apparent infrequent primary involvement of the larynx is to be found in inherent resistance of all mucous membranes to the invasion of the tuberculous germs, and that the exceptions to the rule are in the nasal mucous membrane of the anterior portion of the cartilaginous septum, and the mucosa of the tonsil crypts, where the abrasions are so often present, and where the conditions are exceptionally favorable for the growth of the bacilli. The site for the tuberculous infection of the nose is at the point where it is or may be daily denuded of its epithelial covering, and where the deposit of tubercle bacilli is abundant. It would be strange, indeed, if tuberculous infection did not occur under these circumstances. The tonsillar crypts form ideal sites for the growth of the bacilli, being warm, practically without motion, and plugged with secretion, food, and desquamated epithelium. In these hidden recesses the bacilli flourish and remain constantly in contact with the mucous membrane. The crypts are also the site of frequent inflammations, during which the epithelium may be impaired, thus affording a favorable condition for the invasion of the tubercle bacilli into deeper lymphatic tissue. Indeed, during inflammations the intercellular spaces become larger and permit the bacilli to pass through. It is more than probable that when the bacilli are indefinitely lodged on a mucous membrane they may penetrate through these spaces without an abrasion being present. The favorable conditions existing in the nose and tonsils are not present in the larynx, hence the tubercle bacilli rarely primarily infect the larynx. When, however, pulmonary tuberculosis is established, and the expectorated sputum constantly bathes the laryngeal mucous membrane, the conditions for infection are much more favorable. The constant presence of the bacilli, the mechanical irritation, the abrasions produced by coughing, and the lowered resistance of the cellular structures in general combine to favor such an infection. It is probable, therefore, that infection is usually secondary to the pulmonary involvement, and not primary.

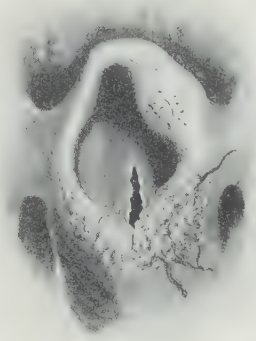
Pathology.—The first apparent change in the larynx may be an ischemia of the mucous membrane. This is usually referred to as an "ashen-gray" color, which is said to be pathognomonic of tuberculosis. It is not always so, however, as it may occur in any general anemia. I have in several instances been enabled to make a diagnosis of tuberculosis by the "ashen-gray" color before the stethoscope showed positive evidences of the disease in the lungs. I referred these cases back to their physician, with the suggestion that the tuberculin test be tried, and in each instance a typical reaction occurred. I contend, therefore,

that while the "ashen-gray" color is not pathognomonic of tuberculosis, it is, nevertheless, a valuable early sign in many cases, especially when there is also a pulse of 100 or more and a daily rise of temperature. It should be stated that the mucous membrane of the larynx is not always of an "ashen-gray" color in tuberculosis, but, on the contrary, it may be quite red, inflamed, and indurated. The vocal cords may be hyperemic and swollen until their identity is lost in the reddened mucous membrane, or they may be lax, flabby, and nodular.

The histological changes occur chiefly in the aryteno-epiglottidean folds, the interarytenoid space, and the epiglottis. The cartilages may become involved, thus giving rise to perichondritis and chondritis. Cicatricial contraction takes place as the healing process progresses. This may give rise to more or less dyspnea.

When the arytenoid cartilage is affected the club-shaped infiltration tumor is present (Fig. 232). When the infiltration extends to the aryteno-epiglottic ligament the picture is quite characteristic of tuberculosis of the larynx.

FIG. 232



Tuberculosis of the larynx. (Author's case.)

The epiglottis is often involved in the process, and when infiltrated presents the turban shape so often referred to. The infiltration may extend to both sides of the larynx or be limited to one. When both are affected the view of the deeper portions of the larynx is hidden. The tendency to ulceration is quite constant. It is rare for a well-advanced case of laryngeal tuberculosis to be free from it. The ulcers may be of any size within the limits of the area involved, and may be superficial or may extend to the cartilages. They may be discrete or confluent, single or multiple, and on one or both sides. When the cartilage is involved by ulceration there is a purulent discharge from the mixed infection. Tuberculous ulcers develop more slowly than syphilitic ulcers, are less destructive, and are followed by less cicatricial contraction.

Symptoms.—The symptoms of an ordinary case of laryngeal tuberculosis are characteristic. As the laryngeal involvement is usually secondary to the pulmonary, the preceding history may afford an excellent index. There is more or less cough, often without expectoration, and there may be a sense of prickling or dryness in the throat. The voice may be hoarse or aphonic, especially when the infiltration is extensive. The dyspnea is in proportion to the degree of infiltration and the cicatricial contraction. Pain may or may not be present. In some cases it is quite severe, and local applications of cocaine and orthoform, or injections of morphine, are necessary to control it. In one of the author's

cases, illustrated in Fig. 232, though the patient is aphonic, and has been for several years, there is no pain. Dyspnea is a constant factor, though not alarming in severity. During the past ten years the patient has gained twenty-six pounds in weight. Difficult or painful deglutition has been a more or less prominent symptom. The laryngoscopic examination shows the lesions described under pathology.

Diagnosis.—Laryngeal tuberculosis must be differentiated from syphilis, carcinoma, and lupus.

Syphilis of the larynx presents a "punched-out" ulcer with a yellowish exudate upon a dark red base. It spreads rapidly. The voice is low-pitched and hoarse, or raucous, but rarely aphonic. Pain is present upon phonation. The tuberculous ulcer is superficial and its base is covered with a grayish exudate. It spreads rather slowly, is painful upon deglutition, and the voice is weak and softly hoarse or aphonic.

In carcinoma the base of the ulcer is raised by the crowding of the deeper infiltration; it is red and constantly painful, and the voice is continuously hoarse.

In lupus there is usually no pain, ulceration, edema, or discharge; dyspnea is slight or absent, the general health good, and a lupoid lesion is usually present upon the skin.

Prognosis.—The prognosis in laryngeal tuberculosis is grave, though not necessarily fatal. According to Harpy there were 14 spontaneous recoveries in 3000 cases. Under appropriate treatment the percentage of recoveries is increased. As a rule, however, the patient may be expected to live only for a comparatively short time—a few months or years. Death may occur from inanition, suffocation, or hemorrhage.

Treatment.—The treatment of laryngeal tuberculosis, excepting the local symptoms, is the same as that of pulmonary tuberculosis. At present the "outdoor" treatment, especially in the earlier stages, is enthusiastically recommended. The buildings should be so arranged that the patients practically live outdoors the year round. While this at first thought seems impossible during the winter months, it is, nevertheless, being done with comparative comfort. The house or tent affords protection from the severe cold and from the winds, while fires make life not only tolerable, but cheerful and comfortable. The object is to keep the patients in a pure circulating atmosphere as much as possible. The whole system is thus invigorated and the lungs are supplied with fresh oxygen. The vital forces are augmented and the reparative processes are often quickly and permanently restored. In mild cases, and in the incipient stage, little or no medicinal treatment is required, the "outdoor" treatment being quite sufficient. In well-advanced cases where there is great infiltration and ulceration of the laryngeal tissues the "outdoor" treatment is as ineffectual as any other.

Innumerable remedies are recommended for the cure and relief of laryngeal tuberculosis, among them being the following:

For the relief of cough: Codeine, $\frac{1}{8}$ to $\frac{1}{2}$ grain every three hours. Morphine sulphate, $\frac{1}{30}$ to $\frac{1}{16}$ grain every three hours.

For the relief of pain: Spraying the larynx with a 0.5 per cent. solu-

tion of cocaine. If there is painful deglutition, a 2 to 8 per cent. solution of cocaine may be applied locally, just before eating. Insufflations of orthoform powder may relieve the pain, is non-poisonous, and its effects last longer than those of cocaine.

For curative effects, Gallagher, Levy, Lockard, and Johnson recommend local applications of formaldehyde to the larynx. Gallagher was one of the first to report beneficial results from this treatment. It should be used in solution, gradually increasing in strength from a 0.5 per cent. to a 10 per cent. solution. The patient may be intrusted with a 1 to 500 solution for home treatment, but greater strengths should be applied by the attending physician.

Gallagher reports excellent results with the following method of treatment:

1. Anesthesia slight.
2. Cleanse, spray with 1 to 3 per cent. formaldehyde solution.
3. Local application, 5 to 10 per cent. formaldehyde.
4. ℞.—Orthoform . . . . . 7 parts
   Aristol . . . . . 1 part
   } insufflation
5. Deep intratracheal injection of
   ℞.—Menthol . . . . . gr. x
   Ol. eucalyptus . . . . . ʒj to ʒij
   Ol. cinnamon . . . . . gtt. j to gtt. x
   Glycerol . . . . . q. s. ad ʒj

The above daily. Curettage is used when deemed necessary.

Menthol is another remedy of positive value. It may be used in combination with camphor and orthoform. Freudenthal uses it in emulsion in the following mixture:

℞.—Menthol . . . . . 1 to 15 parts
Ol. amyg. dulc. . . . . 30 parts
Vitelli ovarum . . . . . 25 parts
Orthoform . . . . . 12½ parts
Aquæ des. . . . . q. s. ad 100 parts

Ft. emulsio.

The above is injected intratracheally and often yields excellent results.

Lactic acid has had and still has its advocates. Begin with a 10 per cent. solution and increase to 75 per cent., or even to full strength. It should only be used when there are ulcerations, or after curettement. It should be rubbed into the ulcerated or raw surface with a cotton-wound applicator at intervals of from five to ten days. The pain is severe and continues for four or five hours.

Radiotherapy.—According to Gleitsmann, the Finsen light and the ultra-violet rays are less penetrating than the Röntgen rays, and yet the latter has not produced the expected results in laryngeal diseases. The bacilli are at first increased, and only after a prolonged use of a low vacuum tube is improvement noticeable. The Cooper Hewitt light, or mercurial waves, the search light, the actinolight, and the leukodescent lamp may be used to relieve the pain, and in some instances actual improvement follows. It is too early to predict marked curative power from these sources. I have used the leukodescent lamp, but my

experience with it is too limited to state that it does more than relieve the pain. The chief value of the leukodescent lamp is in the blue-violet rays and the radiant heat. These in combination exert a favorable influence in acute catarrhal and suppurative inflammations, hence are of service in combating the mixed infection usually present in tuberculosis. The use of radium as reported by J. C. Beck relieves the pain just as other forms of radiant rays do. The direct rays of the sun, if concentrated, act in much the same way.

Curettage should be limited to the ulcerated areas, while the parts which are simply infiltrated and have an unbroken surface should be carefully avoided. It has been conclusively shown that the infiltrated areas may remain quiescent indefinitely. When the tuberculous ulcer has been curetted, the sluggish process stimulated, and the overlying necrotic tissue removed, the local treatment given in the preceding paragraphs should be continued.

TUBERCULOUS LARYNGITIS IN PREGNANT WOMEN

Lohnberg observed 5 cases in two years. In 2 there was no evidence of tuberculosis elsewhere, and in the others the laryngitis was the principal lesion. This was true in the cases reported by Türck. Lohnberg has collected 21 similar cases from the literature. The evidence is in favor of the assumption that pregnancy affords a predisposition to this affection and whips the latent process to a gallop. Furthermore, he says that every pregnant woman with diffused laryngeal tuberculosis is immediately doomed, and possibly also those with only a single tubercle. The only treatment is the palliative use of menthol-orthoform emulsion, formaldehyde, etc., but these lose their efficacy after a time, and relief is only obtained from morphine and tablets of cocaine.

Pregnant women should be carefully examined on the slightest suspicion of trouble in the throat, and should be placed upon the treatment outlined above, and especially the outdoor treatment. Every woman affected with tuberculosis should be warned that the tuberculous process may be aggravated by pregnancy. It therefore follows that an unmarried woman suffering from tuberculosis should not marry until a cure has been effected.

TUBERCULOSIS OF THE MIDDLE EAR AND MASTOID PROCESS

Tuberculosis of the middle ear may be primary or secondary. A. W. Milligan believes the primary form, especially in young children, is more common than is generally supposed. Secondary tuberculosis of the middle ear is usually a complication of a tuberculous process in some other part of the upper respiratory tract, rather than a complication of a similar disease of the bones, glands, or abdominal viscera. In a series of cases reported some years ago, Milligan found 16 per cent. of all adenoid

cases to be tuberculous. This is a possible explanation of the frequent involvement of the middle ear.

Symptoms.—The symptoms of tuberculosis of the middle ear vary with the acuity, intensity, or the chronicity of the process; also with a simple or a mixed infection.

The acute variety is characterized by some redness of the drum membrane, slight pain, and multiple perforation. The hearing is considerably impaired. The facial nerve may be paralyzed. If the infection becomes mixed, the nature of the disease is obscured by the greater intensity and destructive character of the inflammatory process.

Diagnosis.—The chronic variety, which is the usual form, is readily diagnosticated, as it runs a slow course and is characterized by little impairment of hearing (though this is variable), tinnitus, a sense of fulness in the affected ear or ears, and an almost or quite complete absence of pain. In the early stage there are multiple perforations, each of which is the site of a tubercle which has broken down. Later these coalesce and form larger perforations, which often result in a complete destruction of the membrana tympani.

To confirm the diagnosis, the secretions and the granulation tissue should be examined for the tubercle bacilli and giant cells. Should they not be found, a guinea-pig should be inoculated with some of the tissue, and at the end of five to eight weeks examined for the results of the test. In one of my cases the microscopic findings were negative, but the inoculation experiment was positive. Climatic treatment in Colorado and permanent residence there resulted in an apparent cure.

Milligan draws the following conclusions:

(*a*) A final and exact diagnosis is imperative both from the point of view of prognosis and of treatment.

(*b*) The disease is most frequently found as secondary to a tuberculous process in other regions of the body.

(*c*) Primary tuberculous disease of the middle ear is probably of more frequent occurrence than is usually supposed.

(*d*) The prognosis is always grave, but in a certain proportion of cases suitably planned surgical intervention will eradicate the disease.

(*e*) In many cases it is advisable to conduct the treatment in stages.

(*f*) When less than 10 per cent. of the hearing power remains no attempts should be made to preserve the ear as an organ of sense.

(*g*) When more than 10 per cent. of the hearing power remains in a patient otherwise in apparent health, a definite attempt should be made to preserve the remaining hearing power.

(*h*) When the tuberculous origin of the ear disease has been scientifically demonstrated, the case should be regarded as infectious, and precautions taken accordingly.

Robert Levy, who has had exceptional opportunities to study middle-ear diseases in tuberculous patients in Colorado, summarizes as follows:

Any of the usual affections may affect the tuberculous as well as the non-tuberculous.

The usual modifications of an acute otitis in a tuberculous subject are manifested in the course of the disease.

It is doubtful whether the Bacillus tuberculosis is present as a distinctly etiological factor or as an accident.

Clinical tuberculous otitis occurs with moderate frequency in Colorado, being secondary to lesions of the respiratory organs.

Tuberculous otitis may develop when the general symptoms of tuberculosis have been arrested and the patient's condition is unusually good.

Tubercle bacilli may find their way into the middle ear through the Eustachian tube, the lymph channels, and the blood current.

Unusual care must be exercised in the application of the nasal douche in tuberculous patients. The discharge may be temporarily arrested.

It must be exceedingly rare for miliary tuberculosis to develop from an otitis as the focus of infection.

Serumtherapy is apparently of some, though uncertain value.

Prognosis.—Generally speaking the prognosis is unfavorable. There are, however, numerous exceptions to the rule.

Unfavorable.—(*a*) It is especially unfavorable in acute cases.

(*b*) Rapid destruction of bony tissue of the labyrinth and mastoid process is another unfavorable sign.

(*c*) Mixed infection adds to the destructive nature of the process.

(*d*) Well-advanced pulmonary tuberculosis renders the prognosis unfavorable.

(*e*) Marked general debility from any cause is an unfavorable sign.

More Favorable.—(*a*) In children the disease is often local or secondary to diseased tonsils and cervical glands. The removal of the tonsils and glands, and the diseased centre in the mastoid process is usually followed by complete recovery.

(*b*) In adults otherwise healthy the prognosis under simple treatment is good.

Treatment.—General and climatic treatment must be conscientiously carried out.

Goldstein reports four cases which he considers primary tuberculous infections. All of these cases, he says, were seen more than three years previous to his report; three are still living, and careful physical examination fails to show any tuberculous infection. There were no evidences in the histories of these cases or in their clinical development either of an acquired or hereditary tuberculosis. Of the four cases, three involved the mastoid cells extensively and showed an unusually active and rapid invasion. All of the cases developed from a preëxisting otitis media suppurativa chronica, and appeared to him as direct infection by the Bacillus tuberculosis. In the three cases in which the mastoid operation was performed the wounds healed by firm granulations, and all evidence of tuberculosis ceased with the removal of the local process. This is in direct contrast to the healing of wounds in patients in whom the systemic tuberculous invasion is present. The data which has been furnished in the cases herein reported point to a definitely localized specific infection of the cavum tympani and mastoid cells, with the

characteristic development of a tuberculous process as it occurs in bone tissue, and with the definite demonstration of the Bacillus tuberculosis in one case.

The treatment should be selected with reference to the type of manifestation, the age, and general health of the patient.

(*a*) In primary tuberculosis of the mastoid process, good results may be obtained by the mastoid operation, especially in children. In children it may be necessary to remove the tonsils and cervical glands, as failure to do so subjects the patient to the chance of a return of the process.

(*b*) When the *pulmonary tuberculosis is not advanced*, the mastoid operation is indicated, and may be followed by very satisfactory results. These cases also do well in a suitably selected climate or in tent colonies, with adequate nourishment and with local treatment. The tuberculin treatment is of value if Koch's new tuberculin is given under opsonic control.

(*c*) When the *pulmonary tuberculosis is well advanced*, operative treatment is useless. Even in the more favorable cases, the operation may be followed by only temporary improvement. If the patient is greatly debilitated from any cause, operative treatment is contra-indicated. In such cases the necrotic process usually continues, and the bony walls remain denuded and covered with pus.

(*d*) When there is *mastoid swelling or redness* an early operation for the relief of the abscess is indicated, regardless of the general character of the disease.

(*e*) *Climatic or open-air treatment* and reconstructive remedies should be used in those cases in which there is little or no involvement of the lungs; outdoor air in a *cloudy climate* is recommended.

O. J. Stein recommends the use of formaldehyde, a few minims of which are dropped on a gauze dressing which is placed in the meatus and auricle. This should be covered with a thin layer of cotton and sealed with collodion to prevent external evaporation. The fumes of the formaldehyde penetrate to the diseased area and exert a favorable influence upon it. (See Treatment of Laryngeal Tuberculosis.)

SYPHILIS OF THE NOSE, PHARYNX, FAUCES, AND TONSILS

The fauces and pharynx are second only to the skin as sites for the manifestation of constitutional syphilis, particularly in the secondary stage. This may be accounted for in part by the presence of a large number of lymphoid glands, the excessive friction, and the complex embryological union of tissues in this region.

Congenital syphilis is more common in the pharynx than in the nose. In the cases shown in Figs. 233 and 234, the pharynx and nose were involved. John Mackenzie says that 50 per cent. of the congenital cases develop in the first year of life, $33\frac{1}{3}$ per cent. within the first six months.

Primary lesion of the pharynx and tonsils is second in frequency to that of the genitalia, owing to the number of syphilitic nurses and sexual

perverts, and to the use of unsterilized surgical instruments in office practice. In one of my cases the primary lesion occurred on the left tonsil, which was incised for quinsy by a practitioner who was syphilitic.

When I first saw the patient there was an ugly superficial ulcer with indurated edges on the upper portion of the tonsil. Within a few days the typical secondary rash appeared, thus confirming the diagnosis.

Females are more often affected than males, and one or both tonsils may be the seat of the primary lesion.

The primary lesion is usually of short duration, though when it occurs on the tonsils the inflammation may be so great as to extend the period of ulceration to the second stage. This has been true in some of my cases.

FIG. 233

Syphilitic scars of the fauces and pharynx causing a partial constriction of the isthmus between the epipharynx and mesopharynx. (Author's case.)

FIG. 234

Author's case of congenital syphilis of the nose.

The secondary lesion consists of the usual erythema of the face and body and mucous membranes. It may appear from six to eight weeks after the initial lesion or even as late as several months. The erythematous patches in the throat have been described as ulcerations, though Lennox Browne claimed that they are not true ulcers, but simple abrasions of the surface epithelium.

The tertiary lesions appear from three to twenty-five years after the primary manifestation, and may be ulcerative, gangrenous, or gummatous, and very destructive to both soft and bony tissues.

Symptoms.—The symptoms of the primary stage are ulcers with indurated edges, which cause pain in the ear if the arch of the fauces is affected. If the inflammation extends to the pharyngeal orifice of the

Eustachian tube there is some deafness and tinnitus. The lymphatic glands of the neck are usually enlarged.

In the secondary stage there may be cough or a tickling sensation in the throat. In some cases pain or a dull aching is complained of. Dysphagia and a pseudomembranous angina, accompanied by a slight elevation of temperature, may be present. There may also be erythematous patches on the skin and in the throat, those in the throat often being mistaken for superficial ulcerations. Upon close examination they are found to be mere abrasions or elevations of the superficial epithelium.

In the tertiary stage the odor is characteristic, and is known as syphilitic ozena. There is some pain, but it is not as severe as the lesion seems to warrant. The pain is increased upon deglutition.

SYPHILIS OF THE LARYNX

The primary, secondary, and tertiary manifestations of syphilis may appear in the larynx, though the primary lesion is extremely rare. Syphilis of the larynx is estimated as comprising from 1 to 15 per cent. of all cases of syphilis. Its occurrence in the pharynx is given as about 10 per cent., and in the nose as nearly 3 per cent. of all cases. About one-fifth of all the cases of syphilis appear, therefore, to affect some portion of the upper respiratory tract.

Syphilis of the larynx occurs most frequently between the twentieth and fiftieth years of life. In the congenital form it appears either in the first few months of life or at about the age of puberty. When it occurs soon after birth the lesions are usually secondary. If the second stage is completed in utero the disease may only become manifest in the third stage after the lapse of several (usually from two to fifteen) years.

Secondary erythema of the larynx usually occurs as an accompaniment of the same process in the pharynx, but whether hereditary or acquired it is in the tertiary stage that relief is usually sought. Males are more often affected than females.

Gross Pathology.—The lesion is usually bilateral and appears upon the true and false cords as a catarrhal inflammation with hyperemic spots and abraded epithelial areas. Condylomata may occur on the epiglottis or upon the laryngeal mucous membrane, and cause considerable stenosis.

Symptoms.—Though the ulceration takes place very rapidly, the pain is usually slight. The lesion first appears in the form of a clear-cut, deep ulcer. Induration is not always present, though there may be slight thickening at the edges of the ulcer. Edema is not a marked feature. At the bottom of the ulcer the cartilage may be necrosed and may be the seat of suppuration; that is, perichondritis and chondritis of the laryngeal cartilages may be present. The mucous membrane is hyperemic and darkly congested. The condition is improved by the administration of the iodides, though this may be temporary. Hemorrhages sometimes occur, and in rare instances endanger life.

The vocal changes are unilateral paralysis (though it may be bilateral), with a raucous hoarseness or aphonia. Cough is in some subjects an early symptom. Dysphagia may or may not be present. If the syphilitic lesion is located on the posterior aspect adjacent to the mouth of the esophagus of the larynx, dysphagia is usually a marked symptom.

Prognosis.—Syphilis of the larynx usually yields to treatment, though it may leave the vocal apparatus somewhat impaired as to its anatomical and physiological integrity. Life is not usually in any great danger, except in those cases in which the hemorrhage is unusually severe, or in which the stenosis causes suffocation. When on account of the suffocation it becomes necessary to perform tracheotomy, the patient should be warned that in all probability he will have to wear a tracheal tube the balance of his life.

Treatment.—The general treatment should be as for syphilis elsewhere in the body. Local treatment to relieve the cough or pain may become necessary. In case perichondritis and necrosis of the laryngeal cartilages is present, it is best to first administer the iodides in full doses, in order to diminish the acute pathological process, and then, if necessary, to remove the fragments of diseased cartilage. This may be done by direct laryngoscopy, or by laryngofissure (see Laryngoscopy and Laryngofissure); the former is preferable, for if the other method is adopted, it may become necessary to repeat the operation a number of times.

In cases of extreme stenosis, tracheotomy should be performed and a tracheal cannula introduced.

SYPHILIS OF THE EXTERNAL EAR

Primary chancre of the external ear is so rare that less than half a dozen cases have been reported in the literature.

The secondary manifestations may be papular, pustular, macular, ulcerous, or condylomatous. The entire auricle may be destroyed by extensive ulcerations, or it may be greatly deformed. The manifestations in the ear are usually secondary to a similar affection of the adjacent skin.

Condyloma of the meatus is rare; it occurs in the proportion of about 1 to every 240 cases of general syphilis (Deprès and Buck).

The course of condyloma in the external meatus is as follows:

(*a*) In the beginning there is a red efflorescence of the skin, other symptoms being absent.

(*b*) A little later, diffuse swelling of the walls of the meatus occurs.

(*c*) The skin begins to be slightly broken and secretion is thrown upon the surface.

(*d*) Finally, warty growths, of a grayish-red color, form in the cartilaginous portion of the auditory meatus, and, more rarely, in the osseous portion. They may be large enough to block the meatus.

(*e*) Pain usually develops with the appearance of the condyloma, especially if the skin is ulcerated. It is intensified by movements of the lower jaw, as the glenoid fossa is in very close relation to the antero-

inferior wall of the meatus. Deafness and tinnitus develop in proportion to the degree of the meatal obstruction. Fever is exceptional.

(*f*) Resolution may take place either with extensive destruction of the tissue or with little or no changes whatsoever. In some cases the ulceration continues for many months. Under general treatment resolution takes place quickly, and little or no scar tissue forms. Stricture of the meatus is rare.

Diagnosis.—The diagnosis should be based upon the history of specific disease elsewhere in the body, the characteristic glandular swelling, and the appearance of the local lesion.

Prognosis.—The prognosis of condyloma and the other secondary forms of syphilitic manifestation is favorable under the internal administration of mercury and iodides.

Gummatous formations of the external ear are usually simultaneous in their appearance with the same process in the middle ear. They may appear later as deep ulcers with elevated margins.

Treatment.—The local treatment of the primary chancre should consist in cleansing the parts with black wash and then applying the following ointment:

℞.—Unguent. hydrargyri,
Lanolin . . . . . āā ℥iv—M.
Sig.—To be applied with cotton pads held in place with a light bandage.

Mercury should also be given internally, or it may be rubbed into the skin in the form of blue ointment.

Condylomata and other secondary syphilitic manifestations should be treated by the internal administration of mercury and the local application of a powder composed of equal parts of calomel and the oxide of zinc, which should be applied once or twice daily.

To reduce the exuberant granulations, apply a strong solution of the nitrate of silver.

Gumma should be treated by the internal administration of mercury and the iodide of potash or iodonucleoid to the point of toleration.

LEPROSY

Synonyms.—Elephantiasis græcorum; leontiasis; satyriasis; French, la petæ; German, der Aussatz; Norwegian, spedalskhed.

Leprosy is a chronic infectious disease caused by the Bacillus lepræ. It is characterized by the presence of tuberculous nodules in the skin and mucous membranes (tuberculous leprosy), or by changes in the nerves (anesthetic leprosy). At first these forms may be separate, but ultimately they exist in combination. In the characteristic tuberculous form there are disturbances of sensation.

It is customary to divide leprosy into two general forms, the tuberculous and the anesthetic, lepra tuberosa or tuberculous leprosy, and lepra anesthetica seu nervosa. It is also sometimes subdivided into:

(*a*) Tuberculous nodular.
(*b*) Non-tuberculous.
(*c*) Mixed tuberculous.

Etiology.—Geography.—In Europe it is most common in Norway, the Swedish, Finnish, and Russian Coasts, the East sea; then in Asia, India, China, Africa, Egypt, Abyssinia, Morocco; and in America (California and Mexico). It is also found in Australia and the Sandwich Islands.

The Bacillus lepræ was discovered by Hansen, of Bergen, in 1871, and is universally recognized as the cause of the disease.

Modes of Infection.—There are three possible modes of infection, viz.:

(*a*) *Inoculation.*—It has not been proved that leprosy is contracted by accidental inoculation, though it is highly probable.

(*b*) *Heredity.*—For years it was thought to be transmitted, though it is probably not.

(*c*) *By Contagion.*—The disease is contagious. The bacilli are given off from the nasal secretions, open sores, and the excretions of the body. Osler says it is probable that the bacilli may enter the body in many ways through the mucous membranes and through the skin. Sticker believes that the initial lesion is the ulcer upon the cartilaginous part of the nasal septum. If this is true the disease assumes greater importance to the rhinologist and suggests the advisability of maintaining thorough cleanliness of the nose on the part of those associated with leprous patients.

Pathology.—The Bacillus lepræ has many points of resemblance to the tubercle bacillus, but can be readily differentiated from it. It is cultivated with extreme difficulty, and, in fact, there is some doubt as to whether it is capable of growth on artificial media (Osler). Lepra tuberosa, or tuberculous leprosy, attacks chiefly the integument and the mucous membrane of the nose, palate, roof of the mouth, larynx, and pharynx. On the skin the first changes show themselves in the form of infiltrations; the skin in one or more places over areas of several centimeters becomes elevated and assumes a brownish-red or dull red color. In the region of the infiltration the sensibility disappears, partly or completely, and on hairy parts the hair of the affected area falls out. On mucous membranes the lesions show themselves either as small patches or tubercles, or as round, flat infiltrations, which become ulcerated and heal with cicatricial contraction. The results are often conspicuous disturbances of the affected part, the disappearance of the cartilaginous nasal septum, the soft palate, and the epiglottis. Stenosis of the larynx is one of the most common occurrences. Characteristic tubercles also often develop on the conjunctiva bulbi, especially at the corneal borders. The disease has a remarkably regular and progressive course, inasmuch as new lesions are always appearing. The outbreaks arise with the initial eruptions. Under febrile action, the erythematous reddening of the affected parts develops, and is soon followed by the formation of tubercles and nodules. At the site of the older lesions, usually at the time of the fresh outbreaks, changes take place, and miliary abscesses or blebs develop, either of which may end in ulceration. It

is deserving of mention; that at the time of these fresh outbreaks the lepra bacillus may be demonstrated in the blood, in which, at other times, it is absent.

Lepra Anesthetica seu Nervosa.—Anesthetic leprosy is characterized by sensibility and trophic disturbances of the skin and muscles. The formation of new tissue, which produces the nodular growths of the tuberculous form, is small or entirely absent. The disease begins as a leprous polyneuritis. Anesthetic leprosy, in typical cases, has no resemblance to tuberculous leprosy. It usually begins with pains in the limbs, and areas of hyperesthesia, or of numbness. Bullæ may form very early, maculæ appear on the trunk and extremities, and, after existing for a variable length of time, disappear, leaving areas of anesthesia, though anesthesia may develop independently of the maculæ. Superficial nerve trunks may be large and nodular. The bullæ change to destructive ulcers. The fingers and toes are likely to contract and necrose. This type runs a very chronic course and may not be severe in its results (Osler).

Mixed tuberculated lepra is the least common form; it constitutes about one-sixth of all cases, about one-half of which are apparently hereditary, each parent often having had a different form. It begins with either a tuberculous or a non-tuberculous symptom; most frequently the latter are more prominent for a few months, fever and the usual phenomena of tuberculization then occurring. Destruction of the cartilage of the nose sometimes ensues; the soft palate also may be destroyed by ulcerations. The balance of the symptoms are a compound of the other varieties.

Prognosis.—The disease is very chronic, progressive, and probably incurable. The tuberculous form is destructive. The nervous form may not greatly impair the patient's usefulness, as in the case of the clergyman who continued his career for thirty years after contracting the disease.

There are no specific remedies for the disease. General tonics should be combined with local treatment to meet the indications, and this is all that can be done.

GLANDERS

Synonyms.—Equinia maliasmus; malleus; malleus humidus; farcy; morve; farcin; rotz.

Glanders is a contagious disease affecting horses and asses. It is communicable to man. It is caused by the bacillus mallei. When it affects the mucous membrane it is called glanders, and when it affects the skin and lymphatic glands it is called farcy.

Etiology.—Glanders originates in horses and asses, but is communicable to man, and from man to man. It is naturally more often found in men engaged in occupations which bring them in contact with beasts of burden. Though the bacillus may gain entrance through the follicles of the skin, it more often does so through an abraded or a wounded surface.

Cases are reported of surgeons being infected while operating upon patients who had the disease.

Pathology.—There are numerous closely associated nodules of low grade embryonal or granulation tissue, which readily break down and suppurate. The ulcers thus formed have undermined edges, which are the remnants of the wall of the preceding abscess. The process spreads by continuation, though later it may be carried to distant parts. It usually appears first in the skin, and then extends to the mucous membrane of the nose, though it may have its origin in the mucosa. Baumgarten says it is a disease which stands midway between abscess and tuberculosis.

The nasal lesions are usually in the form of numerous closely grouped granulation nodules in the submucous tissue. There is a profuse proliferation of leukocytes and connective tissue cells, with which are admixed numerous bacilli of glanders. The proliferation continues until the pressure diminishes the nutrition of the mass, especially at its centre, liquefaction necrosis then ensues and the nodules become abscesses. The outer wall soon breaks down and the contents are discharged into the nasal cavities. The abscesses are thus converted into open ulcers with undermined edges. Cross-sections of the masses before breaking down show them to be composed almost entirely of leukocytes, connective-tissue cells, and fibrous tissue. Many Bacilli mallei are embedded in the masses of proliferated cells. In the acute form there are numerous multinuclear leukocytes in the adjoining tissue. In the chronic form the bone and deeper structures may be destroyed. Gangrene of the softer tissues may occur.

Symptoms.—In the acute form the period of incubation is from three to four days. The acute symptoms often simulate rheumatism or typhoid fever in its initial stage. A little later the nodules appear either upon the skin or the nasal mucosa, according to the point of infection. They rapidly increase in size, as described under pathology, until (in nasal glanders) the purulent contents empty into the nose. The upper air passages are not often involved primarily in man. The progress of the disease is rapid, and usually leads to a fatal issue in a few days, or in two or three weeks.

The chronic form is fatal in about 50 per cent. of the cases after two months to two years. This type bears a close resemblance to syphilis and tuberculosis. The lymph glands of the neck are often much enlarged in the acute form. Chronic glanders often presents the symptoms of a persistent coryza. The diagnosis is difficult. It may be necessary to inoculate a male guinea-pig with the nasal secretions to determine the diagnosis. At the end of two days, in a positive case, the testicles of the pig are swollen and the skin of the scrotum reddened. The testicles continue to increase in size and finally suppurate. After two or three weeks death occurs, and the postmortem reveals nodules in the viscera. The use of "mallein" is highly recommended for diagnostic purposes. It is used in the same manner as the tuberculin test in tuberculosis. In all suspected cases remove a piece of the tissue and examine sections

with the microscope; make agar cultures and inject them into the peritoneal cavity of a guinea-pig, and watch the reactions. Also use injections of mallein, and watch the results. Above all, study the clinical phenomena, and from all the evidence obtainable arrive at a diagnosis.

Prognosis.—The prognosis in the acute form is grave, for nearly all cases die in a few days. In the chronic form the mortality is about 50 per cent., and death occurs in from two months to one or more years.

Treatment.—In acute cases there is little hope of recovery. If seen early the tissue around the point of original infection should be either extensively cauterized or removed *en masse*. The wound thus created should be frequently bathed in a solution of the chloride of zinc (one to eight). All animals and horses suspected of being infected should be killed and their bodies burned. In chronic cases, tonics and the iodide of potash should be given, though no specific remedies are known.

Glanders of the pharynx is usually an extension of the same process from the nose, though it may be primary in the pharynx. Nodules form here, as in the nose, and are attended by about the same general symptoms. The cervical and sublingual glands are early involved, break down, suppurate, and discharge externally.

The chronic form is not attended with the same distinct phenomena, and is often mistaken for granular pharyngitis. The nodules are mistaken for the lymphoid masses which occur in chronic follicular pharyngitis, though, if watched long enough, they will be seen to grow gradually larger and larger, until serious mechanical obstruction results. Such a process in the pharynx should arouse a suspicion of glanders, and the mallein test, or guinea-pig experiment as given under Symptoms should be made.

Glanders of the larynx is rare, and when present is associated with the same process higher up in the respiratory tract.

ACTINOMYCOSIS OF THE NOSE

Synonyms.—Lumpy jaw; holdfast, or wooden tongue.

Definition.—Actinomycosis is a parasitic, infectious, and incurable disease which was first observed in cattle and later in man. It is characterized by the manifestations of chronic inflammation, with or without suppuration. It often results in the formation of granulation tumors, especially about the jaw and neck.

Etiology.—The exciting cause is the ray fungus of actinomyces. The predisposing causes are an abraded mucous surface, or a diseased membrane. The infectious material may be carried by water or food, and by straws, chaff, grain, needles, etc. The fungus probably grows upon wheat and oats, hence farmers should be cautioned against chewing wheat and oat straws, as they seem to be a prolific source of infection. Shoemakers occasionally contract the disease from the habit of holding a needle or awl in the mouth. Kissing may be the means of transmission from one person to another. It occurs chiefly in young adults.

Pathology.—The actinomyces were formerly thought to be mould fungi, but Bostroem, in 1885, proved by cultivation that they are a variety of cladothrix, belonging to the schizomycetes. The diseased mass is made up of granulation tissue, which, except for the ray fungus, would be mistaken for round-cell sarcoma. Epithelioid elements and giant cells are sometimes present. In the granular mass, or in the pus, the fungus itself appears in the form of small, yellow, brown, or green masses, about the size of a pinhead, which, upon microscopic examination, are found to be composed of a central interwoven mass of threads, from which radiate club-shaped ended rays. In man the clubbed bodies are frequently absent (Senn). The histological lesions are alike in the actinomycotic nodule, and in the tuberculous follicle, only the germ body differs. Water, or a weak solution of sodium chloride, causes the rays to swell enormously and lose their shape; ether and chloroform have no action upon them. The gross pathological anatomy of the disease is everywhere associated with chronic indurations, with softening and liquefaction, and with resulting sinuses and cysts. The head, neck, and especially the jaw, and the cervical fascia are the sites of the disease. In the cervical fascia the disease gives the neck a brawny hardness. The lymphatic glands are not, as a rule, extensively involved. In the ox the tongue is often affected.

The lesion may be self-limited, as in tuberculosis, by cicatricial envelopment.

The kernel-like nodules are usually multiple. They may coalesce, and the resulting masses may "heal out." When bone tissue is affected, the destruction is central, while peripherally there is hyperplasia.

ACTINOMYCOSIS OF THE PHARYNX AND TONSILS

Symptoms.—The symptoms vary according to the part involved. The affection is chronic, but occasionally runs a rapid course. The granulation tissue is abundant and the mass resembles a tumor. Previous to suppuration it is quite firm, and if progressing rapidly it is surrounded by diffuse edema. Pain and tenderness are rarely present. When suppuration occurs the mass increases rapidly in size.

The frequency of occurrence in different parts of the body in 500 cases, as collected by Poucet and Berard, is as follows: Head and lungs, 55 per cent.; thorax and lungs, 20 per cent.; abdomen, 20 per cent.; other parts, 5 per cent. In France the face and neck were affected in 85 per cent. of the 66 cases reported.

The symptoms may be grouped in two classes: (*a*) Those referable to local tumefaction and purulent discharge, and (*b*) those referable to the general intoxication of the system by the suppurative products, or their metastatic spread, and which do not differ from those of chronic suppuration. The local symptoms are of slow development, and are largely those of gradual mechanical interference of the pharyngeal function. At the site, or sites, of inoculation a small rounded and reddish elevation

appears, and is accompanied by the usual subjective annoyances of an attending pharyngitis. The adjacent tissues become swollen and tumefied, and the evidences of an acute inflammation soon change to the more permanent engorgement and solidity of a chronic condition. The swelling is irregular, but well outlined, firm to probe palpation, and not oversensitive, but slowly increases in size. Suppuration and the formation of angry-looking sinuses follow, from which issue a purulent discharge, in which are the small yellowish pellets, or masses, composed largely of the typical ray fungus. The discharge is persistent, and the sinuses extend deeply and produce extensive destruction of tissue. The spread of the process does not, as a rule, occur, and it shows a tendency, if it occurs elsewhere, to do so as an isolated swelling rather than as a connected overgrowth from the original pharyngeal focus. Pain is a variable quantity, and depends largely upon the seat and extent of the peculiar swelling. Usually there is a more or less continuous, heavy ache which is felt locally, and this may, at times, be eased or intensified into acute distress. Fetor of the breath and gastric disturbances from the purulent discharge are often present. The appearance of the disease elsewhere by metastasis is to be expected, especially in the lungs or the alimentary tract, though no portion of the body is free from possible invasion. The systemic symptoms may be severe or slight, according to the degree of involvement and the exit of the suppurative products, and do not differ in their character from those usually observed in any other suppurative condition. Death occurs from slow exhaustion or through some intercurrent affection or complication (Kyle).

Diagnosis.—Actinomycosis should be differentiated from:

(*a*) Sarcoma.
(*b*) Tuberculous infection.
(*c*) Carcinoma (of the tongue).
(*d*) Syphilis.
(*e*) Epulis (in jaw).
(*f*) Lupus.

It is, perhaps, impossible to make a positive clinical diagnosis of actinomycosis. A microscopic examination showing the ray fungus, or inoculation of a guinea-pig, may be necessary to establish it. The presence of the yellowish particles in the purulent discharge is quite characteristic, though not conclusive. Actinomycosis is probably not as rare as is generally supposed, as it is sometimes mistakenly diagnosticated as sarcoma, carcinoma, osteomyelitis, syphilis, etc.

(*a*) *Sarcoma* is histologically quite similar to actinomycosis. A careful microscopic examination will, however, in actinomycosis show the presence of the ray fungus and some giant cells. Sarcoma does not break down and suppurate so early. Both occur quite frequently in the young.

(*b*) *Tuberculous disease* is attended by an enlargement of the regional lymphatics. In actinomycosis the regional glands are not enlarged. An examination of the sputum or the inoculation of a guinea-pig will show the tubercle bacilli if present.

(*c*) *Carcinoma* of the tongue is usually found near the base, whereas actinomycosis affects the tip. Then, too, in carcinoma there are lancinating pains, ulceration, and cachexia.

(*d*) *Syphilis*, in the gummatous stage, is more amenable to treatment by means of the iodides. The general history of the case is also an aid in the differential diagnosis. Acute progressive actinomycosis may very strikingly resemble acute phlegmonous inflammation and osteomyelitis.

Treatment.—The iodides are efficacious in recent cases. In old cases in which there is a mixed infection they are less efficient. The remedy should be given until marked iodism results. The injection of a 5 per cent. solution of the permanganate of potash into the cysts, when present, has produced good results. Cauterization of the skin and soft parts with the solid stick of silver nitrate is a valuable aid in those cases in which there is a fistula and suppuration. Gautier reports excellent results from the injection of a 10 per cent. solution of the iodide of potash into the mass. Electric needles may be inserted in the tumor, and 50 milliamperes of current passed through it. Every minute a few drops of the iodide of potash solution should be injected until a total of 20 minims is used. The electric current decomposes the iodide solution into nascent iodine and potash. The chemicals thus liberated in the actinomycotic tissue act as a deterrent to the further progress of the disease. A general anesthetic should be administered for this treatment. It should be repeated in eight days.

The surgical treatment of actinomycosis varies from simple incision to the complete removal of the entire mass. The disease is best suited to surgical treatment before the stage of suppuration and extension to the regional glands. When it has progressed thus far it is no longer simple actinomycosis, as it is complicated by a mixed or streptococcal and staphylococcal infection. A simple incision is sometimes effectual, as is, indeed, spontaneous rupture. Should excision be resorted to, it should be complete, and followed by the thermocautery, to prevent the spread of infection to the exposed lymph spaces. After suppuration is established, treat as for tuberculosis, *i. e.*, curette and pack with iododorm gauze.

The disease seems to be self-limited by the formation of a capsule of connective tissue and by spontaneous rupture.

Iodide of potash or iodonucleoid are probably the most reliable internal remedies.

ACTINOMYCOSIS OF THE MIDDLE EAR

Actinomycosis of the middle ear is very rare, and the only literature on the subject is the clinical report of a case by Zaufal, of Prague, and a more extended report of the same case, with the postmortem findings, by J. C. Beck, of Chicago, and a second case of Mojocchi, of Italy. The clinical aspect of Beck's case was as follows: Carl J. was fifty-four years old, a farmer, always healthy, with a negative history of aural, nasal, and pharyngeal disease, until six months previous to the examination. At

that time there was a swelling back of the left ear and left side of the neck. The swelling, at first hard, soon softened, and was never painful. Later a third swelling appeared on the left side of the neck, which opened and discharged pus through a fistula. At this time the hearing became defective. The functional tests of hearing showed a negative Rinné, and Weber lateralizing to the left side, thus showing middle-ear disease. There was no secretion from the external auditory meatus, but the post-superior wall, at the fundus, sagged as in mastoiditis. A swelling the size of the palm of the hand was situated over the mastoid and the region posterior and inferior to it. It did not fluctuate. A smaller swelling, anterior to this, had a fistulous opening in the region of the tip of the mastoid process. Compression expelled a greenish pus, containing small granules. The subsequent microscopic findings showed the ray fungus of actinomycosis in abundance. A radical mastoid operation was performed, but the healing process was unsatisfactory. Five weeks later the patient died from an intracranial hemorrhage, due to the ulceration of a large bloodvessel in the region of the actinomycotic process. The postmortem was held by Chiari, who found the muscles of the neck on the left side and the upper cervical vertebra infiltrated with pus containing yellowish particles. There was no suppurative process in the cavum tympani. A fistulous tract was traced with a fine probe from the cavum tympani toward the exposed incisure mastoidea. The left sigmoid sinus was filled with a substance of a light yellow color, and was adherent. The cervical glands on the left side were enlarged, and cross-sections showed whitish discolorations. Sections of the tonsils and the contents of the lacunæ were negative as to actinomycosis. The ulcerated artery causing the fatal hemorrhage was examined microscopically by Beck, who found the ray fungi in its walls. This is the first reported case in which the ray fungus has been found in the wall of a bloodvessel.

The only other case of actinomycosis of the middle ear on record is reported by Majocchi, of Italy. In his case the primary infection was in the lung, and the infection of the ear probably occurred during a fit of coughing.

PART II

THE PHARYNX AND FAUCES

CHAPTER XVII

DISEASES OF THE EPIPHARYNX AND BASE OF THE TONGUE

ACUTE LACUNAR INFLAMMATION OF THE PHARYNGEAL TONSIL

According to Felix Peltesohn, the pharyngeal tonsil consists of six fairly symmetrical folds separated by deep furrows running in a sagittal direction, which may be separated from each other like the leaves of a book. Posteriorly and sometimes anteriorly there is a curved fold connecting all of them. In the middle there is a deep fissure—the recessus medium—to which, in some instances, a blind canal leads, and which was formerly erroneously described as an independent structure, the bursa pharyngea, which, when infected, is known as Thornwaldt's disease.

Bickel, in defining a tonsil, says it is characterized (*a*) by its well-defined shape, (*b*) by a diffused infiltration of lymph cells and follicles, and (*c*) by crypts or lacunæ, that is, mucus pockets lined with epithelium, around which the lymphatic tissue is arranged.

If we take his definition literally only the pharyngeal and faucial tonsils are real tonsils, as the lymphoid tissue in the other parts of the so-called "tonsillar ring" does not have crypts or lacunæ. The faucial tonsil also has a distinct fibrous investing capsule.

Symptoms.—Angina lacunaris of the pharyngeal tonsil, like that of the faucial tonsils, is an infectious disease. It is rarely recognized as such by physicians on account of its hidden location back of the postnares and the soft palate. The condition may be seen, however, with a postnasal mirror. The crypts or lacunæ are filled with a yellowish-white exudate, composed of epithelium, inflammatory exudate, and pus cocci. An inexperienced physician might easily arrive at the erroneous conclusion that the spots were "ulcers;" indeed, the same error has often been made concerning the faucial tonsils. During the acute stage the pharyngeal tonsils are red and swollen.

That the disease is infectious is shown by the clinical data—namely, the initial chill, the rise of temperature, the prostration, swelling of

the spleen and cervical glands, the prolonged convalescence, and the presence of a great variety of infectious germs.

The secretion is often so fluid as to ooze from the crypts and coalesce with that from an adjoining crypt.

Acute lacunar inflammation of the pharyngeal tonsil does not occur as often as acute lacunar inflammation of the faucial tonsils. This is probably due, in part, to the filtrating function of the vibrissæ and moist mucous membrane of the nose.

It occurs most often during the first twenty years of life, because the lymphoid adenoid tissue is more developed and more sensitive during this period of life. It has a strong tendency to recur. The nose becomes obstructed and there is pain upon swallowing, but it is not definitely located as when the faucial tonsils are diseased. The voice becomes nasal, or void of resonance, as in hypertrophy of adenoids. The glands at the angle of the jaw and in the deep cervical region are swollen and painful upon pressure.

The fever is cyclical, being less severe of mornings and greater at night. It continues for several days and leaves the patient quite exhausted. The pharyngeal tonsils continue swollen for some time, often permanently after the fever subsides, and cause more or less nasal obstruction.

To one not accustomed to examining the epipharynx the following suggestion by Peltesohn is of great value in making a diagnosis: If the tongue is drawn far enough forward with a tongue depressor to see behind the palatine arch, the salpingopharyngeal fold, the so-called "lateral column," may be found to be deeply reddened and studded with yellow follicles. This condition is characteristic of angina lacunaris of the pharyngeal tonsil. As the space between the soft palate and the posterior pharyngeal wall is still quite wide in young people, the postrhinoscopic examination may be easily made.

Patients frequently complain of a feeling of fulness and pressure in the ears, but do not often have active inflammation of the middle ear. The nasal secretions are acrid, and often cause nasolabial excoriations.

Diagnosis.—(*a*) Initial infective fever, chill, and cyclical fever.

(*b*) Obstructed nasal passages and non-resonant voice.

(*c*) Most important of all, the red and swollen follicles of the "lateral column" (follicles just back of the posterior faucial pillar), from which a yellowish secretion is exuding.

These signs, together with the postrhinoscopic examination, will lead to a correct diagnosis.

Treatment.—Experience teaches us that during the course of the acute or febrile stage local applications irritate and should not be attempted; even gargles should not be used. The patient should be kept in bed until the fever abates, or a few days longer, as the prostration is severe. He may be given pieces of ice to hold in the mouth, as this seems to afford some relief. Only a light diet should be allowed.

After complete recovery the adenoids, whether large or small, should be thoroughly removed, as otherwise recurrence may take place. In

adults the recurrences are characterized by the formation of crusts in the epipharynx. The crusts, therefore, indicate the need of an adenoid operation.

ADENOIDS

Synonyms.—Adenoid vegetations; pharyngeal adenoids; pharyngeal tonsils; epipharyngeal tonsils.

Definition.—Adenoids are hypertrophied lymph glands which normally exist in the epipharyngeal space. They are chiefly located on the superior and posterior walls of the epipharynx, though they may extend into the fossæ of Rosenmüller and to the mouth of the Eustachian tubes (tuba auditiva Eustachii).

The edges of the walls of the recessus medius sometimes become agglutinated during acute inflammatory processes, and thus convert the groove into a sinus, which becomes infected and continually discharges its secretions into the pharynx (Thornwaldt's disease).

Etiology.—The chief cause of adenoids is the irritation and inflammation which occur in the epipharynx during attacks of one of the exanthematous fevers. It is a well-known pathological law that the lymphatic structures of children become enlarged or hypertrophied in response to bacterial stimulation, whereas the same stimulation in adults does not cause lymphoid hypertrophy to a corresponding degree.

As the exanthematous fevers occur chiefly in early childhood while the special susceptibility exists, it is but natural to find adenoids most frequently during this period of life.

According to the statistics on this subject by McBride and Turner, adenoids are most frequently found *between the sixth and the fifteenth years of life,* though they may occur at any period. In children who were otherwise normal it has been variously estimated that they were present in from 1 to 9 per cent. of all cases examined. In deaf mutes they are present in from 50 to 73 per cent. of all cases examined.

While it cannot be said that adenoids are hereditary, they are, nevertheless, in many instances a *family characteristic,* perhaps on account of a similar environment and similar anatomical conformations predisposing to infection of the epipharyngeal tissues.

Climate probably plays but a small part in the causation of adenoids, though it should be said that a cold, damp, changeable climate subjects the mucosa as well as the general system to repeated shocks, which lower the vitality and render the lymphoid tissue an easy prey to infection.

Pathology.—The distribution of adenoid tissue in the epipharynx is chiefly on the upper and posterior walls, though it may extend to the fossæ of Rosenmüller and to the orifices of the Eustachian tubes. Adenoids are composed of lymphoid tissue enmeshed in a definite though comparatively delicate reticulum of fibrous connective tissue. The essential pathology of adenoids consists in the hypertrophy of the lymphoid tissue of the epipharynx which is normally present there.

According to McBride and Turner, the pharyngeal tonsil is a peripherally placed lymphatic gland, from which efferent ducts pass to the nearest

glands in the cervical chain. Like similar glands elsewhere, the pharyngeal adenoid tissue consists of a fibrous connective-tissue framework, supporting masses of lymphoid cells, but owing to its peripheral position it differs from the more deeply placed lymphatic glands in having an epithelial covering upon its free surface. The supporting framework consists of fibrous septa passing through the substance of the gland, from which a very delicate connective-tissue network ramifies in all directions toward the surface. It carries in it the bloodvessels and the lymphatics, while here and there, lying in clusters in the septa, may be seen many mucous glands whose ducts open on the surface. In the meshes of the delicate network lie masses of leukocytes or lymphoid cells, constituting the lymphoid tissue which forms the main bulk of this tonsil. Groups of these cells are specially differentiated in the form of more or less rounded or oval-shaped areas, having centres of a pale appearance, while their margins are more darkly colored. These areas are the follicles or germ centres of Goodsir. Covering the free surface of the tonsil, and dipping down into its recesses and crypts, is a layer of ciliated epithelium, continuous with that lining the respiratory part of the interior of the nose and the adjacent mucous membrane of the epipharynx. The epithelium consists of more than one layer of cells, the superficial ciliated cells being columnar in type, while the deeper cells forming two or three layers are smaller, and rest upon a well-defined basement membrane.

The Epithelium.—The normal epithelial covering undergoes a certain amount of variation, as might be expected when a growth of this kind, itself subject to variations in size, fills to a varying extent a cavity like the epipharynx, more or less completely surrounded by firmly resisting bony walls, and whose size is intermittently changing through the movements of the soft palate which constitutes its floor. The epithelium is not always of uniform thickness. While preserving its ciliated columnar type its thickness varies in parts, so that the lining of some of the crypts presents an irregular outline. In a certain number of the preparations examined, however, there is a marked change in the character of the epithelium, becoming of the stratified squamous variety and of a very considerable thickness. This change and thickness are not general, but are confined to certain areas on the surface of the hypertrophy. It is not normal to this part of the upper respiratory tract, because the whole of the mucous membrane of the pharynx as low as the level of the lower border of the soft palate is covered with ciliated epithelium, and it is from within the area so covered that the epithelium thus altered and thickened shows that these changes occur among the youngest of the patients examined. With two exceptions at the age of twelve, all were under ten years of age, and in two cases where the thickening was most marked the patients were only four years old. On the other hand, in the sections of the growths removed from patients of fifteen years and upward, with one exception, no thickening of the epithelium was observed, so that we are naturally led to the conclusion that this change in the epithelium is not one necessarily

dependent upon the prolonged existence of the hypertrophy. Occurring, as the examination shows that it does, in the younger patients, it is more reasonable to conclude that it is due to pressure of the growth upon the walls in the smaller epipharynx of the young child. Its presence on the surface and in patches only and less frequently in the crypts are further points in favor of such a view being held. Unfortunately, we are unable to say whether, in those cases in which the epithelium has changed to the pavement type, the adenoid masses were large and more or less completely filled the epipharynx. Such a change in the type of the epithelium as noted here has been observed before, as the result of pressure, and is a point of some histological interest. The pressure to which these growths is subject is intermittent, and is caused chiefly by the elevation of the soft palate in the act of deglutition, pressing the soft, pliant mass upward against the walls of the space, and releasing it again when the act is completed.

The Fibrous and Lymphoid Tissues.—A considerable variation was found to exist in the relative proportion of lymphoid and fibrous tissue in the growths examined; and McBride and Turner endeavored, by a comparison of the appearances observed in patients of different ages, to seek some explanation of the gradual disappearance or shrinking which takes place in the hypertrophied adenoid tissue in course of time. An overgrowth of fibrous tissue around the bloodvessels forms by a process of perivascular sclerosis; at any rate, it is in the neighborhood of these vessels that the fibrous thickening is most evident. If an area be examined in which this change is taking place, some of the bloodvessels present a normal appearance, others again show distinct thickening of their walls in concentric rings, with diminution in the size of the lumen. One specimen showed, in a remarkable manner, many of the bloodvessels completely obliterated, partly owing to the great thickening of the walls and partly to the contraction of the fibrous tissue outside. Round the vessels there is fibrous tissue formation, varying both in amount and in density, according to the stage of development that has been reached; in this way the lymphoid tissue becomes gradually invaded and areas of cell are isolated by the process. There can be no doubt that it is by fibrous-tissue formation that the gradual shrinking of the adenoid mass occurs. In order to ascertain what relation such a process might bear to the age of the patient, a comparative study of the various growths was made with this end in view.

From such an analysis it would appear that a development of fibrous tissue takes place in the substance of the adenoid hypertrophy, commencing around the bloodvessels invading the lymphoid tissue, and replacing it. This process, however, is independent of the age of the patient, and is not one that necessarily commences at or after puberty, but may occur at all ages, and be even more marked in the very young child than in the adult. The observations of McBride and Turner coincides with that of M. Brindel. The practical deduction drawn from these facts is, that we cannot say in any given case that a growth may be satisfactorily left to

disappear *per se*. It may or it may not do so at some early period, but because a patient is approaching puberty or adult life it does not follow that the adenoid hypertrophy will in a short time cease to exist. As we have already stated, such growths do, in certain cases, disappear at puberty, but it is quite possible that here a purely physical, as opposed to a purely histological, explanation may be called to our aid. Obviously, in the small epipharynx of the child the growth may entirely fill the space, while, as adult life is approached, a free space will be left between the adenoid hypertrophy and the palate. In the former case, each respiration will exercise suction upon the mass, while in the latter this physical effect will be much diminished, if not entirely absent.

The foregoing findings should be given wide circulation among the medical profession, as physicians too often advise their patients "to wait for puberty," as the adenoids will "shrink" at that time. "Waiting" for adenoids to "shrink" is always a foolish and dangerous thing. While waiting, the attending inflammation is ever progressing, and may and actually does in 66 per cent. of all cases, invade the Eustachian tubes and middle ear. Furthermore, it is shown that the atrophy does not occur after puberty any more than at a younger age; indeed, the atrophy is independent of the age in the patient. Why wait, therefore, for a process of shrinking which has no definite period of occurrence.

Symptoms.—The symptoms of adenoids may be divided into:

(*a*) Objective.
(*b*) Subjective.
(*c*) Collateral.

Objective Symptoms.—The objective symptoms are those which are appreciated through the special senses of the attending surgeon.

By inspection the physician notes the open mouth, thick, short upper lip (Plate IX), the comparatively expressionless countenance, and with the laryngeal mirror he finds the epipharynx more or less filled with the adenoid masses.

By *the sense of touch* he distinguishes a gelatinous, worm-like mass in the epipharynx. The finger should be anointed with vaseline before it is introduced into the epipharynx, so as to reduce its frictional qualities to the minimum. Even then great care should be exercised lest the delicate mucous membrane of the epipharynx be injured. In spite of these precautions the finger is often streaked with blood upon its removal. I find the digital examination of more value in a majority of the cases than the one with the mirror. It need occupy but a few moments for its performance.

The examining surgeon should stand in front of and to the right of the patient, encircling the head with his left hand and arm to steady it, while the index finger of his right hand is introduced into the epipharynx. McBride and Turner have suggested that if the thumb of the examiner is just outside the patient's right cheek, he can prevent biting by pressing the thumb against the cheek wall. The soft tissues being thus forced between the patient's teeth, he will not bite the examiner's finger.

PLATE IX

An Adenoid Face.

Faulty development of the chest walls is also characteristic of mouth breathing in children.

The sense of smell should also be utilized in the examination for adenoids, as a fetid breath is sometimes present.

The auditory sense should also be utilized in the diagnosis, as the patient's voice is often characteristic. The articulation is muffled and the resonance of the voice is diminished.

The Subjective Symptoms.—Restlessness during the night is a prominent symptom; the patient often throws the covers off during the unconscious rolling and tossing which is so characteristic of mouth breathers. Night terrors are also frequently complained of, especially if the child is troubled with enuresis. I have often noted that night terrors or horrible dreams immediately precede nocturnal urination.

Night terrors are not present in all cases, perhaps not in more than one-third of them; they are in all probability due to reflex causes and to an excess of the half-way products of metabolism. These dreams are often of the most terrible nature, and are often indelibly impressed upon the memory.

Daytime restlessness is also a characteristic sign of adenoids. The child is fretful and peevish, or is inclined to turn from one amusement to another, or from an imposed duty to play.

The mental faculties are often much impaired in adenoid subjects. Aprosexia, or difficult attention, first described by Guye, of Amsterdam, is very often present. The child is listless and has difficulty in applying himself continuously to his play, studies, or other tasks, of which he soon tires. He has fits of abstraction. I once knew of a boy in school who was afflicted with ideal abstraction, though he had a fairly good mind. In those cases, however, in which there is little obstruction, the mental faculties are but little affected.

Taste and smell are sometimes impaired, which is not strange, in view of the fact that the sense of smell and of taste are so intimately associated, and the epipharynx is blocked with adenoids, thus compelling the child to breathe through its mouth.

The breath is often fetid, from the decomposition of the retained secretions and from the disordered stomach which is so often complained of.

Bilious attacks sometimes complicate the case.

An elevation temperature is not an uncommon symptom, as the adenoid growth is frequently the seat of a lacunar or catarrhal inflammation.

Epipharyngeal catarrh is an almost constant accompaniment of adenoids. Indeed, it is doubtful if adenoids of any considerable size are present without a concomitant chronic epipharyngitis, or what is commonly spoken of as a pharyngeal catarrh. This symptom or complication is one of the strongest arguments in favor of the removal of adenoids, as the catarrhal inflammation has a tendency to extend by continuity of tissue into the Eustachian tube and middle ear. In case of an acute infectious exacerbation the middle ear and even the mastoid cells are likely to become involved.

Collateral Symptoms.—*Defective speech* is a symptom of considerable

diagnostic and economic significance. The voice is muffled and articulation is imperfect. The resonance, or timbre, of the voice is greatly impaired.

Aural complications are present in a majority of cases. According to McBride and Turner, who analyzed 307 cases, 255 had involvement of the ear. Of the 255 cases, 144 were suppurative and 111 were more or less deaf with non-suppurative ear disease. They say: "We have more than once noticed in children (affected with adenoids) suffering from non-suppurating otitis media that in those in whom the membrana tympani had assumed an appearance which can but be likened to that of ground glass, especially when there was a permanent pinkish tinge, the prognosis as to improvement by subsequent treatment was not good, sometimes positively bad."

It appears, therefore, that the aural complications, whether of the suppurative or non-suppurative type, may be serious.

Diagnosis.—The diagnosis in most cases is so obvious that it scarcely warrants special mention. There are exceptional cases, however, in which an error may be made. It may be stated as an almost universal rule that *when the tonsils are hypertrophied adenoids are also present.* Conversely, it cannot be said that when adenoids are present the tonsils are also hypertrophied, as statistics show that only 30 per cent. of the cases with adenoids had apparent enlargement of the tonsils. It appears that the adenoids most easily undergo enlargement, the tonsils next, and the lingual less than either of the other lymphatic structures composing Waldeyer's ring.

The *fringe of the adenoids* on the posterior wall of the pharynx, just below the line of the soft palate, is quite characteristic. When these nodules are present in a child, I am quite certain of the diagnosis, even without further examination, though I do not recommend that the examination should stop here.

The epipharyngeal mirror should be used, when possible, to enable the surgeon to see the adenoids and their distribution. In many cases this method of examination cannot be adopted on account of the reflex closure of the palatal muscles against the posterior pharyngeal wall.

When the mirror cannot be used the index finger of the right hand should be introduced through the mouth into the epipharynx for the purpose of detecting the gelatinous worm-like mass of adenoid tissue.

It is not sufficient to merely determine the presence of a large adenoid cushion in the vault, or on the superior posterior wall of the epipharynx, but the examiner should determine whether the fossæ of Rosenmüller or the tubal orifices are covered by the growths. Adenoids are not removed merely because they are enlarged, but because of the epipharyngitis which almost always attends them and on account of their presence in the fossæ of Rosenmüller and the Eustachian orifices, even though they are small.

Fibrous tumors of the epipharynx are sharply defined and are dense in texture, whereas adenoids are not sharply defined and are soft in

texture, hence there need be no difficulty in making a differential diagnosis.

Malignant tumors of the epipharynx can scarcely be mistaken for adenoids if an ordinarily careful examination is made. The hemorrhage, cachexia, and other symptoms readily distinguish the cancerous growths.

Tuberculous and *syphilitic* granulomata rarely simulate adenoid growths. Carel has reported two cases of tertiary syphilis, and Lermoyez a case of tuberculosis of the epipharynx, which closely resembled, in general symptomatology, adenoid growths.

Prognosis.—The prognosis from the standpoint of the mentality of the patient varies from slight retardation to an almost complete arrest of mental development. The improvement in the mental growth after operation is often marvellous, provided the operation is performed during the natural period for such development, *e. g.*, during infancy and childhood. If the removal of the growth is delayed until the individual has practically attained full growth, the mind will rarely develop as it would had they been removed at an earlier period.

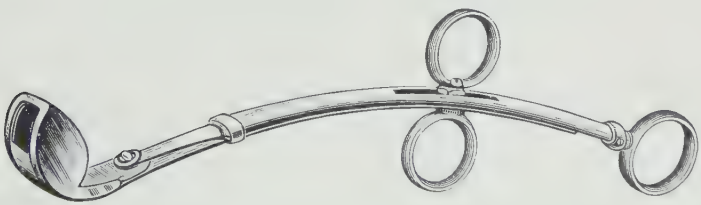
The general health rarely improves during infancy and childhood so long as adenoids remain. If, however, they are removed, the blood becomes red from free oxygenation and all the vital energies are quickened and increased.

The "facial or adenoid expression" improves somewhat with advancing years, though it often remains as a permanent disfigurement through life. If the adenoids are removed sufficiently early in life the "adenoid expression" often disappears, or its further development is prevented.

The early removal of adenoids often prevents serious aural complications, improves the general health, and beautifies the face.

Treatment.—There is but one treatment worthy of the name, and that is the surgical removal of the growth. Astringent applications have been and are still advocated by some writers, but in my opinion their use is but a means to postpone the day when their removal must take place. I can conceive how a congestion and inflammation of the lymphoid masses

FIG. 235

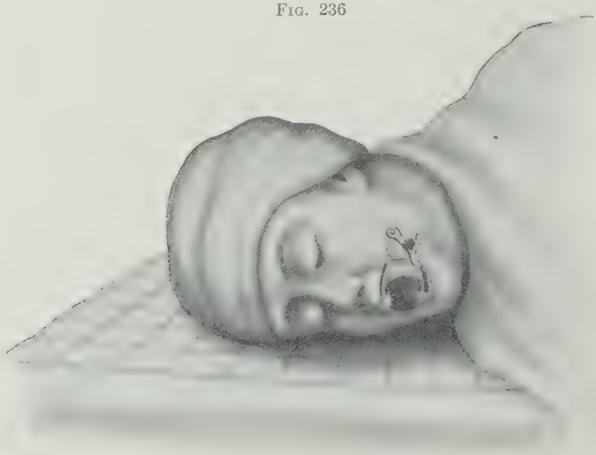


La Force adenotome

might be relieved and greatly improved by the local use of alkaline and astringent washes, but when true hypertrophy has occurred the curette or forceps offer the best means of treatment.

Adenoids may be removed with the Meyer ring curette through the nose, though this is an almost obsolete method. A more rational and effective method is with a Boeckmann curette or some modification of it. During the last few years I have depended more and more upon an instrument of the La Force pattern (Fig. 235).

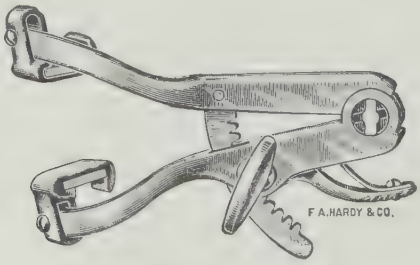
Fig. 236



Lateral position of the patient under general anesthesia for the removal of adenoids and tonsils.

Technique.—The following technique may be employed for simple adenectomy, though in combined adenectomy and tonsillectomy anesthesia by ether is preferable (Figs. 236 and 237).

Fig. 237



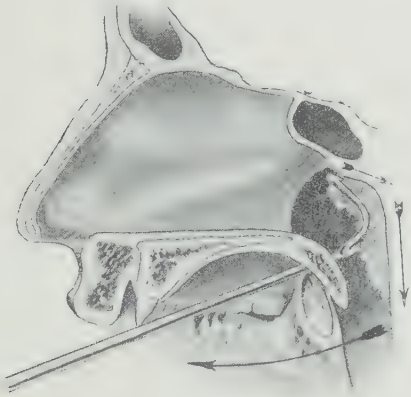
Furguson-Pynchon mouth gag.

(*a*) Nitrous oxide anesthesia.

(*b*) The removal of the adenoids with the La Force adenotome is performed as follows: The blade of the instrument is withdrawn, leaving the fenestra open. The instrument is then introduced into the fauces, the tip turned laterally, engaging behind the patient's right posterior pillar. It is then turned upward into the epipharynx. The adenoid is engaged by pushing the instrument upward and backward. The blade is then pushed home, cutting the adenoid from its

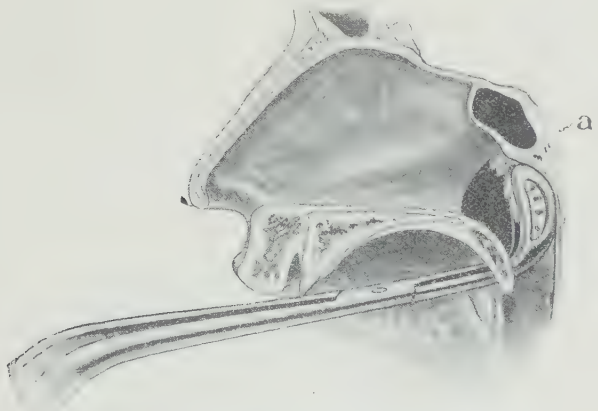
attachment. The instrument is then removed, opened and the adenoids removed from it.

Fig. 238



Removal of adenoids with the Boeckmann-Stubbs curette. The arrows indicate the three movements necessary for the complete operation in a normal epipharynx.

Fig. 239



Removal of adenoids with the Brandegee forceps. The remnant (*a*) left in the anterior portion of the vault just posterior to the septum should be removed with the Stubbs modification of the Boeckmann curette.

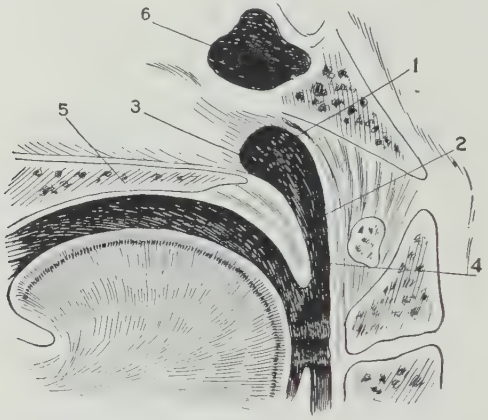
(*c*) Introduce the curette (Fig. 238) in the same manner and engage the mass at the anterior portion of the vault just behind the posterior end of the septum, as the adenotome often fails to remove the adenoid tissue in this position (Fig. 239, *a*).

(*d*) Introduce the right index finger into the epipharynx and rub away any shreds and remnants of adenoid tissue which may remain. Also

explore Rosenmüller's fossæ with the finger tip and remove the fibrous adhesive bands should any be present.

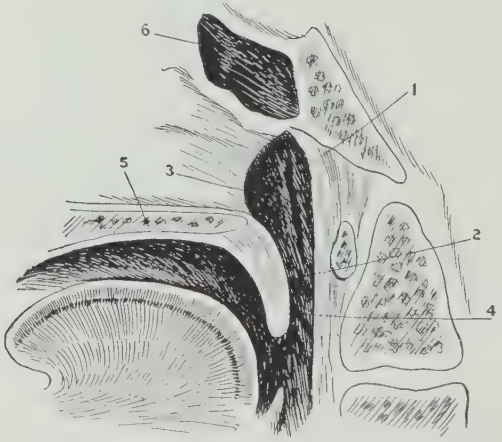
(*e*) The patient's head should then be held over the fountain cuspidor until bleeding stops or consciousness is restored.

FIG. 240



1, normal vault of the epipharynx from which adenoids may be removed with the Boeckmann curette; 2, posterior wall of the pharynx; 3, posterior end of vomer in its normal relation to the hard palate; 4, uvula; 5, hard palate; 6, sphenoid sinus.

FIG. 241



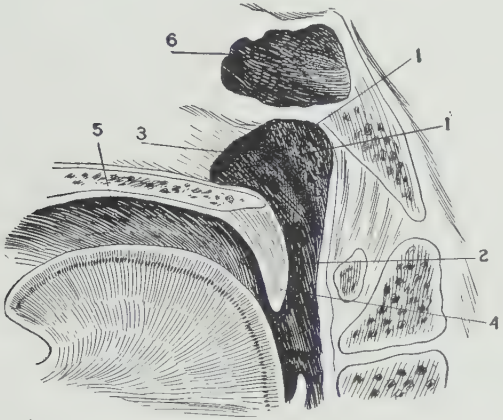
An epipharynx with an angular superior pouch from which adenoids could be removed with the Boeckmann curette, except possibly the upper angle of the pouch. This region might necessitate the use of a special curette. 1, 2, 3, 4, 5, and 6 refer to anatomical points (Fig. 240).

During the operation the patient may be in the sitting posture, preferably in the lap of an assistant. He should be wrapped tightly with a sheet in order to prevent his arms getting in the way during operation.

I sometimes operate without a general anesthetic if the patient is

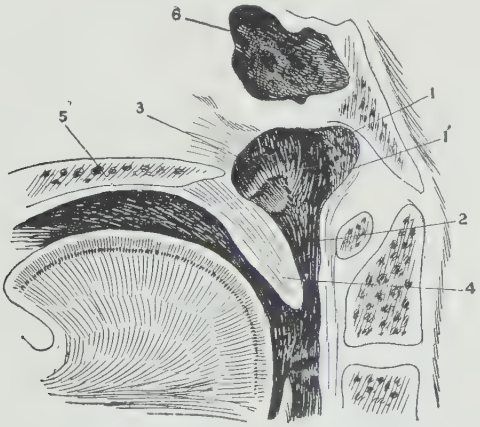
old enough to submit without resistance. The pain is not great and the danger from an anesthetic is obviated. It should be said, however, that the danger from nitrous oxide gas is practically nil, whereas the records show that several cases have died under chloroform.

FIG. 242



An epipharynx with a shallow posterior pouch from which the adenoids could be removed with the Boeckmann curette, except in the posterior portion of the pouch: 1, a slight recess in the posterior wall of the vault of the epipharynx in which adenoids are inaccessible to the Boeckmann curette; 2, 3, 4, 5, and 6 refer to anatomical points. (After Moure).

FIG. 243



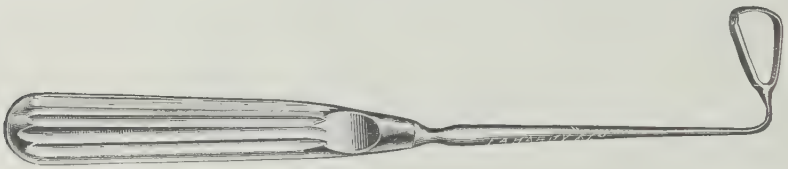
An epipharynx with a deep pouch in the posterior wall, from which adenoids could not be removed with the Boeckmann curette. Such cases should be operated on through the nose with Wilhelm Meyer's ring curette (Fig. 245), or with a special curved curette (Fig. 246).

Stubbs' Method.—According to Stubbs, the blade of the curette should be drawn forward against the septum, lifted upward against the vault, and then pushed directly backward until the posterior wall is reached. The blade of the curette should then be drawn downward over the

posterior wall and quickly brought forward into the cavity of the mouth (Fig. 238). If the curette is as wide as the epipharynx, one introduction of the instrument usually removes the entire growth. Stubbs has modified the Boeckmann curette to adapt it to this technique.

According to Moure, the epipharyngeal space varies greatly in shape, a fact which largely determines the completeness with which adenoids may be removed with the usual form of curette and forceps. If the epipharyngeal space is normal in shape (Fig. 240), the curette and forceps will completely remove the adenoids. If there is a recess in the vault (Fig. 241), these instruments will fail to remove all the tissue. If there is a recess in the posterior wall of the epipharynx (Figs. 242 and 243), the forceps and curette of the usual type will fail to remove all the tissue. These facts may account for the non-success of many adenoid

Fig. 244



Special curette for reaching the recesses in the vault of the pharynx.

Fig. 245

Meyer's ring curette.

Fig. 246

Pynchon's modification of Golding Bird's curette.

operations. If there is a recess in the upper wall of the epipharynx, a specially designed curette (Fig. 244) should be used to complete the operation. If there is a recess in the posterior wall of the epipharynx, the Meyer ring curette (Fig. 245) introduced through the nose, or Pynchon's modification of Golding-Bird's curette shown in Fig. 246, or Quilan's forceps, should be used to complete the operation.

George L. Richards advises the removal of adenoids under general anesthesia with the Shutz adenotome. He believes that by this method a more complete removal is attained. The adenotome is inserted into the epipharynx and pressure is exerted upward and backward while the blade is being closed. This method has the advantage of preserving the specimen intact for inspection. H. Gradle's adenotome is also a good instrument, and is preferred by some operators. The

objection to all such instruments is that they do not adapt themselves to the peculiar conformation of the epipharynx shown in Figs. 240 to 243. They also fail to remove the portion of the growth located in the lateral portions of the pharynx. If, however, the adenotome is followed by the use of a suitable curette, as Stubbs' modification of Boeckmann's model, the result is good.

Whatever method of removal is used, the ultimate aim should be the complete removal of the adenoids, as otherwise they will probably recur.

Sequelæ.—The Face.—The development of the face is often materially modified by the presence of adenoids. The open mouth, the absence of the nasolabial folds, the short upper lip, the protruding and twisted central incisors of the upper jaw, the broad, flat, upper half of the nose, and the narrow, slit-like nasal openings, all conspire to form the so-called "adenoid face." The general expression is one of stupidity. The degree of facial disturbance varies greatly in different cases, usually in proportion to the degree of the nasal respiration, rather than the actual size of the adenoid growths. According to J. E. Schadle, the average capacity of the epipharynx is about 17 c.c., and its lateral is longer than its anteroposterior diameter. If the capacity of the epipharyngeal space is diminished, or its anteroposterior diameter is contracted, a small adenoid mass may produce a greater nasal obstruction than a larger growth in a more roomy epipharynx. The facial expression is more modified in the former than in the latter instance. It should not be deduced from the foregoing statements that the indications for treatment are in proportion to the degree of nasal obstruction *per se*, as there are several other conditions resulting from small as well as large adenoids that necessitate their removal.

Interior of the Nose.—The interior of the nose is also modified in its development. J. S. Thompson called attention to this fact in an article wherein he states that the loss of the physiological stimulation incident to nasal respiration results in underdevelopment of the turbinals, and that deviated septa are common. Such individuals are subject to intranasal disease, for obvious reasons.

Hard Palate.—Adenoid subjects usually have a palate which is "gothic" or arched, especially in its anterior portion. The arch is apparently higher than normal, though, as Newkirk has shown by numerous casts, the increased height is apparent rather than real. The illusion arises from the fact that the lateral diameter of the upper jaw contracts while the height of the arch remains the same; this produces a marked disproportion between its width and height.

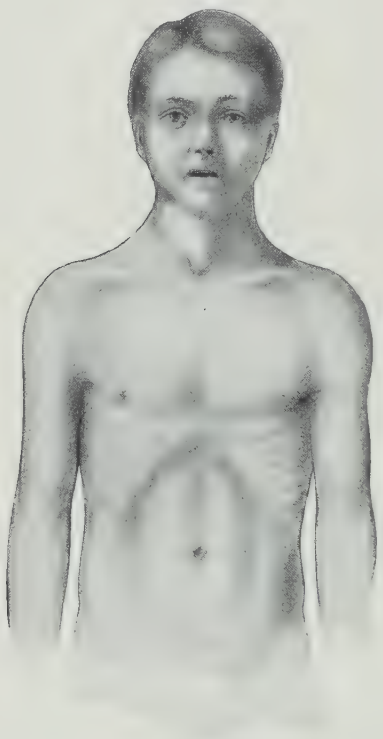
The Teeth.—The contraction of the lateral diameter of the arch sometimes causes the central incisors to protrude and to be twisted upon their axes so as to cause their posterior surfaces to face each other. The teeth are often irregular, and the services of a dentist are required to regulate them.

Epipharyngeal Inflammation.—When adenoids are present the epipharyngeal mucous membrane is almost always the seat of local inflamma-

tions of both the acute and the chronic type. The low resistance of the adenoid tissue, the rarefied or abraded cylindrical epithelium, the retention of the secretions, and the insufficient ventilation of the epipharyngeal space all promote inflammatory processes. The inflammation may be lacunar, either acute or chronic, or it may be a diffused catarrhal inflammation which affects the mucosa covering the adenoids and the adjacent structures.

The Auditory Apparatus.—Adenoids are a prolific source of inflammation in the Eustachian tube, middle ear, and mastoid process. It is a common clinical experience that children with adenoids who complain of recurrent attacks of earache are relieved by tympanic inflation. The Eustachian tubes are closed by catarrhal swelling, or "plugged" with thick, tenacious mucus, and the air in the tympanic cavity becomes absorbed and rarefied.

FIG. 247



Deformity of the chest due to adenoids.

The drumhead is retracted and the mucous membrane which lines the tympanic cavity is hyperemic. Catarrh of the tubes and middle ears is thus established.

Suppurative otitis media is also caused by adenoids. The infective material from the epipharynx enters the tubes and middle ears during the acts of coughing, sneezing, or other violent movements of the pharyngeal and palatine muscles. Then, too, the ciliated columnar epithelium of the tubes may become atrophic or broken down by the pressure of the opposed walls from the catarrhal swelling. The absence of the ciliæ permits easy ingress of the infected secretions into the middle ear, and infection thus becomes established in the tympanic cavity.

Having gained a foothold in the tympanic cavity, it is but another step for the infection to invade the mastoid cells. The inflammation of the middle ear and mastoid process is usually proportionate to the virulency of the microörganisms which cause it. The labyrinth may also become involved in the infective inflammations of the middle ear, though such an occurrence is rare. Deafness, in some degree, is always present in the foregoing aural complications of adenoids.

The Respiratory System.—The anterior nasal openings are narrow and slit-like, while the turbinated bodies are underdeveloped. The conditions

are favorable for the development of catarrhal inflammation of the mucosa of the nose. The lateral walls of the chest are contracted (Fig. 247), thus throwing the ensiform cartilage into prominence. This characteristic deformity is known as "pigeon chest." The lungs are also undersized and respiration is shallow. The transfusion of gases through the walls of the air vesicles is impaired. Too little oxygen passes into the blood, and too little carbon dioxide is thrown off. The patient is both anemic and nervous, and is often irritable to a marked degree.

The Bones.—Frederick Coolidge called attention to the apparent relationship existing between adenoids and the various forms of club foot. I have often verified the saying that "if you will show me a bow-legged man I will show you one who had adenoids in infancy." Adenoids affect the nutrition, partly through anemia and partly through an excess of carbon dioxide in the blood. These two conditions cause faulty metabolism and nutrition. The bones are deficient in lime salts, hence are soft and bend easily under the weight of the body.

The Blood.—Adenoid patients are usually anemic. The red blood corpuscles are deficient in number and in hemoglobin. Carbon dioxide is present in excess. The nutrient elements are diminished in quantity and quality.

Fig. 248

Pharyngeal scissors.

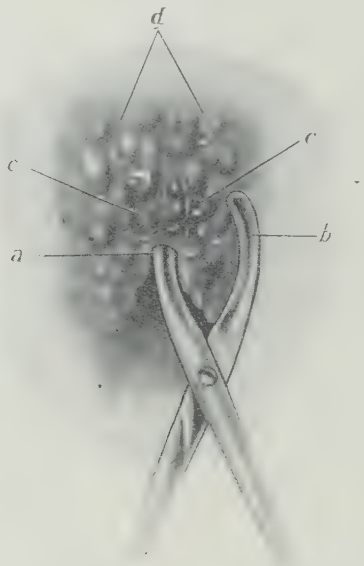
Thornwaldt's Disease.—This condition is characterized by a suppurating canal in the recessus medius or groove between the lateral halves of the adenoids. It is due to the inflammatory adhesion of the median borders of the adenoid masses. That is, the recessus medius, a groove between the lateral halves of the adenoids, becomes converted into a canal. The lining membrane of the canal becomes infected and discharges a purulent secretion. The symptoms are those of chronic pharyngitis attended with a cough.

The canal may be seen by the use of a throat mirror, and a curved probe may be passed upward into it.

The author's method of treating it is to introduce one blade of the curved pharyngeal scissors (Fig. 248) into the canal and then to cut off one lateral half of the adenoid mass (Fig. 249). This is a better way than to attempt to remove the adenoids in the usual manner, as the fibrous canal is so dense that it can be cut with difficulty. The posterior

and remaining portion of the canal wall should be thoroughly curetted to remove the pyogenic membrane.

FIG. 249



Operative treatment of Thornwaldt's disease: *a*, the left blade of the pharyngeal scissors introduced into the suppurating sinus between the lateral halves of the adenoids; *b*, the right blade of the scissors at the border of the adenoid tissue. When the blades are closed the lateral half of the adenoids upon this side is severed. The scissors are then transferred to the other lateral half of the adenoid tissue and closed. This completely severs the lower portion of the adenoid tissue, and obliterates the suppurating sinus. The remaining upper portion of the adenoids, *c*, *c*, *d*, is then removed with the scissors or with a curette.

THE LINGUAL TONSIL

The lingual tonsil is situated on the base of the tongue between the faucial tonsils, and extends anteroposteriorly from the circumvallate papillæ to the epiglottis. It is divided in the median line by the median glosso-epiglottic ligament. The tonsil consists of numerous rounded or circular crater-like elevations, which are composed of lymphoid tissue, which at their circumference are surrounded by connective tissue. In the centre of each crater the mouth of the duct of a mucous gland opens. The crater or crypt is lined by stratified pavement epithelium.

The lingual tonsil usually reaches its greatest development in young children, and, like the other tonsillar structures, may begin to atrophy at the age of puberty, though in adults these structures are often undiminished in size. In the adult the number of the masses is generally greatly reduced, though they may be greatly hypertrophied.

Here, as in the other portions of the tonsillar ring surrounding the oropharynx, leukocytes are thrown out in great abundance.

Acute Catarrhal Lingual Tonsillitis.—Acute catarrhal inflammation of the lingual tonsil is characterized by a moderate rise of temperature, painful deglutition, and a burning, pricking sensation in the throat. There may be some tenderness on pressure in the region of the great cornu of the hyoid bone. Upon inspection the pharynx and the pillars of the fauces may be slightly reddened, while the faucial tonsils may appear normal. The laryngeal mirror shows the masses on the lingual tonsil to be greatly reddened and swollen (Lennox Browne).

Treatment.—The treatment consists in brushing the inflamed masses with a 20 to 50 per cent. solution of the nitrate of silver.

Acute Lacunar Lingual Tonsillitis.—The symptoms of acute catarrhal inflammation are present, and in addition the craters or crypts are lined with a whitish exudate, epithelial debris, and micro-organisms quite similar to the accumulations found in acute faucial lacunar tonsillitis.

Treatment.—The treatment consists of the local application of a 20 to 50 per cent. solution of the nitrate of silver.

Acute Phlegmonous Lingual Tonsillitis.—This process is usually characterized by a purulent accumulation beneath the lymph nodules at the base of the tongue, and is usually limited to one side. The temperature is elevated and the pain upon deglutition is severe. The patient complains of soreness and great tenderness upon pressure in the region of the great cornu of the hyoid bone upon the affected side. Inspection with the throat mirror shows great swelling and redness at the base of the tongue upon the affected side. Palpation with the finger may or may not elicit fluctuation.

Phlegmonous inflammation here, as in the faucial tonsil, may undergo resolution without the formation of an abscess.

Treatment.—Treatment consists of incisions into the swollen tissue.

Hypertrophy of the Lingual Tonsil.—Hypertrophy of the lingual tonsil is rare in children. It usually occurs between the twentieth and the fortieth years of life. It is more common in females than in males. It is probably caused by repeated or continued infection of the lymph structures of the pharynx, fauces, and epipharyngeal tonsils.

Symptoms.—The symptoms are sometimes absent, though the sensation of a foreign body in the throat is usually complained of. There is a pricking sensation, as though a splinter had lodged in the fauces, or the patient complains of the sensation of a lump, a hair, or other foreign body in the throat. Paresthesia of the pharynx presents the same symptoms (Ball), and hence neurosis of the pharynx must be differentiated from this condition. So also must foreign bodies. According to Lennox Browne, troublesome fits of coughing are often present.

During meals the symptoms disappear. Pain is rarely complained of, but the disagreeable sensation already referred to is present. The use of the voice increases the symptoms, and often gives rise to the pricking sensation and the cough.

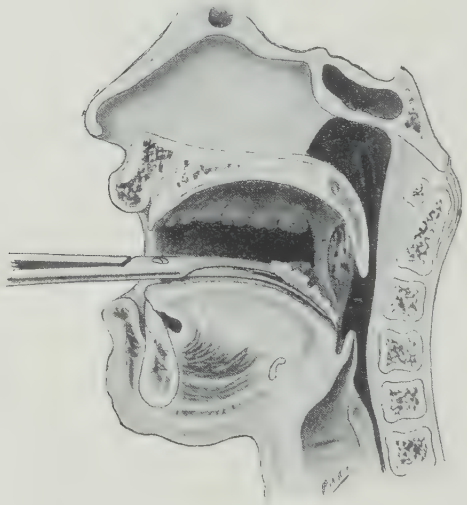
Upon examination with the throat mirror a few enlarged masses are seen upon the base of the tongue. The involvement is usually on both

sides, but may be limited to one. The masses may be so large as to push the epiglottis backward or even to overhang it.

According to Ball, Seifert emphasizes the value of the use of the probe and of cocaine in the diagnosis between paresthesia of the pharynx and hypertrophy of the lingual tonsil. With a probe the surgeon is enabled to locate the sensitive areas giving rise to the symptoms, and the application of cocaine causes these areas upon probing to give forth no symptoms.

Treatment.—The treatment is essentialy surgical. Local applications of glycerin iodine, gr. xx to xxx to the ounce, afford relief by reducing the swelling and sensitiveness. Linear cauterization of the masses is an effective treatment, though the removal of the masses with stout curved scissors has proved to be the best treatment in my experience (Fig. 250).

Fig. 250



Removal of the lingual tonsil with heavy scissors.

Lingual Varix; Varicose Veins.—Lennox Browne, in his treatise on the *Throat and Nose*, says that varix occurs in 10.6 per cent. of the cases at the Central London Throat, Nose, and Ear Hospital. As early as 1863, G. Lewin, of Berlin, reported that the symptoms of pharyngitis varicosa were sensations of scraping, burning, and dryness of the pharynx. Since then many writers have reported similar cases, so that its existence as a rather common form of disease is well established. I have seen cases in my own practice which presented the clinical picture described by Browne and others. It occurs more frequently in males, according to Browne (69 per cent.), though Swain and Seiss found it more frequently in females, while Seifert found it equally prevalent among both. Excessive and improper use of the voice is an exciting cause. It is rare in childhood and most common between the twenty-fifth and forty-

fifth years. Infectious inflammations of the pharynx and faucial tonsil and infection of the lymphoid tissue of the lingual tonsil probably are the chief etiological factors. On account of the greater resistance to these influences possessed by the lingual tonsil, hypertrophy in this region does not occur as early in life as it does in the faucial and pharyngeal tonsils. Hence, chronic infectious processes are often necessary to establish the hypertrophy of the lingual tonsil and varix of the veins. Browne believes that a constitutional or acquired debility of the vasomotor system is the chief cause. Some cases are reported as occurring at the period of the menopause. Constipation and an obstructed portal circulation are etiological factors of some importance.

Pathology.—I am indebted to Escat for the information that, according to Verneuil, "superficial varices only make their appearance when the deep varices have acquired a certain development." Escat also says: "Many kinds of neuralgia, otherwise inexplicable, are today attributed to circulatory troubles in the satellite veins of the nerves, and to a consecutive neuritis." Quenu has thus explained certain neuralgias: "The trunk of the lingual nerve, the evident seat of a glossodynia, is in effect, according to Foucher, accompanied by a satellite vein, and even by two according to Zuckerkandl." This anatomical fact is held by Escat to support his hypothesis, and that of Piotrawski, that all neuroses in this situation may be attributed to varices, superficial and deep.

Symptoms.—As lingual varix is usually associated with hypertrophy of the lingual tonsil, the symptoms are about the same. Upon inspection, tortuous veins, bluish in color, are seen at the base of the tongue partially hidden by the hypertrophied tonsil.

Treatment.—The treatment consists in the application of the galvanocautery to the enlarged veins, and the removal of the hypertrophied lymphoid masses with the cautery point or with scissors. I have frequently resorted to these methods of treatment with satisfactory results. The after-treatment consists in gently massaging the wounds with a cotton-wound applicator dipped in a mixture of equal parts of glycerin. tr. ferri chloridi, and tr. iodini, at intervals of twenty-four hours. This prevents exuberant granulations, and promotes healing with a smooth wound and a minimum of cicatricial contraction.

CHAPTER XVIII

INFLAMMATORY DISEASES OF THE MESOPHARYNX AND FAUCES

SIMPLE ACUTE CATARRHAL PHARYNGITIS

This form of acute pharyngitis is usually accompanied by acute rhinitis, or "cold," though the pharynx may be chiefly affected.

Etiology and Pathology.—The etiology and pathology is the same as that of acute rhinitis. Digestive disorders are an important factor in causing the disease.

Symptoms.—The onset is characterized by malaise and a slight rise in temperature, as in acute rhinitis. The borders of the soft palate and uvula are slightly red, while the adjacent mucous membrane is normal in appearance. As the disease progresses the uvula becomes slightly edematous and the secretions are increased; it may become markedly edematous and painful, though this is not common. The tonsils are not usually involved, though they may be in severe cases. Pain is usually present, especially upon swallowing, and stiffness and aching of the muscles of the neck are complained of. Dysphagia or painful swallowing is a constant symptom.

Diagnosis.—The erythema of secondary syphilis may be confounded with this disease. The differential points are: (*a*) The darker or dusky color (in syphilis) of the mucous membrane; (*b*) the more marked involvement of the tonsils and soft palate, the diminished secretion; (*c*) the line of demarcation between the inflamed area and the hard palate; (*d*) the dusky symmetrical patches on the anterior pillars; (*e*) the opalescent appearance of the mucous membrane of the tonsils and the persistence of the disease, as contrasted with the evanescence of acute catarrhal pharyngitis.

Treatment.—As the acute affection is somewhat dependent upon the presence of chronic rhinitis and sinuitis, these conditions should receive appropriate attention. The methods of treatment given for acute rhinitis are also of value, as the morbid process is almost identical.

The anatomical peculiarities and the associated digestive disorders, however, render special modes of treatment necessary.

Local treatment should vary according to the stage of the inflammation. Broadly speaking, astringents should be used in the first and third stages and sedatives in the second stage (Parker). They may be applied as gargles, sprays, paints, or lozenges. Gargles are suited to inflammations of the soft palate, uvula, and anterior pillars of the fauces. Sprays and paints are especially good methods of making local applications. Preliminary to all local treatment the alimentary tract should be evacuated.

From 5 to 10 grains of calomel, and six hours afterward a tablespoonful of castor oil, should be given. The following morning a tablespoonful of Epsom salt should be given to flush the bowels (Stucky). After this, the patient's condition is favorable for a speedy recovery under simple local treatment.

The following mixture is recommended by Parker:

℞.—Borax		gr. xxiv
Glycerin		♏ xxiv
Tincture of myrrh		♏ xxiv
Aquæ des.	q. s. ad	℥j

Sig.—Use every hour as a gargle.

If preferred, a gargle composed of 6 grains of alum, 15 grains of chlorate of potassium, to the ounce of water, may be used.

The patient may be supplied with lozenges containing krameria or catechu, with instructions to dissolve one of them in his mouth every three hours. A cold compress should be worn across the front of the neck.

After twelve hours, red gum lozenges, which are very sedative, may be substituted for those containing krameria and catechu. A simple gargle containing 15 grains of the chlorate of potash to the ounce of water may also be used every three hours.

The inhalation of steam charged from a croup kettle with the compound tincture of benzoin, one tablespoonful to the pint of boiling water, should be used if the throat is painful.

Pastils containing 3 grains of bismuth and $\frac{1}{4}$ grain of the acetate of morphine may also be dissolved in the mouth every three hours to relieve a painful throat.

Should edema of the uvula occur, it should be scarified or amputated.

CHRONIC PHARYNGITIS; GRANULAR PHARYNGITIS; LACUNAR PHARYNGITIS, OR CLERGYMAN'S SORE THROAT

This disease may or may not be characterized by severe subjective symptoms, such as irritability and dryness of the throat.

Etiology.—The chief factors in the etiology of this disease are gouty and rheumatic diatheses, smoking, improper breathing (public speakers and singers), and the presence of morbid processes in the nose, accessory sinuses, and the epipharynx. Gouty or rheumatic patients complain of throat symptoms, whereas if they are free from gout and rheumatism they often make no such complaint. These conditions probably not only aggravate the pharyngitis, but to a certain extent influence its occurrence. Excessive smoking also aggravates and produces the inflammation. Clergymen, singers, auctioneers, and hucksters, who breathe through their mouths and abuse the vocal apparatus, are frequently affected by chronic pharyngitis. Chronic rhinitis, and especially sinuitis, affecting the posterior ethmoidal and sphenoidal cells is very frequently the chief cause of the disease. The changed respiratory functions of the

nose in these diseases subject the pharynx and the lower respiratory tract in general to irritation. Of even greater importance is the discharge of heavy mucous or mucopurulent secretions from the nose and accessory sinuses into the pharynx. The secretions are charged with pathogenic bacteria, and undergo decomposition, whereby certain irritating chemical products are liberated, and as the secretions flow over the pharynx the pathogenic bacteria attack the weakened mucous membrane and excite inflammatory reactions. The chemical irritation also adds to the reaction.

I wish, therefore, to emphasize the importance of making a careful examination of the nose and accessory sinuses in all cases of chronic pharyngitis.

Pathology.—The changes in the mucous membrane consist at first of an increased hyperemia and local leukocytosis, and later of the deposit of the least differentiated cells or connective-tissue cells. That is, hyperplasia of the mucous membrane occurs. The lymph tissues around the tubular glands of the pharynx are enlarged and are raised above the surface of the mucous membrane. Occasionally the tubular glands in the centre of the lymphoid masses are filled with a whitish exudate or cheesy material.

Symptoms.—Subjective symptoms are not always present, especially if the patient is not gouty or rheumatic, or if he does not misuse his voice. In gouty and rheumatic patients who smoke to excess or breathe improperly the subjective symptoms are usually present.

Subjective Symptoms.—In aggravated cases the voice becomes hoarse after moderate use, especially in public speakers, though the cords are neither red nor inflamed. According to Lennox Browne, the hoarseness is due to a spasm of the muscles of the pharynx and irritation of the superior laryngeal nerve.

Smokers complain of a dryness or of the sense of a foreign body in the throat. They have a constant desire to hawk and expectorate.

Cough may be present, though it is often absent. When present it is irritable and hacking in character.

The secretions in the early stage of the disease are excessive, thick, and tenacious. At a later stage the glandular functions become impaired and the throat dry and glazed.

The digestive tract is disordered, the breath foul, and constipation is generally present.

Objective Symptoms.—Upon examination of the pharynx the mucous membrane appears redder than normal, at least in certain areas. In other areas it is pale and fibrous in appearance, especially in old chronic cases. Enlarged bloodvessels often extend across the posterior pharyngeal wall. The secretion is often thick, heavy, and mucopurulent, though in the later stages it may be scanty and only forms a film over the surface. In these cases the patient complains of dryness of the throat. The uvula is often relaxed and elongated (Fig. 252), and should be amputated.

The lymph follicles of the posterior wall and of the lateral walls behind the posterior pillars of the fauces are enlarged. This condition is often

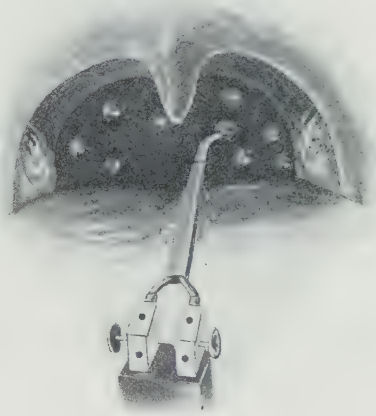
referred to as pharyngitis hyperplastica lateralis, a needless subdivision of chronic pharyngitis. The follicles are sparsely distributed on the posterior wall of the pharynx, but are closely grouped along the lateral walls. They appear as yellowish-red, raised areas on the posterior wall and as nodular, elongated masses behind the posterior faucial pillars.

Prognosis.—In the early congestive stage, simple astringent and demulcent local remedies combined with the regular use of a mild aperient mineral water will effect a cure. In the more advanced cases in which hyperplasia of the mucous membrane has occurred, and in which the lymph follicles are hypertrophied, improvement will only follow the destruction of the tubular glands around which the lymph masses are located.

Treatment.—In mild cases and during the early stage of the disease, or before marked hyperplastic and hypertrophic changes have taken place, the remedies given under acute catarrhal pharyngitis may be used with some success.

Aperient salines should be given daily for a long period to eliminate the gouty and rheumatic toxic material and to free the stomach and intestines of putrefactive substances.

Fig. 251



Showing the cautery point applied to pharyngeal follicular glands in the treatment of follicular pharyngitis. From four to five follicles may be thus treated at a sitting under cocaine anesthesia.

In well-advanced cases the lymphatic nodules, whether discrete or massed, as they may be on the lateral walls behind the posterior pillars of the fauces (pharyngitis hyperplastica lateralis), should be punctured with a cherry-red cautery electrode (Fig. 251). The mucous membrane should be brushed once or twice with a 10 per cent. solution of cocaine, and from four to five hyperplastic follicles burned out with the electrode. A spray of Seiler's solution, to soothe the burned areas, should then be used. At the end of the fifth or sixth day, four or five more follicles may be treated in a similar manner, and so on until they are all destroyed. This course of treatment is often very beneficial, though it may fail if the gouty or rheumatic diathesis is not also corrected. The uvula should be amputated if it is elongated.

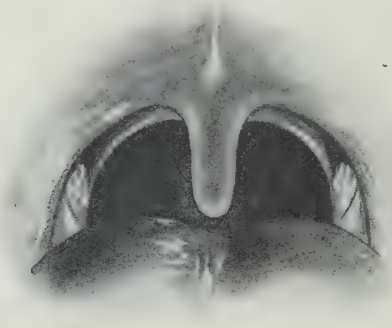
EDEMA OF THE UVULA

Acute inflammation of the faucial structures, especially of the peritonsillar tissue, is frequently attended by edema of the uvula (Fig. 252).

The methods of treatment generally recommended are scarification or

multiple punctures, which allow the excess of serum to escape. A more rational procedure would be to promote a freer flow of the blood through the tissues, and thus remove the obstruction to the blood current through the veins. The application of the rays of light and heat from a 500 candle-power electric lamp to the neck at the angle of the lower

Fig. 252



Edema of the uvula.

jaw acts admirably in this way. The lamp should be suspended at a distance of eighteen inches from the patient and slowly passed back and forth over the neck for from fifteen to thirty minutes, three times daily. The patient's neck should then be sponged with ice-water in order to prolong the hyperemia.

Astringent lozenges containing krameria and alum will be found efficacious in giving comfort to the patient.

ELONGATED UVULA

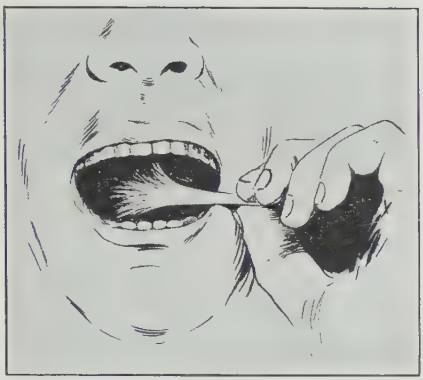
Elongation of the uvula is not a disease *per se*, but is a symptom of a chronic pharyngitis, especially epipharyngitis. The relaxed pendulous condition of the uvula is due to the irritation resulting from the epipharyngeal discharge and to the changed nutrition attending the epipharyngeal infection and inflammation. The uvula may be slender and pendulous, or it may be enlarged (hypertrophied) and pendulous. An elongated and elastic uvula is sometimes observed as an idiopathic condition, as shown in the author's case (Figs. 253 and 254).

Symptoms.—In robust subjects it causes but slight symptoms or none at all. In sensitive patients it often causes a reflex cough when it touches the epiglottis or the base of the tongue. The cough may be spasmodic, and is usually dry. Nausea and vomiting, especially early in the morning, are sometimes complained of. Patients have applied to me for relief from the persistent hacking cough, fearing that they had tuberculosis. An examination of the lungs failed to reveal disease in that region, whereas an examination of the throat showed the presence of a long pendulous

uvula. The amputation of the lower relaxed portion of the uvula immediately stopped all symptoms.

Treatment.—In simple cases, astringent remedies, such as lozenges containing krameria, afford relief. The uvula may also be painted with astringent solutions of alum, tannic acid, or with adrenalin. In the

FIG. 253

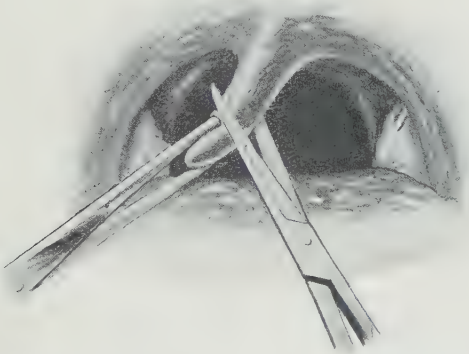


Author's case of elastic uvula. Note the spiral arrangement of the mucous membrane of the uvula when the muscle of the uvula is contracted. (See Fig. 254.)

FIG. 254

Author's case of elastic uvula, evincing no tendency to elongation when at rest. (See Fig. 253.)

FIG. 255



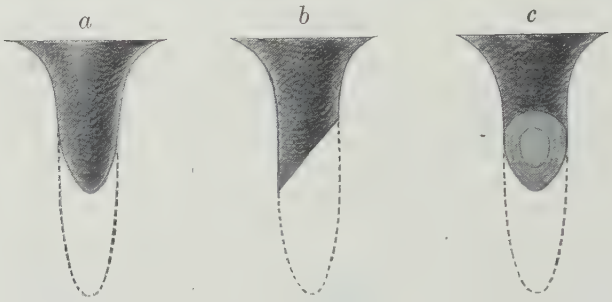
The amputation of the elongated tip of the uvula just below the lower extremity of the muscle. The scissors are so applied that the posterior surface of the uvula will be the wounded surface. This prevents irritation in swallowing food and in breathing through the mouth.

more severe cases amputation is indicated. In all cases the epipharynx and the mesopharynx (oropharynx) should be examined and the diseased conditions treated.

Surgical Treatment.—(*a*) The uvula should be painted with a 10 per cent. solution of cocaine.

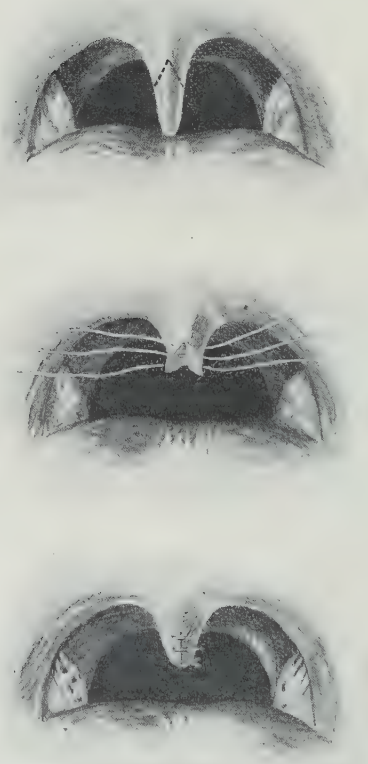
(*b*) The tip of the uvula is then seized with forceps and drawn directly forward.

Fig. 256



Three views of the amputated uvula: *a*, anterior view; *b*, lateral view; *c*, posterior view.

Fig. 257



Casselberry's operation for elongated uvula.

(*c*) While in this position it should be operated upon with heavy blunt scissors, as shown in Fig. 255.

By cutting the uvula from in front while drawn anteriorly, the bevelled cut surface of the stump faces posteriorly. This is a point of practical importance, as in swallowing solid food the raw surface is not irritated by it (Fig. 256).

Casselberry's Operation.—Dr. Wm. E. Casselberry recommends the following technique in the amputation of the uvula:

(*a*) Secure anesthesia by painting the uvula with a 10 per cent. solution of cocaine.

(*b*) Seize the tip of the uvula with forceps and draw it directly forward.

(*c*) While in this position an upward and medianward cut is made with scissors to the central axis of the uvula. A similar cut is made on the opposite side, thus removing a wedge-shaped piece of the uvula, as shown in Fig. 257.

(*d*) The anterior and posterior cut edges of the wound are then secured with two or three black silk sutures, black thread being used, because it

is easier to see at the time of its removal. Yankauer's needles may be used with advantage.

(*e*) The sutures should be removed at the end of three days.

The advantages claimed for this method of operating are that the cut surfaces are sealed and not likely to be irritated by the ingested food, nor infected by ingested and inhaled pathogenic bacteria.

Hemorrhage has been reported after uvulotomy. This may be avoided by limiting the amputation to the portion of the uvula below its muscular fibers; that is, only the thin relaxed portion should be removed, as the bloodvessels of the uvula do not extend beyond the muscular fibers.

RETROPHARYNGEAL ABSCESS

Abscess of the posterior wall of the pharynx may be acute or chronic. It may be situated in the mesopharynx, the hypopharynx, or the epipharynx.

Etiology.—There is an infection beneath the mucous membrane. The morbid germs gain entrance through the lymph vessels, the atrium of invasion being in one of the neighboring tissues which is diseased. Tonsillitis, a postoperative tonsillar wound, a tuberculous tonsil, tuberculous cervical glands, caries of the vertebra, and syphilis of the throat may be the immediate predisposing causes. The author observed one case which followed the complete excision of the tonsil in an adult. Most of the chronic cases occur in tuberculous and strumous children. Postpharyngeal abscess is often associated with tuberculous glands of the neck. The glandular involvement is probably secondary to the pharyngeal abscess, or both may be secondary to a tuberculous affection of some other structure.

Symptoms.—The patient complains of painful deglutition, and, if the swelling is in the hypopharynx, of dyspnea, which may threaten life or even cause death. Cough is constantly present. The voice is much the same as in quinsy. In acute cases the temperature may be elevated from 1° to 2°, whereas in chronic ones it is little altered.

Diagnosis.—The abscess should be differentiated from aneurysm, malformation of the vertebræ, and inflammatory swelling of the mucous membrane.

Aneurysm of an artery in this region has been mistakenly diagnosticated as retropharyngeal abscess, a fatal issue following the incision. The pulsation and bruit present in aneurysm should be sought for in all cases of suspected abscesses of the pharynx. The pulsation may be noted with the eye or finger, while the bruit may be distinguished with the stethoscope introduced through the mouth.

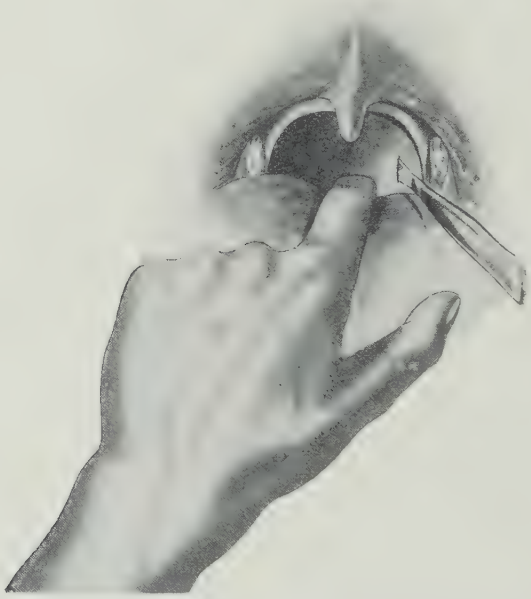
Malformation of the posterior wall of the pharynx, causing bulging of one side, is occasionally found. The hard, firm character of the mass readily distinguishes it from the soft baggy mass which is present in abscess formation.

Acute infectious inflammations of the pharyngeal mucous membrane

sometimes simulates retropharyngeal abscess. The difference in the resistance upon digital examination will determine which of the processes is present.

Prognosis.—The danger in very young subjects is chiefly due to suffocation, and to strangulation upon the spontaneous rupture of the abscess. In older patients this danger is not so great, as their reflexes enable them to ward it off or to anticipate it. Under treatment the prognosis is nearly always good except when the disease is due to tuberculous caries of the vertebræ.

FIG. 258



The oral operation for retropharyngeal abscess. The finger is used as a guide to the fluctuating area and as a tongue depressor, while a short-bladed scalpel is used to open the abscess.

Treatment.—The most important object to be accomplished is the immediate evacuation of the pus. This may be done by (*a*) the internal or (*b*) the external route. The internal operation should always be tried first, and followed by the injection of iodoform glycerin emulsion (Esmarch and Kowalzig). Should simple puncture and evacuation, followed by the injection of the iodoform emulsion, fail, the external operation should be performed.

Technique.—*Internal Operation.*—(*a*) Place the patient upon a table with his head lowered to prevent the larynx being bathed in pus. With children this precaution is especially urgent, because their reflexes are not sufficiently trained to prevent suction of the infected secretion into the trachea and lungs, where it might cause aspiration pneumonia.

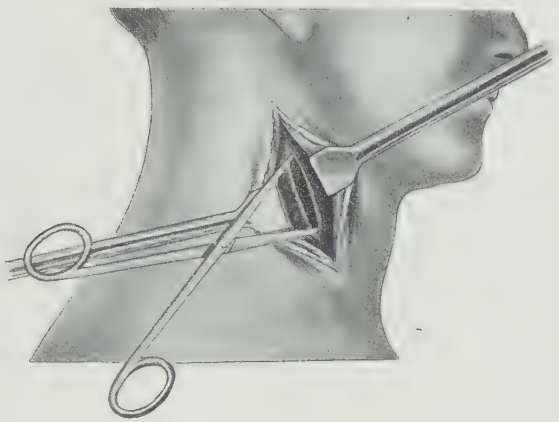
(*b*) Introduce the left index finger into the mouth and place the tip against the soft fluctuating tumor.

(*c*) Introduce a short-bladed scalpel, or a longer one, the proximal end of which is wrapped with a strip of adhesive plaster or cotton, into the mouth, using the above-mentioned finger as a guide (Fig. 258).

(*d*) Incise the abscess wall by the side of the finger. The pus then flows through the incision into the pharyngeal cavity, from which it may be removed with moist gauze sponges, grasped by artery forceps; or it may be expectorated by the patient.

(*e*) After all the pus has been thus removed, irrigate the cavity with warm boric acid solution and inject the iodoform glycerin emulsion into the wound. The injections may be repeated every day or two, and if steady improvement follows, a cure may be expected. If, however, improvement does not follow, the external operation should be performed.

FIG. 259



The external operation for retropharyngeal abscess. The fascia enclosing the abscess is punctured and opened with artery forceps.

External Operation.—Generally speaking, the external operation consists in making an incision either anterior or posterior to the sternomastoid muscle, and extending it inward by blunt dissection to the anterior wall of the vertebral column, where the abscess cavity is located.

If only the retropharyngeal abscess is to be included in the operation the incision should be made posterior to the sternomastoid muscle; if, however, there are diseased cervical glands to be removed at the same time, the incision should be made anterior to the muscle (Fig. 259).

The following steps in the operation should be observed:

(*a*) The field of operation should be shaved and cleansed.

(*b*) General anesthesia.

(*c*) An incision two or three inches long should be made through the skin over either the anterior or the posterior border of the sternomastoid muscle on a plane with the retropharyngeal abscess. The dissection

should be continued until the deep cervical fascia is opened and the border of the sternomastoid muscle is brought to view.

(*d*) The sternomastoid muscle is then separated by blunt dissection from the adjacent tissues, and is drawn forward with a retractor to expose the operative field.

(*e*) Still using blunt dissection, the carotid sheath, with its vessels and nerves, is separated from the vertebra and carefully drawn forward.

(*f*) The dissection is carried in front of the vertebra to the abscess wall.

(*g*) The abscess wall is punctured with closed artery forceps; the forceps is then introduced into the cavity, the blades spread apart, and withdrawn from the cavity (Fig. 259). The abscess is thus freely opened and evacuated.

(*h*) Digital examination of the cavity should be made for necrosed bone, and to note the condition of the soft tissues and abscess contents. If the secretions are thick and caseous, they may be removed by gentle curettage.

(*i*) Irrigation with warm boric acid or the glycerin-iodoform solution completes the evacuation of the contents of the abscess.

(*j*) Introduce a cigarette drain into the wound. This may be withdrawn a little each day after the discharge has ceased, and its use may be abandoned altogether at the end of ten days or two weeks, after which the external wound closes from the bottom by granulation.

If cervical glands are to be removed, or if the abscess points anteriorly to the sternomastoid muscle, the incision should be made anterior to the muscle. The group of glands involved should be removed *en masse*, as to leave some of them almost surely means a secondary operation.

MALFORMATIONS OF THE PHARYNX; STENOSIS OF THE PHARYNX

Malformations of the pharynx may be either (1) congenital or (2) acquired.

Those of congenital origin may be in the form of an imperforate pharynx, from a failure in the embryological development of the anterior end of the foregut, and the invagination of the ectoderm, which forms the cavity of the mouth. The embryological structures in this region are very complex, and it is remarkable that congenital malformations are not more frequent. They usually occur in the form of a constriction or pouch, or of a complete closure.

Acquired malformations are due to inflammatory and degenerative changes in the walls of the pharynx. Syphilis is the most common cause. In the tertiary stage there is more or less destruction of the uvula and soft palate, which is followed by cicatricial contraction and adhesion to adjacent parts. The soft palate in these cases is usually adherent to the posterior wall of the pharynx, and may cause most complete separation of the mesopharynx from the epipharynx. In one of my cases due to congenital syphilis there was a small opening, about the size of a

lead pencil, which communicated with the epipharynx. The scars in syphilis are stellate in their arrangement, *i. e.*, they radiate from the site of the original ulceration. The ingestion of scalding fluid and caustic drugs may produce scar tissue and cicatricial contraction. (See Syphilis of the Pharynx.)

Treatment.—The treatment of syphilitic scar tissue and adhesions result in failure in the majority of cases. The scar tissue may be removed and the adhesions broken down, though they speedily reform and readhere. Obturators have been used in the isthmus between the mesopharynx and epipharynx, to keep the channel open and to prevent adhesions, with occasional success. The tendency for syphilitic scar tissue to reform, in spite of all that can be done, is the chief hindrance to the successful treatment of these cases. If the constriction involves the hypopharynx and dyspnea develops, tracheotomy should be performed.

CHAPTER XIX

FUNCTIONAL NEUROSES OF THE PHARYNX

Neuroses of Sensation.—The train of symptoms in pharyngeal neuroses of sensation is about the same as in the larynx, many of them being due to reciprocal lesions. (See Neuroses of the Larynx.)

Anesthesia of the pharynx is not of any great clinical significance, excepting, perhaps, when it accompanies progressive bulbar disease.

Insane patients generally have it, even though no form of paralysis is present in the pharynx or elsewhere in the body. In cases of marked anesthesia involving the whole pharynx, the soft palate and larynx are usually likewise anesthetic. Diphtheria often causes it, and sometimes it accompanies the other exanthematous fevers. It may be present in local inflammations of the pharyngeal mucosa. (For treatment, see Anesthesia of the Larynx.)

Hyperesthesia of the pharynx is the most frequent of the pharyngeal neuroses. It often occurs in those who are otherwise healthy. These cases do not tolerate the laryngoscopic mirror in throat examinations. They also resist the introduction of the Eustachian catheter. The most sensitive areas in the pharynx are the arch of the soft palate and the vault of the epipharynx.

Hypersensitiveness accompanies both acute and chronic inflammation of the pharynx. It is also a frequent manifestation of hysteria. It is more common in men than women. Habitual smokers and drinkers are subject to it. It is but rarely a symptom of central brain disease. The hypersensitive areas sometimes appear on the tongue.

Paresthesia occurs about as frequently as anesthesia, and less frequently than hyperesthesia, and often baffles the skill of examiners and operators. Tonsillar disease is often the cause of it, hence these organs should be thoroughly examined for diseased conditions. The passage of a bolus of food or foreign body may cause an abrasion, which may be followed by the sense of a foreign body in the throat. The menopause is frequently attended by perverted sensations in the pharynx. Patients at this period sometimes complain of the sensation of a rope or hairs in the throat. Hyperplasia of the lingual tonsil seems in some cases to cause it. The same is true of elongation of the uvula, though the elongated uvula is usually a sign of epipharyngitis, and the paresthesia may be due to the "dropping" from the epipharyngeal region. Granular pharyngitis, especially when it involves the lateral walls (pharyngitis hypertrophica lateralis), gives rise to an irritation between the posterior pillars and the pharyngeal wall, which is sometimes accompanied by paresthesia. It is occasionally associated with globus hystericus.

The perverted sensations complained of are cold, heat, a foreign body, itching, tickling, and the dislocation of the essential parts of the fauces

and pharynx. Patients sometimes complain of swallowing the soft palate, etc. Most of the female cases seen by me have suffered from melancholia during the menopause, and have had a suicidal tendency. One patient committed suicide by drowning some months after she passed from under my observation. The paresthesia may be so marked as to cause a distressing cough and laryngeal or esophageal spasm.

Neuralgia of the pharynx is difficult to differentiate from muscular rheumatism. Neuralgia is not painful upon pressure, while rheumatism is painful with or without pressure. Anemia and chlorosis are often the cause of neuralgia, whereas rheumatism is more often associated with plethora. Enlarged single pharyngeal follicles may become so painful as to simulate neuralgia. Localized pressure upon the follicles causes pain in rheumatic pharyngitis.

The treatment of neuralgia should be addressed to the cause when it can be determined, as well as to the relief of the pain. Iron, strychnine, arsenic, bitter tonics, and the regulation of the bowels should be the basis of the treatment in those cases in which anemia is the cause. In chlorosis, enemata should be given to unload and cleanse the rectum and lower bowel, to stop the absorption of putrefactive material and bacteria into the circulatory system. Exercise in the open air, especially upon cloudy days, is of the greatest value in these cases. Excessive exposure to sunshine is injurious, as it is too stimulating. Oxygen rather than sunshine is the desideratum. Patients should be encouraged to play golf or other outdoor sport, or to work in the flower or vegetable garden, or in the poultry yard. The outdoor exercise should have a constant and alluring motive, or it will soon be abandoned.

Neuroses of Motion.—Neuroses of motion of the pharyngeal muscles may, like that of the larynx, be divided into two general classes:

1. *Akinesis*, or paralysis, which may be unilateral or bilateral. The akinesis, or paralysis, may be still further subdivided into: (*a*) Paralysis due to bulbar disease (central paralysis). (*b*) Paralysis due to diphtheria (peripheral paralysis). (*c*) Paralysis due to or complicating faucial paralysis (central or peripheral paralysis). (*d*) Paralysis of the pharyngeal constrictors (Lennox Browne).

2. *Hyperkinesis,* or spasm.

Paralysis Due to Bulbar Disease; Central Paralysis.—The following central lesions may give rise to pharyngeal paralysis; acute and chronic bulbar myelitis, hemorrhage, tumors, embolism, and basilar meningitis.

Acute Bulbar Paralysis; Central Paralysis.—*Symptoms.*—In acute bulbar myelitis the symptoms develop rapidly, a fatal issue soon following. The symptoms are as follows:

(*a*) Sudden attack.
(*b*) Severe headache.
(*c*) Dysphagia.
(*d*) Respiratory embarrassment
(*e*) Difficulty in articulation.
(*f*) Giddiness.
(*g*) Unsteady gait.

Prognosis.—The prognosis is extremely grave.

Treatment.—While these cases are almost necessarily hopeless, they should be treated, as there is a chance that the diagnosis may be erroneous. Bloodletting by cupping or leeching should be early and freely done to relieve the inflammatory process at the base of the brain. Ice should be applied to the pharynx and to the nape of the neck. The blood tension should be lowered by the administration of cathartics and such other remedies as are employed for spinal myelitis.

Chronic Bulbar Paralysis; Central Paralysis.—Undue exposure to cold, prolonged violent excitement, extreme fatigue, and lack of nutrition are etiological factors. Heredity seems also to largely influence its occurrence. It is more common in males than in females, and is rarely observed before the age of thirty-five. In rare cases it may be due to an injury or to sunstroke. Syphilis and tuberculosis should also be included as causative agents.

Symptoms.—Pharyngeal paralysis may be the first symptom of progressive bulbar disease, though the tongue is usually the first organ affected. The tongue is first involved in a typical case, and this is followed by paralysis of the lips and of the pharyngeal and laryngeal muscles. This order of involvement is almost always present. The paralysis, at first slight, gradually increases in severity.

Diagnosis.—In the beginning the disease may be mistaken for bilateral facial paralysis, though the history of a sudden onset, followed by progressive chronic paralysis of the tongue, pharynx, and larynx, together with the lips, should render the diagnosis of bulbar paralysis almost certain. In bilateral facial paralysis the tongue, pharynx, and larynx are not affected. In rare cases the tongue and fauces are not involved.

Prognosis.—The prognosis is usually grave, though there may be remissions before death occurs. Patients often succumb to inanition or pneumonia.

Treatment.—Galvanism has been used to combat the degeneration of the nerves, and faradism to maintain the muscular vigor, with but little success. Strychnine and arsenic are of greater value. In syphilitic cases the iodides are indicated.

Diphtheritic Paralysis; Peripheral Paralysis.—Paralysis of the pharyngeal muscles is often an early sequel of diphtheria and of pseudomembranous sore throat. The muscle fibers undergo more or less degeneration from the presence of the bacterial toxins, and there is a mechanical hindrance from the cellular infiltration of the tissues. In addition, there is a degeneration of the peripheral nerve fibers from the same causes.

Symptoms.—The voice undergoes great changes on account of the paralysis of the pharyngeal muscles, as they are utilized in articulation and voice placement. The voice has the so-called "nasal quality," closely resembling that present in cleavage of the hard and soft palates. The velum and uvula are relaxed and can only be raised by forced inspiration. One side or both may be affected. The paralysis occurs on or about the fifteenth day after convalescence, at which time ocular symptoms may also develop.

Treatment.—The prophylactic treatment consists in the administration of antitoxin during the diphtheria. After the paralysis has developed, galvanism, faradism, and rectal feeding should be adhered to in order to maintain muscular and nervous tone while the degenerated nerve fibers are being restored. Thick soups, grape juice, etc., may be given per rectum.

Paralysis of the Pharynx Complicating Facial Paralysis.—According to Ziemssen and Bouche, when the lesion is above the geniculate ganglion, the pharyngeal is often associated with facial paralysis. The uvula does not move upon phonation and is deflected to one side. The symptoms are the same as those in diphtheritic paralysis, and include such structures as are supplied by the seventh nerve.

Paralysis of the constrictor muscles of the pharynx is always accompanied by paralysis of the esophagus. The dysphagia is, therefore, exceedingly well marked, and is often the only distinctive symptom.

Hyperkinesis, or Spasm of the Pharynx.—*Etiology.*—Spasm of the muscles of the pharynx is a rare affection. It may occur from insignificant causes, as uvulitis, foreign bodies, globus hystericus, enlarged pharyngeal follicles, neuralgia, and local chronic inflammations, or it may be an early symptom of a serious central lesion.

The more dangerous form of spasm of the pharynx is encountered in hydrophobia, edema of the glottis, brain tumors, paralysis agitans, and other affections of the nerves.

Symptoms.—Chronic spasm of the pharynx involving the soft palate and uvula may be the chief symptom. The levator palati is the muscle affected. The spasm of this muscle draws the soft palate upward a number of times in rapid succession, after which it relaxes. During the spasm there is a clicking noise as the palate leaves the pharyngeal wall. The click is audible to those near by.

Prognosis.—The prognosis is fair in those cases due to simple causes, provided appropriate treatment is instituted. If due to a serious central lesion, hydrophobia, edema of the glottis, brain tumor, or paralysis agitans, it is grave.

Treatment.—If the spasms are due to a foreign body, it should be removed. If due to local inflammation, appropriate remedies, elsewhere described, should be used. When due to saprophytic absorption from the rectum, the lower bowel should be flushed by enemata, outdoor exercise advised, and a nutritious but unstimulating diet followed. When due to hydrophobia, this should be treated rather than the spasms of the pharynx which are incidental to the disease. Stimulants of any sort should be avoided in all cases.

CHAPTER XX

NEOPLASMS OF THE PHARYNX

BENIGN NEOPLASMS

(*a*) **Papillomata.**—Papillomata rarely occur on the walls of the pharynx, but are common in the faucial region. They are most frequently found upon the uvula, the free borders of the pillars of the fauces, and the tonsils. The histological differences between the mucous membrane of the posterior wall of the pharynx and the mucosa of the uvula, pillars, and tonsils account for the location of the tumors. The posterior wall of the pharynx is covered by squamous epithelium, whereas the other structures are covered by columnar, and in many places by columnar ciliated epithelium. In spite of the varying structural differences, papillomata appear in all parts of the pharynx and fauces, though more frequently in the fauces.

They may be single or multiple, sessile or pedunculated. Behind the fauces, or in the pharynx proper, they are rarely pedunculated, and are chiefly limited to the ragged excrescences following syphilitic and lupous inflammations. Papillomata are composed of elevations of epitheliat cells which contain a connective-tissue core more or less richly supplied with bloodvessels. The epithelial elevations grow outward, while in epitheliomata they grow inward. The elevations vary from tumors as small as a pinhead to those of considerable size. They often contain "pearls" or "nests," which may be mistaken for the nests or pearls of epitheliomata. The cells in papillomata are uniform, whereas in epitheliomata they are multiform. Epitheliomata are likely to be mistakenly diagnosticated as papillomata, and *vice versa*.

Primary papillomata are usually surrounded by an inflammatory area. Secondary papillomata are the result of a preëxisting inflammation, as syphilis (Fig. 260).

The presence of a papillomatous growth in the fauces or pharynx often excites a reflex cough, with a sense of fulness and tickling in the throat.

Treatment.—The treatment of papilloma of the pharynx is usually so simple that a detailed description of the procedures need not be given. The tumor should be removed to its base with a knife, snare, cutting forceps, or cautery. The base of the growth should be removed or cauterized with a solid stick of nitrate of silver or the galvanocautery. If this is not done they are likely to recur.

(*b*) **Teratomata.**—Lennox Browne says: The connection between teratomata and cystomata is so intimate and their origin so obscure

that it is expedient to describe them together. I shall not do this, but will attempt to characterize them as distinct pathological entities.

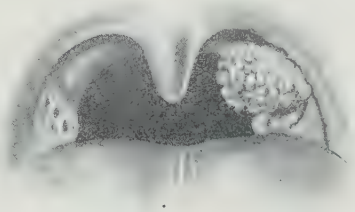
Teratomata are usually congenital and consist of tissue growths springing from two or three embryological germinal layers. They appear, therefore, most frequently in those regions where the various germinal layers are in close apposition (Browne). The pharynx, which develops from the junction of the neural and the dermal epiblasts with the hypoblasts of the foregut is, therefore, a suitable location for the growth of teratomata. Bland-Sutton called attention to this fact in 1886.

Teratomata generally occur in the epipharynx, though in quite a few recorded cases they were in the meso- and hypopharynx. They were sometimes called "hairy pharyngeal polypi," as they are usually pedunculated cysts filled with hair and other histological structures.

Conitzen reported 11 "hairy polypi," or teratomata, which were cystic and contained hair, cartilage, skin, and bone. The cysts are usually pedunculated, and may be attached to any part of the pharynx.

Treatment.—The treatment consists in the removal of the growth with the snare, knife, or cautery. Cauterization of the base seems to prevent recurrences.

Fig. 260



Author's case of follicular tonsillitis and syphilitic papilloma arising from the left supratonsillar fossa.

(*c*) **Cystomata.**—These usually occur after the twentieth year of life, more often in middle and advanced age. They are usually retention cysts or mucoceles, due to the closure of the mouths, of the pharyngeal follicles, either by inflammatory contraction, epithelial plugs, or by the flaccid folds of membrane in advanced life. The cysts contain a glairy fluid, though in some cases it is inspissated and much thickened. They are usually superficially located, though Raugi speaks of the occurrence of a cyst in the submucous tissue. This tumor was difficult to see, and he thinks this type must occur much more often than is generally believed.

Cysts are usually sessile, and often give rise to the symptoms described under reflex neuroses, as asthma, migraine, etc.

Treatment.—The treatment consists in the enucleation of the cyst membrane, though thorough cauterization of the lining of the sac is usually followed by the obliteration of the tumor.

(*d*) **Lymphomata, or Lymphadenomata.**—This variety of benign tumor is the most frequent growth in the pharynx. This is to be expected on account of the widely disseminated lymphoid tissue and the numerous lymphoidal vestiges. The matrix of the tumor is connective tissue, in the meshes of which are aggregated the lymphoid cells. The cell groups are often crowded together and vary greatly in size. They, like lymphoidal tumors elsewhere, have a strong tendency to multiply.

They may be attended with or may follow mediastinal complications of a like nature (Villar). A single tumor, especially when pedunculated, at times offers some diagnostic difficulties. But when we take into consideration that the adjacent lymphatic glands in the neck are enlarged and soft, the tumor in the pharynx, though pedunculated, should be suspected to be lymphomatous.

(*e*) **Myxomata.**—Myxoma of the pharynx is exceedingly rare. Browne in his large experience never saw a case. Closely allied to them, however, are the so-called mucoceles due to dilatations of the mucous glands. The mucoceles are important as they are readily recognized and are easily eradicated by excision or the actual cautery.

(*f*) **Fibromata.**—The structural arrangement is often so like that of sarcomata that it is difficult to differentiate them. The clinical history is, therefore, the guide in diagnosis. In very rare instances a myxomatous tumor may have the tendencies and aspects of a fibroma, just as primary fibromata may become mucoid in character. Fibromata are rare in advanced age, but are quite common in young and middle adult life. This seems to be true of nearly all neoplasms springing from the mesoblast.

Fibromata may be sessile, but are more often pedunculated. They are composed of densely packed spindle cells, with an undeveloped matrix of connective tissue. They are encapsulated, and often attain a large size. Bruns reports a case in which the entire fauces was filled by a fibroma. They are usually single and of slow growth. They have their origin in the fibrous tissue and the periosteum of any part of the pharynx. The covering of the basilar process of the occipital bone and body of the sphenoid are favorite sites. As the pterygoid plate of the sphenoid and the perpendicular plate of the palate bone, the posterior ends of the upper turbinated bodies, and the posterior portion of the vomer are all covered with fibrous tissue and periosteum, fibromata usually develop in this region. Large fibromata are frequently attended with inflammatory processes, hence adhesion to the adjacent structures is common.

Etiology.—They are rare in females. Age is a decided factor in their occurrence, adolescence being the favorite period. As age advances there is a tendency for the growths to recede or undergo spontaneous cure. In this respect they resemble adenoids and other lymphatic enlargements.

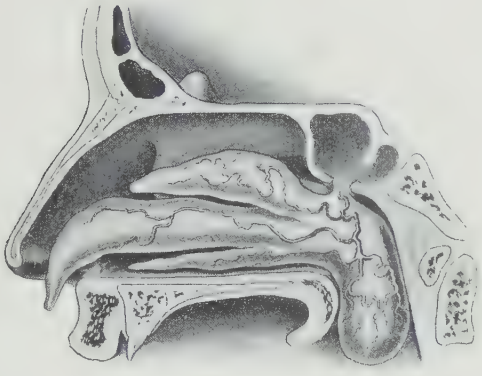
Symptoms.—The early symptoms are those of epipharyngeal catarrh, with more or less hemorrhage. The bleeding sometimes becomes an alarming complication. The voice becomes "flat" or "dead" in quality, and respiration and deglutition are impeded as the process advances. At a later stage, there is pain and mucopurulent discharge. When the growth has attained considerable size, the "frog face" becomes well marked, the maxillary bones are separated, and exophthalmos becomes a prominent symptom. Aprosexia and drowsiness are often present. In one of the author's cases the patient often dropped into sleep or slight stupor while in the treatment chair. Greville Macdonald reports vomiting as an annoying symptom.

If the growth extends upward it may encroach upon the cranial contents and give rise to such symptoms as paralysis, etc.; this is followed in nearly every instance by death.

The foregoing symptoms increase in severity as the growth extends, until the absorption of bony tissue is considerable, unless the tumor extends beyond the nasal and pharyngeal chambers, as into the cranial cavity. In this event the pressure necrosis of the bony tissue is not so great.

Examination shows the tumor to be a rounded mass, of a pinkish or dark purple color. The veins are frequently varicosed, hence the examination by digital or instrumental aids should be done carefully, to avoid injuring them. The growth may project into the posterior nares, or its direction may be toward the antrum and other sinuses. Under finger pressure it is firm and elastic, and if small its base may be out-

FIG. 261



Author's case of soft fibroma of the epipharynx, springing from the base of the sphenoid and sending finger-like prolongations into the nasal chambers.

lined. If pedunculated, it is movable, unless it has become fixed by inflammatory adhesions. If it extends through the sphenomaxillary fissure, it may be felt under the zygoma. As adhesions are usually present, its outline is difficult to distinguish.

In Fig. 261 is shown a soft fibroma of large size in a lad aged fourteen years. It had its origin from the base of the sphenoid bone and extended into the nasal chambers by three finger-like processes. Numerous inflammatory adhesions were present around the choanæ. A general surgeon of repute made five futile attempts to remove the growth because he was not intimately familiar with the anatomy of the pharynx. (See Treatment.)

Diagnosis.—The histological resemblance to sarcoma is often so close that a differentiation is difficult, unless the age, sex, and origin are such as to point to its fibrous nature. Sarcoma is rarely or never, whereas

soft fibroma is frequently, pedunculated. Hard fibromata are usually sessile.

Prognosis.—The prognosis is favorable in proportion to its early recognition and extirpation. It is also favorable when the age of the patient exceeds twenty-five years. In other words, small fibromata which do not fill the epipharyngeal space are more favorable under operative treatment than those which completely fill it. The tendency of the growth to undergo retrograde changes after the twenty-fifth year accounts for the more favorable prognosis in those cases in which it occurs after this period.

Some patients even recover spontaneously. It is advisable in nearly all cases to remove the growth by surgical interference, as it is too great a risk to wait for a spontaneous cure. An additional reason for operating is to relieve the patient as speedily as possible of the pain and other distressing symptoms characteristic of these growths.

Treatment.—Small growths, especially if they are pedunculated, and those limited to the epipharyngeal space may be removed with a heavy snare or ecraseur, either through the nose or mouth, or with adenoid forceps. The galvanocautery snare may also be used through these routes. When the growth is large and sessile, and has extensive inflammatory adhesions to the adjacent structures, it may be necessary to perform an external or more radical operation. Large soft pedunculated fibromata like the author's case shown in Fig. 261 may be removed as follows:

(*a*) Prepare the patient as for a major operation. General anesthesia.

(*b*) Place the patient in Rose's position.

(*c*) Be prepared to ligate the external carotid artery, and to introduce postnasal tampons. In the author's case, the hemorrhage was very great, and necessitated the ligation of the external carotid artery. Respiration ceased at the same time, and artificial respiration was practised while the carotid artery was being ligated. The hemorrhage occurred when the inflammatory adhesions around the choanæ were being broken down with the finger. The patient was emaciated and anemic, which doubtless rendered the bleeding more severe.

(*d*) Break down the inflammatory adhesions around the choanæ with the finger, which should be introduced through the mouth.

(*e*) Introduce curved pharyngeal scissors (Fig. 248) through the mouth into the epipharynx posterior to the body of the tumor until the pedicle of the tumor is reached, and sever it if possible. If the tumor is very large, this may not be possible. In the case shown in Fig. 261, I succeeded in partially severing the pedicle.

(*f*) If the pedicle cannot be severed with the scissors, introduce strong, slightly curved adenoid forceps through the mouth into the vault of the epipharynx, seize the pedicle, and cut it from its attachment to the base of the sphenoid bone. By this method I removed the fibroma shown in Fig. 261. The growth was removed through the mouth; the finger-like extensions into the nasal chambers came away with the body of the tumor, as the adhesions within the nose were not firm.

The patient made a slow, but uneventful recovery, and two years after the operation is in excellent health.

(*g*) **Lipomata.**—Lipomata of the pharynx are rare. When they occur they are usually small and sessile, especially when they develop from dense tissue. When they spring from loose tissue they may attain large size, and are generally pedunculated and multiple. They are oval, smooth, and elastic, hence are sometimes mistaken for retropharyngeal abscess. A puncture readily clears the diagnosis. They usually occur in advanced age. Lennox Browne says that the sessile and deeply seated ones are more often congenital than otherwise.

(*h*) **Angiomata.**—Because of Cruveilhier's submucous plexus, situated at the back of the pharynx, and the rather rich, both superficial and deep, blood supply, we might naturally expect many angiomata. But, on the contrary, they are of rare occurrence. Moritz Schmidt does not cite a case in his excellent review of the tumors of the upper respiratory tract. Guyon had one patient in whom digital examination caused profuse hemorrhage. Electrolysis checked the hemorrhage, and subsequently caused atrophy of the growth. Angiomata of the pharynx, like similar growths elsewhere, are usually cavernous and often erectile in character. Farlow reports five cases of enlarged pulsating arteries in the pharynx. The red currant-like clusters occasionally seen in the pharynx are, strictly speaking, angiomatous.

Treatment.—Most physicians favor non-interference unless the tumors bleed. This attitude is attended by some risk, because a serious hemorrhage may occur at any time. If large, they should be deprived of their arterial blood supply by ligatures around the efferent vessels. If small, they may be treated by electrolysis or by ligation.

Electrolysis is performed as follows: (*a*) Anesthetize the tumor with local applications of a 10 per cent. solution of cocaine.

(*b*) Introduce the needles, connected with the positive pole of the galvanic battery, into the growth.

(*c*) Turn on from 10 to 25 ma. of current for five minutes. Repeat the seances at intervals of about seven days until the growth is obliterated.

The positive pole of the battery liberates nascent oxygen and coagulates the tissue, hence it is the pole which should be applied to a soft growth. If it is desired to reduce a hard or fibrous tumor, the negative pole is applied to the growth, because it liberates hydrogen, which softens the tissue.

Ligation or strangulation may be performed as follows: (*a*) Anesthetize the growth by the local application of a 10 per cent solution of cocaine.

(*b*) Pass a ligature through the tissues, including an artery at the margin of the angioma, and tie it. Yankauer's needles should be used.

(*c*) Continue thus to tie off the larger vessels until the nutrient sources are closed.

(*d*) After three or four days the ligatures should be removed.

MALIGNANT NEOPLASMS OF THE PHARYNX

General Pathology.—Clinically, it is an advantage to make a distinct demarcation between the fauces and the pharynx in treating malignant growths. However, as is well known, their tendency to spread by continuity of tissue and by metastasis, and their insidious beginning, does not permit of an absolute anatomical division. Oftentimes they originate on the borderline between the two regions. It should be borne in mind that when these tumors spring from the larynx they are likely to extend to the pharynx, but that those arising from the pharynx seldom, if ever, extend downward to the larynx. Even those which occur in the hypopharynx have an upward rather than a downward tendency. This is partially explained by the difference in the tissues composing the two parts. In the larynx there is little soft tissue, and the glandular element is less, whereas in the pharynx the soft tissues and lymph glands are more abundant.

Embryologically, the pharynx and the larynx have different origins, and the tendency to extension is thereby somewhat impeded.

The general symptoms are much the same as those of cancer of the larynx. The special symptoms are dependent upon the anatomical and physiological differences in the two regions.

The lower portion of the pharynx is more often the seat of malignancy than the upper. Men are more often affected than women. Carcinomata here, as elsewhere, are more frequent in the old. This is in obedience to the physiological law, that mesoblastic structures are more active in the young, while the epi- and hypoblastic structures are more active in the old. An effort is made by some writers to differentiate between the malignancy of sarcoma and carcinoma. This is of no practical or clinical value, as either is usually the cause of death in whomsoever it occurs. True carcinoma, because of its glandular structure, more readily involves contiguous structures, and more frequently extends by metastasis.

Carcinoma of the pharynx is more frequent than sarcoma. The former is more likely to involve the glandular structures, subjected as it is to persistent irritation, especially in the pharynx. Sarcoma may, however, be due to traumatism.

It is often difficult to differentiate profuse scar tissue from sarcoma, as both are closely allied to embryonal tissue. The clinical phenomena are, therefore, often more reliable than the microscopic findings.

Varieties of Sarcoma.—The various types of sarcoma which appear in the pharynx in their order of frequency are:

1. Round-cell sarcoma.
2. Spindle-cell sarcoma.
3. Myxosarcoma.
4. Lymphosarcoma.

1. **Round-cell Sarcoma.**—The round-cell sarcomata are of two types: (*a*) Large round-cell sarcoma, and (*b*) small round-cell sarcoma. Their

structure is characterized by an aggregation of cells, intercellular cement, and numerous bloodvessels. Occasionally a few fibrous trabeculæ are distributed through the mass of cells. Lymph channels are also found in the cellular masses. The cells vary considerably according to their age and original site of growth. The older part of the tumor is in a state of degeneration, while the newer part is intact. The small round-cell sarcoma is softer than the large round-cell growth, which has more intercellular cement substance. The cells of the large round-cell sarcoma often have oval nuclei, and form the most malignant type of sarcoma. Its local ravages are extensive and the constitutional manifestations are severe.

2. **Spindle-cell Sarcoma.**—This, like the round-cell variety, is divided into two classes: (*a*) Small spindle-cell sarcoma, and (*b*) large spindle-cell sarcoma. The general structure of this variety is quite like the round-cell sarcoma except that the cells are often arranged in bundles. Lymph spaces are absent, whereas they are present in the round-cell variety. The vascular supply is accordingly greater than in the round-cell variety. Many spindle-cell sarcomata have a tendency to undergo degeneration in patches, and are less malignant than the round-cell variety. The spindle-cell sarcoma more often occurs in adults, while the round-cell variety is more often present in the young. The spindle-cell sarcoma develops slower than the round, is firmer, and less likely to ulcerate. It may be pedunculated, while the round-cell variety is seldom or never pedunculated. It is encapsulated and "shells out," while the round-cell is not encapsulated.

The local malignancy is greater than in the round-cell variety, while the general malignancy is not so great. The spindle-cell sarcoma usually springs from the posterior wall of the pharynx, though it may arise from the lateral wall.

3. **Myxosarcoma.**—Myxosarcoma is originally either spindle-cell or round-cell, which, having undergone an early mucoid change, is converted into the myxomatous type. It is locally malignant, rather than constitutionally; that is, it has a tendency to recur, but seldom gives rise to metastasis. It arises most frequently in the loose cellular tissue of the lateral wall of the pharynx, though it may occur in the fauces and the glosso-epiglottic fold.

4. **Lymphosarcoma.**—Lymphosarcoma is a variety of round-cell sarcoma. It possesses a very delicate reticulum, which gives it the appearance of a lymphoid structure. It usually originates in the lymphoid tissue of the pharynx, which is, perhaps, another reason for its resemblance to normal lymphoid or adenoid tissue. When the growth is traversed by numerous fibrous connective-tissue bands, it is more dense in structure. It is necessary to differentiate this neoplasm from benign hyperplasia and lymphoma, which is directly due to inflammatory processes.

Lymphosarcoma grows rapidly, and when removed invariably recurs. It usually involves everything in its course, especially that type which starts in the lymphatic glands. Pharyngeal lymphosarcoma is quite often observed in Hodgkin's disease, which is a true lymphosarcoma.

TRYPSIN TREATMENT OF MALIGNANT NEOPLASMS

According to J. T. Campbell, the trypsin treatment of malignant neoplasms is based upon the statistical findings of von Bergmann, wherein he states (1) that cancer of the stomach stops abruptly at the pylorus; (2) that the small intestine is but rarely the site of cancer; and (3) that cancer of the large intestine and rectum for the most part increases in frequency the farther the distance from the duodenum. In 10,537 cases of cancer of the alimentary tract the stomach was involved 4288 times, the small intestine 20, the large intestine 224, and the rectum 1204 times. The natural and comparative immunity of the duodenum and small intestine, together with the slower rate of growth of cancer of the large intestine, would, therefore, appear to support the treatment of inoperable cancer by preparations of the pancreas, bile salts, intestinal gland extracts, and ferments alone or combined. In November, 1905, Dr. Wade, at the solicitation of Dr. F. Beard, began experiments, first, to determine the action of trypsin upon the living cells of carcinoma, such as Jensen's mouse tumor (an adenosarcoma); second, to test the truth of the conclusion advanced by Beard in 1902 that cancer was an irresponsible trophoblast; and third, the length of treatment and number of injections of trypsin necessary to destroy the tumor.

The results were such as to appear to show that the trypsin caused a degeneration of the cancer cells, a shrinkage of the tumor, and an improved condition of the system in general. Since then several cases of cancer in the human body have been reported wherein trypsin caused apparent shrinkage of the growth, a cessation of the pain, marked gain in weight, and great improvement in the health of the patients. It appears, however, that the improvement was temporary, in some of the cases a recrudescence of the neoplasm occurring later, with a rapid fatal termination. It is too early to accurately judge the merits of the trypsin treatment. It is, however, worth trial in inoperable cases. An operable case should be operated early and thoroughly. Delay and partial removal by operation are dangerous procedures. An early operation and complete removal offer the best chance of a cure. The operation may be followed by the trypsin treatment.

Technique of Trypsin Treatment.—The trypsin comes in sealed ampoules, of 20 minims each, of a glycerin extract prepared from the pancreatic glands, and with such a proportion of the ingredients of the normal salt solution that when diluted with two volumes of sterilized distilled water the medium corresponds in this respect to the normal salt solution; greater dilution may be employed if desired.

At first 5 minims of the trypsin solution diluted with 10 minims of sterilized distilled water should be injected through the skin of the buttocks deep into the subcutaneous tissue, but not into the muscles. The injections may be given every other day, gradually increasing the dose to 10 minims.

The skin should be cleansed with soap and alcohol, and in sensitive patients 0.1 grain of eucaine may be injected a few minutes before the injection of the trypsin.

Some writers recommend the administration of holadin in 3 grain capsules three times a day during the trypsin injections. Holadin is an extract of the entire pancreas gland, containing all the constituents of the digestive and internal secretions of the gland.

EXCISION OF THE EXTERNAL CAROTID ARTERY AND ITS BRANCHES FOR INOPERABLE CANCER OF THE UPPER RESPIRATORY TRACT

The excision of both external carotid arteries and their eight branches may be performed for the purpose of depriving inoperable malignant growths of the nose and pharynx of their blood supply, thereby starving the growths. Malignant tumors require a large blood supply, hence this operation seems to offer some degree of hope. Dawbarn reports encouraging results in a number of cases of inoperable cancer of the head. The operation should never be performed when the growth can be successfully extirpated. The ligation of the external carotids and their branches should be adopted as a last resort. While it may not cure the case, it may prolong life.

Position of the Head.—The shoulders should be placed upon a block or sand cushion, the chin well elevated and everted to the opposite side to expose the region of operation.

Incision.—The incision should extend from the tip of the mastoid process close behind the angle of the jaw to the level of the middle of the larynx. At either extremity the incision is exactly over the external carotid artery. Dawbarn recommends that the incision be curved medianward about 1.5 cm., as the safety of the operation lies anterior to the artery, while danger lies posterior to it.

Exposure of the Artery.—Work from below upward, first exposing the superior thyroid artery, which extends downward to the thyroid gland. By tracing this back to the carotid the external is distinguished from the internal. Pass a chromicized catgut loosely around the external carotid. Examine the carotid and be sure that it bifurcates into the external and internal branches. If it does not it should not be ligated, as the blood supply to the brain would be cut off and death result.

If it does not bifurcate into the external and internal branches, only the branches supplying the external portions of the head should be ligated, the carotid being untied. Having determined that the common carotid bifurcates as usual, continue the dissection upward, exposing each branch and tying it in two places and dividing it. The dissection is thus continued upward until the level of the twelfth cranial nerve is reached, and all the branches of the artery but the terminal two have been controlled. The external carotid is itself tied twice and divided between. The ligature placed loosely around the external carotid below the superior

thyroid branch should not be tied until all the branches are ligated. It should not be tied sooner, because the artery would collapse and render the dissection difficult. The ligature is placed in position early, ready for use in case of accidental hemorrhage in the course of the dissection higher up. The upper portion of the artery should be dissected as it passes under the transverse loop of the twelfth nerve and the conjoined stylohyoid and posterior belly of the digastric and on into the substance of the parotid gland. It should be followed to its bifurcation when possible. The dissection should be done with dissecting forceps or scissors and not with a sharp knife, as it might divide some of the lower branches of the pes anserinus and cause facial paralysis, or else, by cutting through some of the smaller ducts of the parotid gland, cause a salivary fistula (Dawbarn). Use gentle downward traction during the blunt dissection, and when as high as possible, seize the artery with artery forceps and tie as high above it as possible, and then sever the artery below the forceps.

Close the wound by sutures, leaving a cigarette drain at its lower angle, or make a counteropening an inch and a half below the angle and insert the drain through this, entirely closing the original wound.

At the end of five or six days the drain may be discontinued and the counteropening allowed to heal by granulation.

Structures to be avoided: The internal jugular, internal carotid, pneumogastric, the superior laryngeal nerve, the pharyngeal branch of the pneumogastric, and the glossopharyngeal nerves. They all lie behind and deeper than the external carotid artery. Careful dissection should be done.

The opposite carotid should be operated in like manner after an interval of ten days, though both may be done at one time if the patient is in vigorous health. The death rate of this operation is considerable.

CHAPTER XXI

DISEASES OF THE FAUCES AND TONSILS

THE TONSILS AS PORTALS OF INFECTION

Since Strassmann reported 13 cases of tuberculous tonsils in 21 tuberculous cadavers, the tonsils have commanded considerable attention as channels of infection. The opinions of various observers since then have differed somewhat, especially in reference to the tuberculous process in the tonsils. There has been but little questioning of the fact, however, that the tonsils are portals of systemic and glandular infection. There is not, after all, a great divergence of opinion as to whether the tonsils are frequently a path of pathogenic infection; the difference is a question as to certain details, rather than as to the general theory itself. For example, some observers have failed to find tubercle bacilli, or the characteristic tuberculous changes in the tissue of the tonsils, which have been reported by other writers. Notwithstanding this, practically all writers agree that various pathogenic organisms do gain an entrance to the deeper tissue of the tonsils, the lymphatic glands, the lungs, the heart, and, indeed, to the whole system through these organs.

In view of the growing interest and more exact information on this subject, the tonsils have gained a prominence in medical literature which they did not have a quarter of a century ago. A brief *resume* of current thought on this subject will, therefore, be given in connection with a study of the diseases of these organs.

In reference to *primary tuberculosis* of the tonsils, there is a divergence of opinion; some hold that the tuberculous process in these glands is direct, while others contend that the infection reaches them from the lungs through the lymphatics and the bloodvessels, or by the flow of the bronchial secretions over them. Both views are probably correct in selected cases. It is probable, however, that tuberculous infection of the cervical lymphatic glands is usually due to the entrance of the bacilli and other microörganisms through the tonsils. This is borne out clinically by the fact that suppurating or tuberculous glands of the neck are rarely found in phthisical patients. Whereas, if they occurred secondarily to pulmonary infection, they would be frequently found in such patients.

That a latent tuberculous process may exist in the tonsils or in adenoids without presenting distinctive clinical signs thereof, is suggested by the reports of a few cases in which a fatal pulmonary tuberculosis followed the removal of tonsils and adenoids. Friedreich suggests that the removal

of the tonsils may have excited a recrudescence of a latent tuberculous tonsillitis in these cases. It seems to me that these cases point strongly to the conclusion that there is such a condition as latent tuberculosis of the tonsillar ring, which may continually infect the lymphatic glands of the neck, as well as the deeper structures in the thoracic cavity. Latent tuberculosis of the tonsils is not *per se* a serious menace to the health or life of the patient, but the danger arises from the extension of the infection to the contiguous organs.

The experiments of Dieulafoy show that of 96 guinea-pigs inoculated with pieces of tonsils and adenoids, 15 developed tuberculosis. While these experiments are not conclusive in their scope or character, they are, nevertheless, suggestive. We know that tubercle bacilli are found on healthy mucous membranes, and it is possible, though not probable, that in these experiments the infection may have come from the accidental presence of surface bacilli. If it is admitted that the germs giving rise to the infection in the guinea-pig were within the tonsillar epithelium, we practically admit the major proposition, namely, that the tonsils are, or may become, under favorable conditions, the portals of systemic or circumscribed infections in the contiguous glands and organs. In many instances this is also shown by the caseation or the suppuration which takes place in the tonsils.

The facility with which the invasion of pathogenic microörganisms is accomplished through the tonsils depends upon the following factors:

(*a*) The virulency of the invading microörganisms.

(*b*) The pathogenicity of the microörganisms.

(*c*) The general health of the patient.

(*d*) The existence or the absence of the strumous diathesis.

(*e*) The condition of the epithelium of the mucous membrane covering the tonsillar crypts, and the condition of the tonsillar tissue.

Piera has shown that bacteria are much more readily absorbed by the tonsils than is the coloring matter with which Goodale and Jonathan Wright experimented. Jonathan Wright, on the contrary, found the coloring matter to be more quickly absorbed than the bacteria (see p. 385).

According to Piera, the germs pass into the interior of the tonsil, while the coloring matter is absorbed in the clefts of the lacunar epithelium. He also found that the pathogenic germs were more readily absorbed than the non-pathogenic, and that healthy tonsils absorb better than the fibrous. He does not intend to convey the idea, however, that healthy tonsils are a menace to the system, for, on the contrary, they have a protective function. If the healthy tonsil readily absorbs the pathogenic germs, it also has the power of destroying them.

It has been thought that the tonsils are vestigial organs, which once had a function that is now more or less obsolete. Packard has called attention to the fact that tonsils have been traced in the lower animals from the reptiles up to man; and that they are more complex in man, and cannot, therefore, be said to be vestiges. On this subject, Watson Williams says that if the tonsils are in some measure a protection against

the invasion of microörganisms, their protective power is limited, and when this limit is passed, they are a positive source of danger. The crypts and fissures of the tonsils may become "traps" for microbes, and the peculiar anatomical arrangement of their investing epithelium, described by Stöhr, opens the gates to their invasion into the tissues of the tonsil, whence through the lymphatic channels and vessels they may gain an entrance into the system.

Williams also refers to the researches by von Babes, wherein he proves that in pulmonary gangrene the infection may enter through the tonsils as well as through the bronchi. He also says, primary tuberculosis of the tonsils is less rare than is generally believed, and the failure of the faucial tonsils to arrest the development of the bacilli results in tuberculosis of the cervical glands, so commonly observed in weakly children.

It has long been held that rheumatic fever has its origin in infection through the tonsils. Clinical observation certainly supports this view, as acute articular rheumatism is commonly observed following an attack of acute tonsillitis.

Dawson advances the ingenious theory that scarlet fever has its primary lesion in the tonsils. Whether or not this view will be supported by future observations remains to be seen. It has been shown by Kocher that acute suppurative osteomyelitis may be due to an infection by the same route.

Acute tonsillitis may be due to pneumococci, streptococci, and staphylococci, which are almost constantly present in the crypts of the tonsils.

Wright and Walsham have failed to find the tuberculous process in removed tonsils, but this does not necessarily prove that they are not pathways of infection. I have already shown that the tuberculous infection may exist in a latent form; that is, the bacilli may be present within the follicles without giving rise to distinct histological changes. By the term follicles is not meant the crypts or lacunæ, but the lymphoid nodules.

The *lines of defence* against microbic invasions through the upper respiratory tract may be classified as follows:

(*a*) The mucous secretions are regarded as having in some degree bactericidal properties, as they are rich in leukocytes.

(*b*) The epithelial covering of the mucous membrane of the upper respiratory tract is a mechanical barrier.

(*c*) The lymphatic tissue composing Waldeyer's ring (tonsillar ring).

(*d*) The cervical lymphatic glands.

(*e*) The bronchial lymphatic glands.

(*f*) The endothelial lining of the bloodvessels.

(*g*) The endothelial lining of the lymphvessels.

(*h*) The serum of the circulating blood.

(*i*) The leukocytes in the circulation.

It will be seen by the foregoing that the system is pretty well guarded against the invasion of pathogenic microörganisms. Should the first or the second barrier be overcome, the remaining ones are still ready to bar the further progress of the morbific bacteria.

In tuberculous infection of the cervical lymphatic glands the germs excite the reaction of inflammation, as shown by the enlargement of the glands. Under favorable conditions they are harmless on account of the phagocytic action in the leukocytes, which Stöhr has shown are thrown out from the clefts in the epithelial covering of the crypts.

Acute endocarditis, septic thrombophlebitis, and pyemic infarcts of the lungs have also been shown to be due to the absorption of microörganisms through the lymphatic ring.

Recapitulation.—(*a*) Tuberculous tonsils have been found in subjects who died of tuberculosis.

(*b*) Some observers have failed to find the tuberculous process in tonsils and adenoids removed from living patients, while others have been able to demonstrate it.

(*c*) Primary tuberculosis of the tonsils, while not common, cannot be said to be rare.

(*d*) Secondary tuberculosis of the tonsils has been demonstrated.

(*e*) Latent tuberculosis may exist in tonsils and adenoids without presenting distinctive clinical signs.

(*f*) The removal of tonsils and adenoids is sometimes followed by pulmonary tuberculosis. (This is doubtless a mere coincidence.)

(*g*) There are several barriers to the invasion of pathogenic microorganisms into the system.

(*h*) The invasion of the pathogenic microörganisms is promoted by the virulency of the germ, and by certain local and constitutional conditions.

(*i*) The tonsil is a barrier against the invasion of microörganisms, its power in this capacity being limited by the age of the patient and the condition of the tonsil.

(*j*) Rheumatic fever, acute endocarditis, septic thrombophlebitis, pulmonary gangrene, and other infective conditions have their initial lesions in the tonsils.

Practical Applications.—In view of the facility with which microorganisms, especially of the pathogenic type, gain entrance into the system through the tonsils, it becomes necessary under certain conditions to remove the tonsils in their entirety.

I have seen cases in which repeated attacks of acute follicular tonsillitis and concurrent cervical lymphadenitis had taken place. After tonsillectomy, *i. e.*, the complete removal of the tonsils, the tonsillitis necessarily ceased to recur, and there was no further recurrence of the lymphadenitis. It may be logically concluded that the diseased tonsils acted as a permanent incubator for the streptococci and the staphylococci, and the incubator being removed, the cervical lymphadenitis disappeared.

When a latent tuberculous process is present in the tonsils, the cervical glands are infected, and give rise to repeated enlargement and caseous degeneration of the glands. After the complete ablation of the tonsils, including the capsule, great improvement of the glandular disease should occur. While it may not always be advisable to perform tonsillectomy, it is usually advantageous to do so in those cases in which the cervical glands are enlarged.

It is also advisable to perform complete ablation when there is a tuberculous process in the tonsils with an incipient or latent involvement of the lungs. I have removed tonsils in this condition with the most satisfactory results.

Singers and public speakers with a troublesome subacute laryngitis, and whose tonsils are small and fibrous, or enlarged, may be benefited by the complete removal of the tonsils, whereby a possible source of irritation to the larynx through the absorption of microörganisms and septic matter is removed.

CLINICAL ANATOMY OF THE TONSIL

The tonsil is situated in the sinus tonsillaris between the faucial pillars, and has its origin in an invagination of the hypoblast at this point. Later the depression thus formed is subdivided into several compartments which become the permanent crypts of the tonsil. Lymphoid tissue is deposited around the crypts, and thus the tonsillar mass is built up. The inner or exposed surface, including the cryptic depressions, is covered with mucous membrane, while the outer or hidden surface is covered by a fibrous capsule.

According to Landois and Stirling, the development of the faucial tonsil is most easily studied in the rabbit, where the single primary crypt generally remains without branches through life, and there the tonsil first appears in embryos $\frac{5}{8}$ inch long (occipitosacral measurement), or of about twelve days as a shallow epithelial fold whose apex points directly backward into the connective tissue concentrically condensed around the pharynx. At this stage there is no infiltration of the leukocytes in the connective tissue around the crypt, and it is not until the embryos are about twenty-one days old ($1\frac{3}{16}$ inches long) that the leukocytic infiltration becomes evident. The crypt has then become much deeper and broader, and by its ingrowth has produced a condensation of the connective tissues at right angles to the original peripharyngeal condensation, as well as a great increase in the number of capillary bloodvessels. From this stage the elongation of the crypt, the condensation of the connective tissue, the increase in the number of bloodvessels, and the amount of leukocytic infiltration go on *pari passu* until the adult condition is reached. As soon as the leukocytes appear in numbers in the submucous tissue, they proceed to escape through the epithelium, as Stöhr has described.

In the fetus of the pig, the condensation of the connective tissue, especially at the apex of the tonsillar crypts, and the consequent massing of leukocytes, mainly at these points, is particularly well shown.

In the human fetus the process is the same, though complicated by the early ramification of the original epithelial crypt and the appearance of fresh ones. The crypts become so deep that the cells from the surface layers of their epithelium cannot at once be thrown off into the mouth, and remain as a concentrically arranged mass of degenerated cornified

cells filling up the lumen of the crypt; this mass is ultimately forced out by the *vis a tergo* of the leukocytes emigrating through the epithelium. It will at once be seen how closely this resembles the formation of the concentric corpuscles of the thymus.

The prime factor in the formation of the tonsils is the epithelial ingrowth, which partly mechanically compresses the meshes of the connective tissue, and partly causes proliferation of the connective cells and vessels by the slight irritation it produces, thereby making it easier for the leukocytes to escape from the thin-walled capillaries and venocapillaries so formed, and, when they have escaped, causing them to be detained in the finely meshed connective tissue longer than in other situations. As the leukocytes are well supplied with nutriment, they divide by mitosis in large numbers, as Flemming and his pupils first showed, and at a late stage in development (with great variations in individuals), "germ centres" are formed, where a special arrangement of connective tissue and vessels favors the process of division.

The lingual and pharyngeal tonsils develop in the same way as the faucial. His shows that all the tonsils arise behind the membrana pharyngis, and, consequently, all these epithelial ingrowths pass into connective tissue already condensed around the primitive alimentary canal (G. L. Gulland.)

It will be observed that the tonsil is an encapsulated organ, and that it is characterized by from eight to twenty crypts or tubular depressions. Many practitioners have confused the tonsil with the follicular tissue immediately surrounding it. So long as they were able to remove follicular tissue through the wound in the sinus tonsillaris, they thought they were removing tonsillar tissue. In this they were mistaken, as the lymphoid tissue immediately surrounding the tonsil is not encapsulated, nor is it characterized by cryptic depressions, and is therefore not tonsil tissue.

The tonsil does not always completely fill the sinus tonsillaris, the unoccupied space above it being known as the supratonsillar fossa, into which several crypts usually open.

The outer aspect of the tonsil is loosely attached to the superior constrictor muscle of the pharynx, thus subjecting it to compression with every act of deglutition. The palatoglossus and palatopharyngeus muscles of the pillars also compress the tonsil. Grober cites authorities who claim that the compression of the muscles forces food and bacteria into the crypts, rather than out of them.

Crypts.—The crypts are usually tubular and almost invariably extend the entire depth of the tonsil to the capsule on its outer surface. Some, however, are compound, *i. e.*, they divide below the surface into two or more tubules. They are usually comparatively straight, though they may be tortuous in their course. I have examined many hundreds of tonsils which have been removed with their capsules intact, and have never found crypts that did not extend through the follicular tissue to the capsule. Those opening in the supratonsillar fossa usually extend downward and outward, whereas in the lower portion of the tonsil their direc-

tion is outward. The area occupied by the mouths of the supratonsillar crypts constitutes, according to Killian, the hilus of the tonsil. Clinically, the crypts seem to be the source of the greatest amount of local and constitutional disturbances, as they often become filled with food, tissue debris, and bacteria. This is especially true of those capped over by an overlying membrane, as the plica supratonsillaris, and the antero-inferior portion of the tonsil which is covered by the plica tonsillaris. The plica supratonsillaris does not, in all cases, enfold the hilus, or supratonsillar crypts, as the tonsil often fails to fill the supratonsillar space. In other instances, it closely hugs the upper end of the tonsil, thereby completely closing the mouths of these crypts. It is in these cases, particularly, that the contents of the crypts are retained.

Reasoning from a mechanical point of view, one would reach the conclusion that the retention of the infected secretions must necessarily give rise to infectious inflammatory processes. Clinically, we know that this is not always true. The crypts are often found filled with food, tissue debris, and pathogenic bacteria, without any appreciable inflammatory reaction. Nevertheless, as I shall exemplify later, the mechanical closure of the crypts of the plica supratonsillaris and the plica tonsillaris adds greatly to the tendency to inflammatory and other morbid local and general processes.

It may be stated, as a general law in physiological pathology, that mechanical obstruction to the drainage of any secreting cavity tends to result in local morbid processes and in toxic or infectious manifestations in remote parts of the body.

The Epithelium.—The free surface of the tonsil, including the crypts, is covered with stratified pavement epithelium, the deeper layers of which are columnar in type, while the superficial are pavement. Goodale has shown that certain coloring matter, when dusted in the crypts, is readily absorbed into the interior of the tonsil. He claims that the absorption probably takes place through the interspaces between the cells. From this the inference might be made that bacteria are absorbed with equal facility. This conclusion does not, however, coincide with either physiological or clinical data.

Jonathan Wright has shown that there is a vast difference in the absorptive power of the tonsil for dust and for bacteria. He introduced carmine powder and bacteria into the crypts of the tonsils and excised them in fifteen minutes. The microscope showed the carmine particles in great abundance beneath the epithelium and within the intercellular spaces, whereas no bacteria were found beneath the surface. He also observed that the carmine dust remaining on the outside of the tonsil was easily washed away, while the bacteria were more difficult to remove. The adherence of the bacteria to the live animal membrane and their failure to pass through it he ascribed to the viscosity of the bacteria, a biomechanical property of microörganisms. The mechanical affinity existing between the bacteria and a living mucous membrane he considered as one of their defensive and offensive properties of a biomechanical kind, as distinguished from their biochemical products, the toxin and

endotoxin. Dust or carmine powder does not possess this adhesive property, hence it is readily absorbed, whereas the bacteria are not.

We know, however, from abundant clinical experience, that there are conditions under which the bacteria are absorbed through the cryptic epithelium in sufficient numbers to excite marked local and constitutional disturbances. Apparently, the adhesive property of the bacteria has been overcome, or the toxin of the microörganisms within the crypts has converted the live epithelium into inert matter, through which it readily passes. Wright says from the experiments of Goodale and others with colored granules, and from his own observations of dust particles passing the epithelial layer in health, and bacteria passing it in disease, it is evidence that there must be something beyond mechanical obstruction which, under ordinary conditions of health, keeps the tissue beneath the epithelium free of bacterial life, which swarms in some of the crypts on the outer side of the epithelial cells. Hitherto the revelations of the antitoxic power of the blood sera have been insufficient to explain the problem. That explains the nullification of the toxic power of the pathogenic germ after it passes within the tissues, but it does not explain immunity from infection—to translate literally, the freedom from the carrying in of the germ. It seems probable from experimentation with various forms of protoplasm that the animal organism evolves defensive properties to destroy by lysis, when the system through lack of excretory power becomes embarrassed by their presence.

Wright also says that bacterial protoplasms may excite bacteriolytic ferments in the epithelial cells, a property heretofore referred by Metchnikoff to the leukocytes only. In these ways he attempts to show the existence of equilibrium between immunity and infection. A lack of balance or equilibrium is effected by a loss of local tonicity or health, and infection then takes place.

In the epithelial lining of the crypts we find, therefore, the following properties:

(*a*) A biomechanical resistance to the invasion of the microörganisms, viscosity.

(*b*) A biochemical destruction or taming of the microörganisms in the crypts through the agency of a ferment thrown out by the epithelium under the stimulus of the retained bacteria. This process is known as bacteriolysis.

As long as the epithelium of the crypts is in a state of tonicity or health, an equilibrium between immunity and infection is maintained. When the cellular tonicity is impaired, the equilibrium between immunity and infection is lost and infection occurs. When the crypts are closed by the plica supratonsillaris and the plica tonsillaris, or by concretions in the mouths of the crypts, a very active warfare between the retained microörganisms and the epithelial cells is begun. The cells throw out a poisonous ferment, whereas the bacteria throw off a toxin for the purpose of impairing the tonicity of the epithelium. If the siege is continued sufficiently long, the cells give way, and the infectious host penetrates the epithelial barrier and enters the deeper tissues of the tonsil.

Sinus Tonsillaris.—The anterior pillar contains the palatoglossus muscle and forms the anterior boundary, whereas the posterior pillar contains the palatopharyngeus muscle and forms the posterior boundary of the sinus. The pillars meet above to unite with the soft palate. Inferiorly they diverge and enter into the tissues at the base of the tongue and the lateral wall of the pharynx respectively. The outer wall of the sinus tonsillaris is composed of the superior constrictor muscle of the pharynx. The sinus tonsillaris is, therefore, a triangular depression on the lateral wall of the fauces which partially envelops the tonsil.

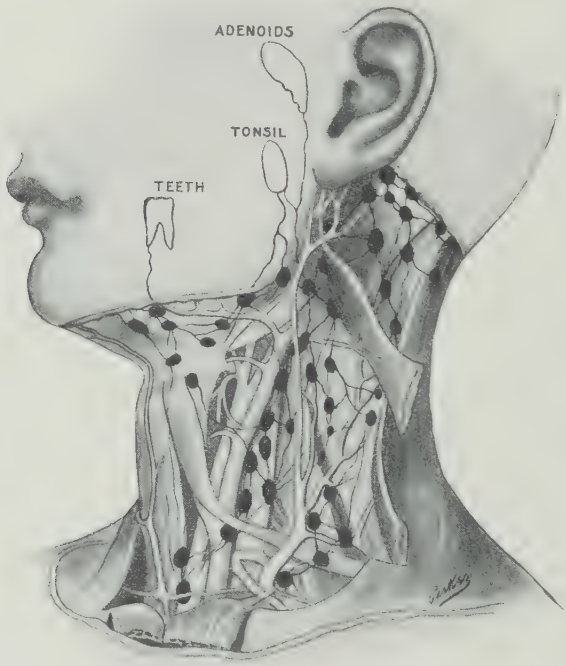
My clinical observations show that the tonsil is loosely attached in the sinus; that is, the so-called adhesions are not present. The extent of the attachment varies in different subjects. Patterson has shown that the supratonsillar fossa may extend downward so as to admit a bent probe between the outer side of the tonsil and the superior constrictor muscle of the pharynx, as far as the inner surface of the lower jaw. Even when the attachment is general, it is not usually so firm as to greatly interfere with the enucleation of the tonsil. The "adhesion" to the anterior pillar so often spoken of is, in my opinion, a myth. It is true that the tonsil has an anatomical connection with the anterior pillar, but the union is not of that firm, fibrous nature usually implied by the term. Indeed, the term "adhesion" is often used in reference to the plica tonsillaris which covers the antero-inferior portion of the tonsil, and which is often attached to the tonsil at its inferior extremity. One writer even speaks of the plica triangularis as an hypertrophy of the anterior pillar, whereas, in fact, it is an embryological structure, which in some of the lower animals develops into the tonsil itself.

The anterior limit of the sinus tonsillaris often extends well under the anterior pillar, thus concealing a large portion of the tonsil. The outline of the tonsil may be readily determined by digital examination or by seizing it with the forceps and drawing it toward the median line of the throat. When thus drawn the anterior shoulder of the tonsil may be seen outlined beneath the anterior pillar, and if still more forcibly drawn inward, the body of the tonsil slips from underneath the pillar, thus showing that it is not markedly adherent, but that, on the contrary, it is loosely attached and by proper procedures may be readily enucleated.

Lymphatics.—The relation of the tonsil to the lymphatic vessels is somewhat different from that which exists between the lymphatic glands and vessels. The difference in the relationship consists in the fact that the lymphatic vessels have their origin in the tonsil, whereas they pass through the lymphatic glands. George B. Wood says the tonsils do not have afferent lymphatic vessels. The question of chief clinical importance is the course and termination of the tonsillar lymphatic vessels which drain into the deep cervical chain underneath the sternocleidomastoid muscle, from thence to the thoracic glands, and finally into the thoracic duct. By this route infection is carried to all parts of the body. The tonsil, under certain conditions, being peculiarly susceptible to infection, becomes, therefore, the atrium of infection for a great variety of diseases extraneous to itself. Literature is rich with clinical reports of diseases illustrating this fact (Fig. 262).

In reference to the tonsil as the portal of infection in tuberculous processes, it is generally admitted that this often takes place through the tonsil, and extends thence through the lymphatics of the deep cervical chain on into the thorax. It then passes through the hilus of the lung into the visceral pulmonary lymphatics. The apex of the right lung is the most frequent seat for the inception of the pulmonary tuberculous disease. This has, heretofore, been attributed to the fact that this area is less directly in line with the respiratory air current, and that this portion of the lung has less motion than other portions of either lung. It forms, therefore, a peculiarly favorable location for the development of the tubercle bacillus.

FIG. 262



The lymphatic glands and vessels of the neck which drain the teeth, tonsils, adenoids, pharynx, and mastoid region.

Dr. J. Grober has questioned the existence of this route of pulmonary infection, or at least he has advanced a rival hypothetical explanation, based upon a series of experiments upon lower animals. He reports the following three suggestive experiments out of a total of twenty-eight:

First experiment, September 16, 1902. A young rabbit was anesthetized by ether and 1 c.c. of a sterilized emulsion of black Chinese paint injected into the left tonsil.

September 23, 1902, the autopsy showed black particles in the blood.

Behind the left tonsil there was a mass composed of the coloring matter and leukocytes. The lymph glands on the left side of the neck, as far as the upper border of the thyroid cartilage, were stained black. The microscope demonstrated that the lymph vessels were filled with free coloring matter, as well as leukocytes which inclosed small particles of pigment.

The glands and lymph vessels were fairly packed with the coloring matter. Beyond the zone of the lymph glands and vessels little coloring matter was found.

Second experiment. A small dog was narcotized by morphine injections. Six and one-half c.c. of the sterilized emulsion of black pigment was injected into the tonsil.

The autopsy, after complete exsanguination, showed the following conditions: Very little coloring matter in the leukocytes, none being free in the blood. The tonsil and the loose connective tissue containing the afferent lymphatic vessels of the tonsil were of a deep black color.

Along the muscles of the neck, as far as the hyoid bone and to the median line, there were streaks of pigment. The pigmented area also spread downward below the hyoid bone, where it extended 1 cm. beyond the median line. The coloring matter was traced to the bony opening of the thorax and to the parietal pleura, which, when stripped off and examined by transmitted light, showed the black pigmentation. The lymph vessels of the paratracheal connective tissue and of the esophagus, as far as 2 or 3 cm. above the bifurcation of the trachea, were also colored, whereas on the left or uninjected side no such phenomenon was found. All the lymph glands on the lateral wall of the pharynx, hyoid bone, larynx, along the deep vessels of the neck and supraclavicular fossa on the right side were black. The parietal pleura at the apex showed an exudate, but no adhesion to the visceral pleura.

The microscope showed that in all the above-mentioned organs there were no other changes. In the glands the coloring matter occupied the paravascular spaces. In the lymph vessels between the supraclavicular glands and the parietal pleura of the apex there was a large number of leukocytes which were filled with coloring matter. Free coloring matter was also present in this region. In the apex of the lung there were no signs of an inflammatory reaction. The coloring matter here seemed to be freely deposited within the connective tissue. In the above-mentioned second experiment there was coloring matter in the leukocytes.

Third experiment, April 4, 1903. A small dog was placed under morphine narcosis and 5 c.c. of coloring matter injected into the tonsil. April 13, the same experiment was performed on the opposite side.

May 10, the autopsy, after exsanguination, showed a large amount of coloring matter free in the blood; the leukocytes, the tonsil and connective tissue, and the connective tissue of the neck on both sides along the larynx to the aperture of the thorax were colored symmetrically. The lymphatic glands along the large bloodvessels, as well as those in the supraclavicular region, were deeply stained. The coloring matter was also found within the lymphatic vessels and in the paravascular spaces.

A fibrous exudate was found in the apices of both lungs, thus forming a bridge of inflammatory material from the parietal to the visceral pleura. The coloring matter was also present in the exudate. The microscopic appearance of the apices presented a light grayish coloration. The glands in the mediastinum were stained on the left side, as were also the bronchial glands. In the left lung there were three other small fibrinous exudates in which the coloring matter was present.

From these experiments, Grober builds the hypothesis that tuberculous infection of the apex of the lung may take place *via* the deep lymphatic chain, the supraclavicular glands, and thence to the parietal lymphatic vessels where an inflammatory exudate is thrown across to the visceral pleura. The tubercle bacilli travel across this inflammatory bridge and enter the apex of the lung.

While these experiments are not conclusive, they are interesting and open a field for further observations.

Blood Supply.—The tonsillar artery, a branch of the facial, is the chief vessel to the tonsil, though the ascending palatine, another branch of the facial, sometimes takes its place. The tonsillar artery passes upward on the outer side of the superior constrictor muscle, through which it passes and gives off branches to the tonsil and soft palate. The ascending palatine, another branch of the facial, also sends branches through the superior constrictor muscle to the tonsil. The ascending pharyngeal also passes upward outside of the superior constrictor, and when the ascending palatine artery is small, it gives off a tonsillar branch which is correspondingly larger. The dorsalis linguæ, a branch of the lingual artery, ascends to the base of the tongue and sends branches to the tonsil and pillars of the fauces. The descending or posterior palatine artery, a branch of the internal maxillary, supplies the tonsil and soft palate from above, forming anastomoses with the ascending palatine. The small meningeal artery sends more branches to the tonsils, though they are of minor importance.

Clinical Application.—Without reviewing the literature, which is rich in reports of cases showing the tonsil to be the portal of infection for many diseases in remote parts of the body, I have attempted to show under what conditions it becomes the portal or atrium of infection. Under conditions of local equilibrium or health of the epithelial lining of the tonsillar crypts, infection does not take place, whereas when the local equilibrium is lost, infection occurs. That the local equilibrium of the cryptic epithelium is frequently lost, is apparent to every clinician. In addition to the diseases arising through the tonsil as a portal of infection, there are those limited to, or having their focal centre in, the tonsil itself. Perhaps the strongest indictment against the tonsil is that it is often the atrium of infection in pulmonary tuberculosis. Whether the route of infection is *via* the deep lymphatics and the hilus of the lung, or the deep lymphatics and the parietal pleura at the apex, as shown by analogy in the experiments of Grober, is immaterial. The question of prime importance is, Do pulmonary or other types of tuberculosis have their origin through the tonsil as a portal of infection? In

view of my own observations, and of others, I must answer in the affirmative. Just what percentage has not been fully determined. Various writers report that from 4 to 10 per cent. of tonsils (removed) show local tuberculous lesions, such as tubercle bacilli and giant cells.

The structures of the tonsil which seem to favor infection are the crypts, especially those in the supratonsillar fossa and those covered by the plica tonsillaris. Wright has suggested that the imperfect drainage of the crypt leads to the ultimate loss of tonicity (equilibrium) in the epithelial cells which line them, thereby opening the way to systemic infection through the tonsil.

The question naturally presented at this juncture is, What is the rational method of procedure to protect the system from further infection? The choice of remedial measures seems to lie between internal medication, local applications, and surgical interference.

As to the first and second methods of treatment, it may be said that there are cases which may be satisfactorily treated by them; especially by relieving the distressing local inflammatory symptoms; indeed, many cases may be practically cured by such treatment. There are many others, however, in which such measures are wholly inadequate, either to relieve immediate symptoms or to ward off future attacks. In these cases we have usually resorted to some surgical procedure, such as opening the crypts and plunging the cautery point obliquely across them, decapitation (partial removal of the tonsil), and the complete removal of the tonsil.

The literature shows a wide divergence of opinion as to what constitutes the best method of surgical treatment, although it shows that nearly all writers agree that some sort of surgical procedure is indicated.

What does the anatomy indicate? It shows certain crypts so situated as to afford poor drainage of their contents, even though the superior constrictor, palatoglossus, and palatopharyngeus muscles compress the tonsil with each act of deglutition. This is especially true of those crypts which discharge into the supratonsillar fossa. Kauffmann has suggested that the supratonsillar crypts be opened with a sharp knife, and that the incised surface be painted with 5 to 20 per cent. trichloracetic acid. By thus opening the crypts their contents are drained. The applications of acid excite a violent inflammatory reaction which results in the contraction of the tissue of the tonsil. The process is often an extremely painful one, and may result in cellulitis and the formation of scar tissue. Furthermore, it does not always prevent further infection through the tonsil. It is, therefore, often necessary to repeat the incisions and applications of acid.

The patient is entitled to immunity from tonsillar infection if it can be established without seriously jeopardizing either his health or life. When the tonsil becomes a well-established atrium of infection, the physical economy of the patient is constantly menaced by conditions ranging all the way from a follicular tonsillitis to endocarditis and pulmonary tuberculosis. Measures should, therefore, be adopted which will insure future freedom from infection through the tonsil.

It has been shown by abundant clinical experience that cauterization of the lumen of the crypts or obliquely across them into the surrounding follicular tissue does not adequately meet the indications.

The same is true of "decapitation," or partial removal of the tonsil. Decapitation leaves the deep and more diseased portion of the crypts, and, while it may afford some relief of the symptoms, it is often followed by recurrent infections and by the reformation of the tonsillar tissue.

The complete removal of the tonsil with its capsule intact is, so far as I know, the only surgical procedure that guarantees immunity from infection through the sinus tonsillaris.

The function of the tonsil and the effect of its complete removal upon the general condition of the patient must be considered; so, also, must the question of hemorrhage. In reference to the effect of the removal of the tonsil upon the general system, it may be said that there is little evidence that it has any deleterious result. Masini, however, believes that the tonsil has an internal secretion comparable with that given off by the suprarenal gland. He arrived at this conclusion after experiments with the aqueous extract of the tonsil, intravenous injections of which gave the same results as those obtained from the injection of suprarenal extract.

The last word concerning the treatment of the tonsil cannot be spoken until its exact function is established. Clinically, there is little to show that its removal causes evil effects, whereas there is much evidence to show that good results, especially from its complete removal.

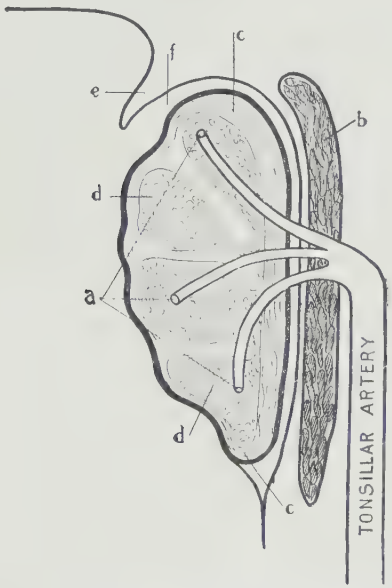
I have attempted its complete removal with the capsule intact in about 5000 cases during the past ten years, and, barring one or two instances in which there was a temporary paresis of the palatopharyngeus muscle, one case of cervical cellulitis, and a half-dozen moderately severe hemorrhages, I have seen no unfortunate result. The general health of many patients was greatly improved and recurrent septic inflammation within the sinus tonsillaris was eliminated. Recurrence of the tonsillar tissue has not taken place in a single instance. Should it grow again, this is almost *prima facie* evidence that the entire tonsil was not removed, nor have I seen a case in which the patient suffered from the loss of an "internal secretion," which some writers claim is produced by the tonsil, though they have never proved it.

When the tonsil has been completely removed, with its fibrous envelope, the source of infection is removed. It is, of course, possible for the follicular tissue which surrounds the tonsil to become diseased, but this should be differentiated from tonsillar disease. When the tonsil is not removed with its capsule intact, it is difficult to determine whether it has been entirely removed; and if a part of it is left, regeneration is likely to occur. The tonsil, if removed in its entirety, should show a distinctly defined mass of lymphoid tissue enveloped within a smooth, glistening, fibrous capsule on its outer, and with mucous membrane on its median, aspect. Lymphoid tissue which is not thus characterized is not tonsillar tissue.

Hemorrhage.—The danger from hemorrhage is, perhaps, the greatest

objection to the operation. Is this a real or an imaginary obstacle? It is both in adults. It is real in that severe hemorrhage does occasionally occur in operations on the tonsils. It is imaginary as to the reputed frequency of its occurrence and the degree of danger attending it. A knowledge of the possible sources of hemorrhage will enable the operator to largely exclude its occurrence. Furthermore, there are certain matters in the technique of local anesthesia, and in the after-treatment which, if properly applied, will greatly reduce the frequency and severity of hemorrhage. Clinically, I have observed that the most frequent site of arterial hemorrhage is at about the middle portion of the sinus tonsillaris, where the tonsillar branch of the facial pierces the superior constrictor muscle of the pharynx. Other points of hemorrhage are usually limited to the inferior portion of the sinus tonsillaris, where the tonsillar venous plexus is located, and to the anterior and posterior pillars.

FIG. 263



a, subdivisions of the tonsillar artery; *b*, superior constrictor muscle of the pharynx; *c*, *c*, fibrous capsule of the tonsil; *d*, lymph follicles or substance of the tonsil; *e*, plica supratonsillaris; *f*, supratonsillar fossa.

In another part of this chapter I have referred to the fact that three arteries, the tonsillar, the ascending palatine, and the ascending pharyngeal, pass upward on the outside of the superior constrictor muscle, which they pierce as they turn inward to ramify the tonsil and faucial pillars. Just before entering the tonsil they break up into several branches (Fig. 263). It is obvious that the smaller the branches cut during an operation, the less serious the hemorrhage. The clinical application of this fact is that if the arterial branches are severed as they enter the

capsule of the tonsil, the chance of hemorrhage is reduced to the minimum; whereas, if they are severed on the outer aspect of the superior constrictor muscle before they are broken up into smaller branches, the danger from both primary and secondary hemorrhage is greatly increased. It may be said that the operator should not injure the superior constrictor muscle in this operation, and this is true. Indeed, if he thoroughly appreciates the clinical significance of the anatomy of the tonsillar region, he probably will not injure it.

As to the anterior pillar, it should be borne in mind that there are arterial twigs coursing through it. The main trunks of the arterial branches are external to the palatoglossus muscle. Hence, it follows that in order to injure them, it is necessary either to pass the instrument behind the muscle or to include the musculature of the anterior pillar in the grasp of the tonsillotome, knife, blunt dissector, or scissors, and thus sever the muscle and vessels of the anterior pillar. The same statements may be made in reference to the posterior pillar.

The technique should, therefore, be such as to avoid injuring the muscles bounding the sinus tonsillaris, namely, the superior constrictor of the pharynx, the palatoglossus and the palatopharyngeus muscles, for by such technique only the small branches of the tonsillar arteries are injured.

CHAPTER XXII

INFLAMMATORY DISEASES OF THE TONSIL

General Considerations.—The inflammatory diseases of the tonsils are usually subdivided into various types, according to whether the process is acute or chronic, and is limited to the crypts or extends to the substance or parenchyma of the tonsil. As a matter of fact, this classification is somewhat artificial, as it is now well established that all, or nearly all, inflammations of the tonsil are due to infection through the epithelium of the crypts. The manifestations may be acute or chronic in type; it may appear as an acute or chronic lacunar inflammation, with the typical exudate at the mouths of the lacunæ or crypts; or it may be manifested in the form of a parenchymatous inflammation, in which the whole substance of the tonsil is involved. Inflammations of the tonsils are not surrounded by any profound mysteries other than those of a biochemical nature, which are common to all inflammatory processes. The fact of chief importance is that in all types of tonsillar inflammation there is a lesion of the epithelium which lines the crypts, and that some form of pathogenic bacteria has penetrated it. The determination of the type and virulence of the microörganisms is of even greater importance than the determination of the type of tonsillar inflammation under the older classification. The bacteriological findings at least afford some useful information as to the virulence of the infecting microörganism, and, therefore, influence the mode of treatment to a certain extent. If the virulence is marked, immediate surgical procedure is contraindicated; indeed, the presence of an acute inflammation would of itself constitute a contraindication to operative interference.

Much remains to be learned concerning inflammations of the tonsils. It may still be questioned whether it is good practice to remove tonsils in the wholesale manner now in vogue. The function of the tonsil in a child and in an adult is still an open question. When does its function cease or become so altered by disease as to justify its removal? Should the tonsil be partially or completely removed? When removed, what organ performs its functions? These and other questions are not fully answered. We know from clinical experience that when a tonsil shows a tendency to become the seat of recurrent inflammations the patient's health and life are conserved by its entire removal. Are there other methods of treatment that will better conserve the health and life of the patient? It is doubtful, though this is still an open question. The removal of the debris from the crypts, from time to time, would no doubt avert many acute exacerbations; the topical application of solutions of silver might also prevent acute manifestations, but in the long run such

methods of procedure must fail. The complete removal of the tonsil during a quiescent period must always succeed in preventing inflammation of the tonsil for all time to come. Will a tonsil thus removed recur? Never, if it is completely removed. Can it be removed by dissection with its capsule intact? Yes; with the most happy results.

ACUTE LACUNAR TONSILLITIS

Synonyms.—Acute follicular tonsillitis; infective tonsillitis, cryptic tonsillitis.

Etiology.—The chief causes of this and other forms of tonsillitis are the local impairment of the epithelium of the crypts and the invasion of certain pathogenic bacteria, as has been shown in the Tonsils as Portals of Infection and the Clinical Anatomy of the Tonsil. There are other factors which enter the etiology, and they will be discussed in the following analysis:

Local Lesion of the Tonsil.—As shown by Goodale and Wright the crypts of the tonsil are the seat of absorption for dust and microorganisms, whereas the surface epithelium of the tonsil has but little part in this process. They have shown that dust, as carmine powder, is readily absorbed through the healthy epithelium of the crypts, whereas bacteria are not. Bacteria are only absorbed through dead or impaired cryptic epithelium. Hence, the prime requisite for tonsillar infection is an impairment of the cryptal epithelium. This condition may be brought about by the retention of exfoliated epithelium and other debris in the crypts of the tonsil. The retention is formed by the constriction of the mouths of crypts from previous inflammation, and by the plica supratonsillaris and the plica tonsillaris, which cover the mouths of some of the crypts in such a manner as to prevent the expulsion of their contents. The toxin thrown out by the imprisoned microorganisms causes the death of the cryptal epithelium, and thus opens the way for the invasion of the microörganisms into the tonsil and the general lymphatic and circulatory systems; hence, the constitutional symptoms in this disease.

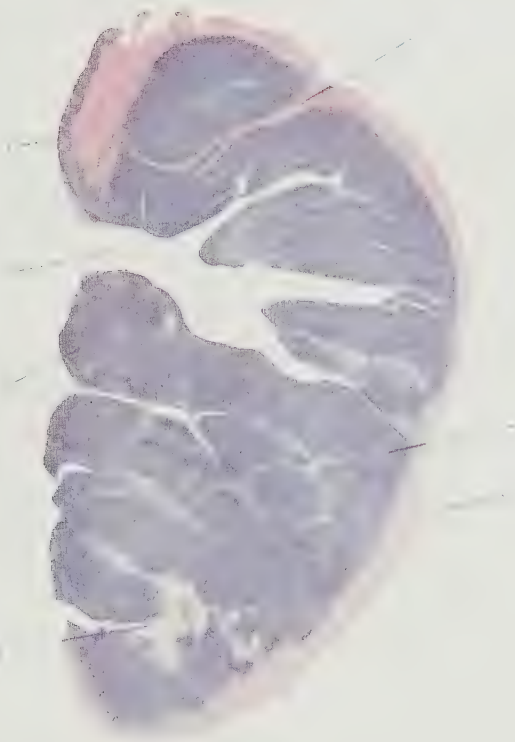
Bacteriology.—The bacteriology of acute tonsillitis embraces several pathogenic microörganisms, including the Streptococcus pyogenes, the Staphylococcus aureus and albus, the pneumococcus, and the leptothrix.

Age.—The disease is most common in young adults between the twentieth and thirtieth years of life. It is also common in children, and more rare after the fortieth year of life.

Catching Cold.—Tonsillitis is frequently the immediate result of catching cold, though this is but one way in which the resistance may be lowered, which favors the growth of the pathogenic bacteria.

Surgical Trauma.—The inflammations following surgical procedures in the nose and epipharynx frequently extend to the tonsil, and are of bacterial origin.

PLATE X



Section of a Tonsil Radically Removed on Account of Chronic Lacunar Disease. (Lumière's process photograph.)

1. Margo supratonsillaris.
2. Fibrous capsule of tonsil.
3. Trabeculæ or septa.
4. Degenerated and mechanically lacerated crypt.
5. Dilated tonsillar crypt.
6. Epithelial surface.
7. Lymphoid tissue.

PLATE XI

Acute Lacunar Tonsillitis

This disease may usually be cured by one application of a 90 per cent. solution of the nitrate of silver.

Specific Fevers.—Tonsillitis is often associated with specific fevers, such as scarlatina and diphtheria, and is of bacterial origin.

Pathology.—In acute lacunar tonsillitis the tonsil is swollen, though the chief changes occur in the crypts, where there is an accumulation of leukocytes and dead epithelial cells intermixed with pathogenic bacteria. The transudation of leukocytes occurs chiefly through the cryptic membrane rather than the surface mucosa. The accumulated material in the crypts or lacunæ is sometimes entangled in a fibrous exudate or pseudomembrane, though the pseudomembrane is not always present.

Symptoms.—The Subjective Symptoms.—In this, as in other acute infectious processes, the onset is sudden and is attended by malaise and fever. Chilly sensations or light rigors may mark the attack. The temperature gradually rises until the end of the first to the third day to 102° or 103°, and in young children it may rise as high as 104° to 105°. The febrile movement is accompanied by soreness upon swallowing, which as the disease progresses may become quite painful. The inflammation extends to the pharyngeal mucous membrane, and even, in exceptional cases, to the Eustachian tube and the middle ear. There may be pain in the ear through reflex sources without actual inflammation in the tympanum. Tinnitis may also be present. The gland under the angle of the jaw is usually swollen and tender, as it is in a state of great physiological activity in its attempt to check the invading host of bacteria which has passed through the impaired epithelial barrier in the crypts of the tonsil. The swollen condition of the tonsil and surrounding muscles renders rotary motions of the head somewhat painful. The same condition also renders articulation and phonation imperfect, the voice being thick and indistinct. The tongue is coated with a yellowish-brown fur, and the breath is fetid and offensive. Transient albuminuria is sometimes present, especially if the attack is severe and prolonged. Casts may also be found in the urine. Such a condition is common to all acute infectious processes in any part of the body, and do not necessarily point to grave results.

The acute symptoms rarely extend beyond the third, fourth, or the fifth day. The febrile movement and the swelling and soreness rapidly subside until the temperature is normal and the act of deglutition and the rotation of the head may be performed with comfort to the patient. The patient, though convalescent, is often left in a very weakened condition.

The Objective Symptoms.—At the onset the tonsils are swollen and red, while the crypts may not present the characteristic yellowish furred appearance, especially in the central and posterior aspects of the tonsil. The pharyngeal mucosa and the pillars of the fauces are usually redder than normal. At a later period, the tonsil and pharynx are still more swollen, and a creamy discharge is seen extruding from the mouths of one or more of the crypts. The patches are not usually true membranous products, as found in pseudomembranous and diphtheritic inflammations, but are the secretions and debris which completely fill the crypts. (See Plate XI.)

Occasionally a fibrinous exudate is admixed with the debris, which gives it some of the characteristics of an inflammatory membrane. The protruding secretion and debris are easily wiped away, in contradistinction to the diphtheritic membrane, which is closely adherent to the epithelium.

I have seen cases of diphtheria which closely resembled acute follicular tonsillitis, inasmuch as the membrane was loosely attached, on account of the solvent action of antitoxin administered eighteen to twenty-four hours previously.

Pharyngeal and lingual tonsils are usually simultaneously inflamed with the faucial tonsil, and the yellowish exudate or debris peculiar to the faucial tonsil is found in the shallow crypts of the pharyngeal tonsil and still more shallow depressions of the lingual tonsil. The debris is similar in composition to that found in the crypts of the faucial tonsils. If the febrile symptoms continue after the faucial tonsil appears to be well, the pharyngeal and lingual tonsils should be examined with a laryngeal mirror for evidences of inflammatory processes.

Complications and Sequelæ.—Complications and sequelæ are comparatively rare. The case usually ends favorably in seven or eight days, though it may cause acute articular rheumatism, endocarditis (I know of two such cases), and other affections remote from the tonsils. Under appropriate treatment the duration of the disease is often much shorter than this; one application of a strong aqueous solution of silver nitrate often terminates the disease within a few hours. Occasionally, when only one tonsil is diseased, the other is affected at the close of the first attack. When this is the case the febrile and other symptoms are repeated. The follicular inflammation is rarely followed by a phlegmonous inflammation of the tonsil or of the peritonsillar tissue (quinsy). The cervical glands, beginning with the one under the angle of the jaw, may suppurate. Purulent otitis media, pericarditis, pleuritis, erythema nodosum, and erythema multiforme have been reported as sequelæ of acute tonsillitis. Transient albuminuria is a rather common complication.

Diagnosis.—The following table will aid in the differential diagnosis between acute lacunar tonsillitis and diphtheria, although there are cases in which the differential points are obscure, and dependence must be placed upon the bacteriological findings.

Acute lacunar tonsillitis.	Diphtheria
1. Onset marked by sharp rise of temperature.	1. Onset, rise more gradual.
2. Rapid, bounding pulse.	2. Slow, feeble pulse.
3. Depression not marked.	3. Depression marked.
4. Exudation limited to the tonsil, especially the mouths of the crypts.	4. Exudation extends beyond the tonsils and is not limited to the crypts.
5. Exudate not adherent.	5. Exudate closely adherent.
6. Exudate soft and friable.	6. Exudate firm and leathery.
7. Exudate not distinctly membranous.	7. Exudate membranous and may be removed in strips.
8. Swollen glands uncommon except in severe cases.	8. Swollen glands common even in mild cases.
9. Albuminuria occasionally present.	9. Albuminuria common.
10. Klebs-Loeffler bacillus absent.	10. Klebs-Loeffler bacillus present.

I have seen cases in which repeated examinations failed to show the Klebs-Loeffler bacilli, which were finally shown at subsequent examinations. Absolute dependence must not, therefore, be placed upon negative microscopic findings; if, however, the Klebs-Loeffler bacilli are found, the case should be pronounced diphtheria, even though the clinical phenomena do not corroborate the microscopic findings.

Treatment.—This type of tonsillitis is more amenable to treatment than any other. One application of a 50 to 90 per cent. solution of nitrate of silver, if applied locally during the first twenty-four hours of the disease, will in nearly every instance abort the attack. I have repeatedly used silver in this way, and upon the following day the disease is under complete control. A second application is rarely required. The febrile and other symptoms rapidly decline and convalescence is quickly established. This may appear to be an overstatement of the facts, but it is in accordance with my experience. I have tried other remedies, but none of them have equalled the nitrate of silver. This strength of silver may appear to be caustic in action and unsuited for the treatment of acute tonsillar inflammation. As a matter of fact, it unites with the mucin so readily that its caustic action is greatly diminished before it reaches the mucous membrane. It coagulates the secretions and blanches the mucous membrane, thereby checking the inflammatory infiltration of the tissues. It also entangles the pathogenic bacteria in the albuminate of silver and prevents further activity on their part. It appeals to me as an ideal remedy in the early stage of the disease, and is worthy of extended trial.

In applying silver to the tonsil the excess of fluid should be squeezed from the cotton-wound applicator to prevent it trickling to the larynx, where it will produce violent spasm of the intrinsic muscles. The silver salts are not well tolerated by the motor nerves and muscles of the larynx, and severe suffocative symptoms may be produced by inattention to the technique of its application. I have seen cases in which severe cyanosis resulted from this cause. A little attention on the part of the physician will obviate this distressing occurrence. Guaiacol, 25 to 50 per cent. in olive oil, is the next most effective remedy. It should be applied locally two or three times daily for two days. The effect is very beneficial, though not so immediate as that of the nitrate of silver. It produces a hot, peppery sensation for about thirty seconds, followed by a sense of relief.

The carbonate of guaiacol given internally in 5 grain doses every three hours exerts a very beneficial action upon the course of the disease.

The tincture of the chloride of iron in eight parts of glycerin given in teaspoonful doses every two hours is another good remedy.

The salicylate of sodium, the benzoate of sodium, and the chlorate of potash are also recommended, but the silver solution is so much superior to either of the other remedies mentioned that it should be used in nearly all cases to the exclusion of the other remedies.

A laxative followed by a saline cathartic should be given early in the course of the disease.

If there is a history of repeated attacks of acute lacunar tonsillitis, the tonsils should be removed by complete dissection during the interval between the attacks. This procedure alone offers a considerable hope of permanent relief from the attacks and their more serious complications and sequelæ.

VINCENT'S ANGINA[1]

Synonyms.—Ulcerative tonsillitis; pseudomembranous angina.

Etiology.—Since Vincent described a spirillum associated with a fusiform bacillus found in certain forms of ulcerative tonsillitis, the condition has been called Vincent's Angina. The disease is most frequently found in young persons, though it occurs often in those of middle and later life. A debilitated state of health, local irritative lesions in the mouth, such as decayed teeth, inflamed gums, and oral uncleanliness, favor the development of the disease, which is by no means an uncommon one.

Pathology.—The lesions commonly involve one tonsil, usually at its upper part, may spread to the soft palate, the other tonsil, the pharynx, or the gums. It may even spread to the larynx or trachea, as in a case reported by Bruce. Farrel reported a case in a man, aged fifty years, in whom the lesion involved the tonsils, pharynx, and larynx. The membrane covering the patches is a pseudomembrane, and is formed by the necrosis of the superficial layers of the mucous membrane, not by exudation. The patches are of a grayish-white color, surrounded by a red inflamed areola, but separated from each other by healthy tissue. On removal of the membrane, which is granular and cheesy in consistency, an ulcerative area is exposed, varying in extent and depth. The ulcerated areas bleed freely, and are soon covered by a new membrane. The ulceration at times is very destructive, destroying the whole or a portion of the tonsil, and invades healthy tissue. T. H. Halstead removed the tonsils from a young girl, aged twenty years, several weeks following the apparent cure of a slight attack of Vincent's angina. The wound became immediately infected, the pseudomembrane extending across the soft palate and uvula. The patient suffered very great dysphagia for three weeks, and some dyspnea for a few days. The uvula sloughed off, and there was deep ulceration of the soft palate. The characteristic fusiform bacillus and spirillum of Vincent were present throughout in large numbers. In one of my cases, a physician, aged thirty-eight years, the fusiform bacillus and spirillum were present, and the symptoms had been present for two months. The membrane covered the upper portion of the left tonsil and pillars of the fauces. It yielded in one month to the local applications of a 10 per cent. extract of silver solution. Iodine did not seem to affect the case favorably. Immediately after the cessation of the symptoms, the tonsil was enucleated by dissection. After the lapse of three months there was no recurrence.

[1] The author is indebted to Dr. T. H. Halstead for much of the data on Vincent's angina.

The microscopic examination of a fresh smear taken directly from the ulcer, or a section of the pseudomembrane, stained with Loeffler's methyl blue or fuchsin, show fusiform bacilli, twice as long as wide, pointed at the ends, and with this a spirillum forming a network around the bacilli. The spirillum is 10 to 20 microns in length. This being the only fusiform bacillus occurring in the mouth, is readily recognized when found associated with the spirillum. As yet these bacteria have not been grown on any known culture media.

Diagnosis.—The diagnosis is made positive by the discovery of the typical Vicent's bacteria, the fusiform bacillus and spirillum. Unquestionably many cases, occurring in both children and adults, suspected of being diphtheria, but in whom Loeffler's bacillus is not found, are cases of Vincent's angina. Such suspected cases, in whom the culture is negative for diphtheria, should be examined for Vincent's angina. The same may be said of doubtful cases of suspected syphilis, both in the secondary and tertiary stage. An examination of a smear would clear up the diagnosis.

Differential Diagnosis.—The diseases usually confounded with it are diphtheria and syphilis. Many cases of what are called "ulcerative sore throat," gangrenous tonsillitis, are in reality Vincent's angina.

Symptoms.—The usual symptoms are of a subacute, or mild tonsillitis, headache, general malaise, chilly sensations, temperature varying from normal to 102.5°. There may be no constitutional disturbances, the patient complaining only of more or less pain in swallowing, or he may have discovered the yellowish patch on examining his throat with a mirror, because of a slight feeling of discomfort. Again, the symptoms are most violent, great pain in swallowing or talking, breath fetid, more or less gastric disturbance, submaxillary and cervical glands enlarged and tender. The ulcer may be single, or the membrane may spread like diphtheria and as rapidly.

The disease is acute, subacute, and often becomes chronic, the ulcers persisting for weeks or months. One attack is likely to be followed, months or even a few years later, by a recurrence.

Prognosis.—While most attacks are more or less mild, the patient suffering only local discomfort, the disease tends to persist for several weeks, and recurrence may occur at any time. Complications are seldom troublesome, and a fatal issue is not to be expected unless the larynx or trachea becomes invaded in a child.

Treatment.—Local treatment is of most importance. The galvanocautery and chemical cauterizing agents have been used without much success. The tincture of iodine applied to the ulcerated area several times a day, the applications being limited to the ulcer, is probably the best local agent for destroying the bacteria and promoting healing.

A 10 per cent. solution of nitrate of silver and a 30 grain to 1 ounce solution of zinc sulphate are also recommended. Indeed, I have obtained better results with this remedy than with iodine. (See case cited under Pathology.) Mouth washes, such as Seiler's solution, or a solution of chlorate of potash, are advantageous. Most relief from the pain in

swallowing will be obtained from the use of orthoform, in powder or tablet form. General tonic treatment is indicated.

To prevent infecting others, drinking and eating utensils should be sterilized and kept separate. Sputum and mouth discharges should be burned. Following the attack, local disease of the mouth and teeth should be attended to, but operative work had best be postponed until making sure by the microscope of the absence of the specific bacteria.

CHRONIC LACUNAR TONSILLITIS

Definition.—Chronic lacunar tonsillitis is characterized by the presence of caseous material composed of layers of desquamated epithelial cells enclosing cholesterin crystals, fatty matter, leukocytes, microorganisms, and occasionally calcareous deposits. The masses vary in size from that of a grain of wheat to that of a small bean. The crypts most often involved are those which open into the supratonsillar fossa and those covered by the plica tonsillaris, for the reasons already given in the Clinical Anatomy of the Tonsil. The tonsil may or may not be hypertrophied, though it is generally in that condition.

Etiology.—One of the chief causes of the disease is the retention of the desquamated epithelium, bacteria, and debris in the crypts, which in turn is due in part to the anatomical barriers afforded by the plicæ supratonsillaris and tonsillaris. In addition to this, there is a diseased condition of the epithelium lining the crypts, due to previous acute inflammations. This disease usually occurs in adults.

Symptoms.—The subjective symptoms are not usually severe in character. The patient may complain of pain upon swallowing saliva, but not upon swallowing solid food (Ball). Neuralgic pains sometimes shoot toward the ear. Some patients have the sensation, lasting perhaps for only a minute or two, of a foreign body lodged in the tonsil.

The objective symptoms are more marked and characteristic than the subjective ones. The patient coughs up the caseous masses, which have a fetid odor, and he consults a physician, who upon examination notes the fetid breath and the yellowish masses in the crypts of the tonsil. Upon exerting pressure upon the tonsil with a flat instrument the caseous masses are forced from the crypts. If they are full to overflowing, the yellowish spots appear at the mouths of the crypts much as they do in the acute form of the disease.

The tonsils are usually enlarged, and are often greater than they appear to be upon superficial examination, as they are covered by the plica triangularis and plica supratonsillaris; indeed, some of the largest tonsils I have ever removed were thus concealed from view. The plica tonsillaris is not an "adhesion" or inflammatory product, as some authors state, but is an embryological structure, as stated in the section on the Clinical Anatomy of the Tonsil. When the anterior and median surfaces of the tonsil are completely covered by an unusually large plica tonsillaris, the mouths of the crypts cannot be seen without a throat mirror,

or putting the patient "on the gag" (Pynchon). By resorting to the latter of these expedients, the tonsil is rotated forward so that its median surface may be seen by direct inspection. A blunt tonsil hook introduced into the crypts or into the pocket formed by the union of the plica tonsillaris with the tonsil will remove the caseous plugs and develop the fetid odor to its full extent.

Occasionally the mouth of a crypt becomes closed by inflammatory adhesions (caseous encyst), and the yellowish color shows through the thin membranous covering over the mouth of the crypt.

A tonsil thus affected is subject to acute exacerbations, generally of a mild type, the mucous membrane becoming slightly reddened. There is also some soreness upon swallowing. The temperature is but little elevated and attracts no attention. The patient sometimes complains of slight huskiness of the voice, and has fits of coughing which result from the local irritation of the tonsil. During these attacks he often coughs up the caseous masses. The repeated removal of the plugs affords some relief, and their tendency to reform is diminished, though a cure by this procedure does not often occur.

Treatment.—If the symptoms annoy the patient, and recur at frequent intervals, or if the patient has had rheumatism, enlarged glands in the neck, or other evidences of infection in a remote part of the body, which may reasonably be assigned to absorption through the tonsils, they should be removed in their entirety.

Slitting the crypt walls, followed by the application of a 20 per cent. solution of trichloracetic acid or of strong solution of iodine, has been strongly advocated by Kauffmann and Hollinger. Personally, I do not recommend this mode of treatment, as it is, at the best, a makeshift and fails to meet the fundamental requirements of the condition. The tonsil crypts are diseased, chronically infected, and have a tendency to continue in a diseased state. The rational procedure is, therefore, to remove the tonsil completely, preferably with its capsule intact. (For a description of the operation, see Surgery of the Tonsils.)

CALCULUS OF THE TONSIL

Small quantities of calcareous or gritty particles are often found in the centre of the caseous plugs filling the crypts of the tonsil in chronic lacunar tonsillitis. They sometimes become quite large and fill the crypts, and are known as calculi of the tonsil. The etiology is not clear beyond the fact that they are usually found in tonsils affected by chronic inflammation.

Symptoms.—The symptoms are identical with those of chronic lacunar tonsillitis with caseous plugs in the crypts; that is, there are recurrent attacks of mild tonsillitis with redness, which is especially marked around the affected crypts.

Treatment.—The treatment consists in the removal of the calculus, or the removal of the tonsil, as in chronic lacunar tonsillitis. If the calculus

is not easily disengaged from the crypt, an incision of the wall of the crypt facilitates its removal. Pain may be obviated by injecting a 4 per cent. solution of cocaine into the substance of the tonsil in the region of the calculus.

PHLEGMONOUS TONSILLITIS AND PERITONSILLITIS (QUINSY)

Phlegmonous tonsillitis is an acute abscess within the substance of the tonsil, whereas peritonsillitis is an acute abscess in the peritonsillar tissue. The processes are the same, but the location of the purulent accumulation is different. Peritonsillar abscess or peritonsillitis (quinsy) is much more common than phlegmonous tonsillitis.

Etiology.—The causation is about the same as that given under acute lacunar tonsillitis. Peritonsillitis (quinsy) probably results from an infection of the crypts in the supratonsillar fossa, which are large, slit-like cavities with irregular outlines, and which are in intimate relationship with the posterior and outer aspect of the tonsil. The disease is common in young adults and rare in children.

Symptoms.—Phlegmonous tonsillitis is more rare and less severe than peritonsillitis. Otherwise the symptoms are much the same. The onset of the peritonsillitis is gradual, though there may have been a preceding acute lacunar tonsillitis, with its sudden onset and severe symptoms. The temperature rarely exceeds 99° or 100°, whereas in acute tonsillitis it often rises to 103°.

The pain progressively increases with the extension of the purulent accumulation until it is almost unbearable. The muscles of mastication are encroached upon by the abscess, hence the patient has the greatest difficulty in opening the mouth sufficiently wide to permit of an examination of the throat. Swallowing becomes difficult and painful. The disease is usually limited to one side. The saliva dribbles from the mouth and forms one of the characteristic symptoms. Lateral movement of the head produces pain on account of the infiltration of the tissues of the neck in the region of the tonsil.

Thick viscid secretion forms in the throat, and it is with the greatest difficulty that the patient succeeds in removing it. The tongue is heavily coated and the breath fetid. Breathing is interfered with on account of the swollen mucous and submucous tissue of the pharynx. The patient has an anxious expression of countenance. During sleep he often has suffocative attacks which awaken him. Laryngeal dyspnea from extension of the edema to the laryngeal tissue is fortunately rare.

Objective Symptoms.—At the onset there is slight redness and swelling upon one side. Both tonsils are rarely affected at the same time. If both are affected, the second usually begins as the first subsides. If both are affected at once, the suffocative symptoms are more severe and alarming. As the disease progresses the redness, tenderness, pain, and swelling increase in severity. If the abscess is in the tonsil, it is pushed toward the median line or even beyond it. If the abscess is in the peri-

tonsillar tissue, the swelling often appears to be in the region of the upper portion of the anterior pillar. As a matter of fact, the apparent swelling in this region is often the anterior border of the tonsil projected against the pillar by the pus behind the tonsil. Incisions in this region often fail to reach the pus cavity for this reason; that is, the incision is carried directly into the tonsil instead of into the pus cavity outside of the tonsil. If the depth of the incision is carried beyond the outer border of the tonsil, the pus will be more often found. It should be remembered that the anterior third of the tonsil projects forward beneath the anterior pillar; hence, in making an incision through the anterior pillar to evacuate the pus, it should be made far enough anteriorly to escape the anterior border of the tonsil, and should be directed in an outward and a backward direction, outside of the capsule of the tonsil. If these anatomical facts are borne in mind, the anterior incision will nearly always evacuate the pus. If a posterior incision is to be made, it should be directed outward through the posterior pillar, or in its immediate vicinity, as the pus pocket often extends posteriorly to the tonsil.

The soft palate and uvula, as well as the pharyngeal mucous membrane, are red and edematous. The region of the tonsil is of a deep, dusky red color. The crypts are often filled with a pulp-like debris, which was probably the original source of infection. The infection does not originate in the peritonsillar tissue, but in the supratonsillar crypts of the tonsil.

Digital examination of the tonsillar region shows more or less distinct fluctuation. The focal centre of fluctuation is sometimes located about one-quarter of an inch external to the free border of the anterior pillar; at the junction of the upper third with the middle third of the tonsil; or it may be posterior to the tonsil.

The duration of the disease embraces from five to fourteen days when allowed to run its course, though it may extend over a longer period. The termination is marked by the spontaneous or artificial discharge of fetid pus. When the discharge is spontaneous, it usually takes place through the anterior pillar, though it occasionally occurs through one of the crypts.

Complications and Sequelæ.—Complications and sequelæ are rare. Cases are on record, however, in which the following conditions were present:

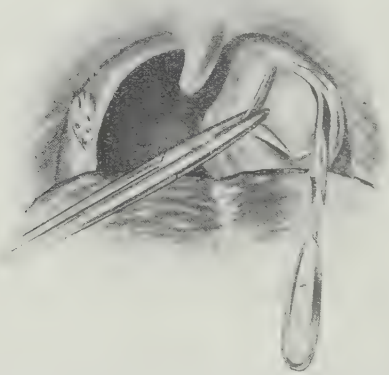
(*a*) Edema of the glottis from the downward extension of the process.
(*b*) Strangulation from the spontaneous rupture of the abscess.
(*c*) Ulceration thrombophlebitis of one of the large veins of the neck.
(*d*) Ulceration of one of the large arteries in the submaxillary region.
(*e*) Chronic peritonsillitis with an intermittent flow of pus (Ball).
(*f*) Encysted abscess in the tonsil.

Treatment.—The Onset.—If the case is seen early when infiltration and redness of the mucous membrane and the deeper tissues are present, but no pus, cold applied in the mouth or externally at the angle of the jaw diminishes the pain, and, indeed, may abort the attack. Cold may be applied internally by means of iced gargles or by sucking cracked

ice. It should be applied externally with a Leiter coil. It should be borne in mind that cold applications are indicated in the early stage of acute inflammation, whereas hot applications are indicated in the later stages. In very acute inflammation, proliferation and local leukocytosis are active; whereas, in the later stage, cell proliferation and local leukocytosis are lessened, though the proliferated cells remain permanently; hence, heat is indicated to increase the leukocytosis, as the lymphocytes are needed to clear up the inflammatory products and the polynuclear leukocytes to destroy the bacteria.

Pain may be relieved by the inhalation of hot vapors or steam, or by the application of hot poultices or a hot Leiter coil to the neck and angle of the jaw. Local applications of cocaine may also be used for the same purpose. The leukodescent 500 candle-power lamp, when available, provides an excellent mode of treatment. The rays of the lamp

FIG. 264



The author's dissection back of the capsule of the tonsil to evacuate a peritonsillar abscess. The dissection is started as though the tonsil were to be removed.

should be applied over the neck and angle of the jaw upon the affected side. The lamp should first be moved over the neck a few times at a distance of six inches, and then more slowly for ten to thirty minutes at a distance of eighteen inches. Such treatments will relieve the pain and reduce the swelling more readily and certainly than cold applications, as they promote the reaction of inflammation and convert the passive into an active congestion.

Surgical Treatment.—When the process is well established the evacuation of the pus is imperatively indicated. The point of the incision (in quinsy) should be determined by the location of pouching or fluctuation. It is usually in front of the anterior pillar on a level with the junction of the upper and middle thirds of the tonsil, though it may be in the posterior pillar or through the tonsil. Some recent writers have advocated the posterior pillar as the most favorable site for the

incision, whereas most of the earlier authors recommend the anterior pillar. As a matter of fact, many of the failures to evacuate the pus through the anterior incision are due to a failure to take into account the fact that the tonsil often extends forward beneath the anterior pillar. The incision as usually made, therefore, penetrates the tonsil instead of the tissue outside of it (Fig. 264).

The Author's Operation.—(*a*) Inject a 4 per cent. solution of cocaine through the anterior pillar into the peritonsillar tissue.

(*b*) Seize the anterior portion of the tonsil with forceps and pull it medianward and forward to reverse the position of the anterior pillar.

(*c*) Make an incision at the junction of the anterior pillar and the tonsil, thereby separating the pillar from the tonsil.

(*d*) Introduce a blunt dissector through the incision and separate the capsule of the tonsil from the superior constrictor muscle (bed of the sinus tonsillaris) until the abscess cavity is reached.

This method of operating can never fail to evacuate the pus. Other methods are inaccurate and are often attended with failure.

HYPERTROPHY OF THE TONSIL.

This subject is closely akin to chronic lacunar tonsillitis, as in that disease the tonsil is nearly always hypertrophied. Likewise the hypertrophic tonsil is nearly always subject to chronic lacunar inflammation. Nevertheless, it is practical to consider hypertrophy of the tonsil as a separate entity, as there are certain general considerations which justify it.

Hypertrophy of the tonsil usually begins about the second year of life and continues until young adulthood. Instances have been noted in which the babe seemed to have been born with enlarged tonsils. It is therefore occasionally congenital. While the hypertrophic process may continue into young adult life, it generally ceases to develop actively after puberty, and often seems to undergo an atrophic change. The connective-tissue element develops in excess of the other structures, and the tonsil becomes firmer and firmer, and shrinks on account of the contraction of the connective tissue. The difference between a child's tonsil and that of an adult is thus explained: In a child the enlargement is due to an increase in all the cellular structures composing the tonsil, whereas in an adult the connective-tissue cells are increased in excess of the other cellular elements (hyperplasia). In a child, the tonsil is soft and smooth in outline, whereas in an adult it is often much harder and is nodular in outline. In some children the hypertrophied tonsil is so loosely attached to the sinus tonsillaris that it can be easily removed in its entirety, with its capsule intact, with the tonsillotome. In others it is more firmly attached, and the tonsillotome only removes the superficial portion. In a few adults the tonsil is loosely attached, though it is ordinarily more firmly attached than in children. The exact size of the tonsil is not always shown by the ordinary examination, as only the superficial portion (median) is visible. The greater portion of the tonsil

may be hidden beneath the anterior pillar, the plica tonsillaris, and the plica supratonsillaris. Wilson has shown by the examination of a number of cadavers that the average height of the tonsil above the margo supratonsillaris is about ½ inch. Hence, too much importance should not be attached to the apparent size of the tonsil. It should be palpated with the index finger through the mouth, and its boundaries defined and its movability (degree of attachment) determined. In this way a good idea of the degree of enlargement and the ease with which it may be removed may be estimated.

The so-called submerged tonsil (Pynchon) is one that has undergone fibroid changes and is hidden behind the anterior pillar and the plica tonsillaris. Pynchon speaks of the plica tonsillaris as "an hypertrophy of the free border of the anterior pillar," whereas it is a normal structure appearing in embryonal life, and in some of the lower animals develops into the tonsil itself. There is no muscular tissue in the plica tonsillaris, and it should be removed with the tonsil. When it is left in place, it may form a pocket or pouch where food and other debris collect, and is the source of considerable local irritation.

The hypertrophic and hyperplastic tonsils may have healthy crypts, but, as a rule, the reverse is true. The lining epithelium of some of the crypts is usually of low vitality, often hornified, and is unable to resist the invasion of pathogenic microörganisms. During the transitional stage between hypertrophy and hyperplasia of the tonsil, hyperkeratosis of the cryptic epithelium may take place (hyperkeratosis of the tonsil). The leptothrix (mycosis tonsillaris) is an adventitious complication and not a disease *per se* (G. B. Wood). The hyperkeratosis is a self-limited condition, and usually disappears spontaneously in from one to three years.

If an hypertrophied or hyperplastic tonsil gives rise to untoward local symptoms, to constitutional disturbances, or to local morbid lesions in remote portions of the body, it should be removed in its entirety.

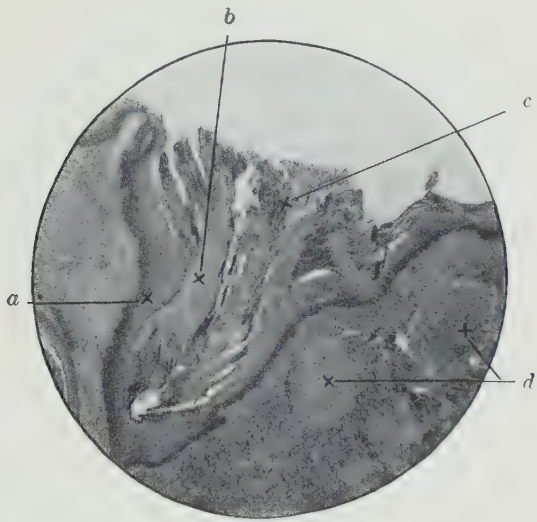
Treatment.—Palliative treatment directed toward the removal of the caseous plugs from the crypts, and from the pocket formed by the union of the plica tonsillaris with the tonsil, may be instituted when for any reason an operation cannot be performed. The incision of the crypt walls and the application of acids or iodine, as advocated by Kauffmann, Ball and others, may also be tried, but the best results are obtained by the complete removal of the tonsil with its capsule intact.

HYPERKERATOSIS OF THE TONSIL; MYCOSIS LEPTOTHRICIA

According to Dr. George B. Wood, hyperkeratosis of the tonsillar tissues of the throat is a disease, or, better, a condition, characterized by the appearance of numerous white projections not only from the cryptal orifices of the tonsils proper, but also from the orifices of the lymph follicles on the posterior and lateral pharyngeal walls and on the lateral glosso-epiglottidean folds. This condition does not occur on portions of

the throat where there is no lymphoid tissue. The lymphoid tissue of the upper respiratory tract, however, is so ubiquitous that occasionally we may see the little white projections on almost any part of the mucosa. In the large majority of cases the condition is limited to the faucial and lingual tonsils. That it reaches its greatest development on the base of the tongue and at a position just behind the lateral glosso-epiglottidean folds and the posterior part of the inferior poles of the tonsils is due almost entirely to mechanical reasons. The contractions of the muscles during swallowing prevent food from coming in intimate contact with the surface of these parts, and therefore permit the projections to grow undisturbed. Although the horny material is quite resistant to trauma, the bacterial accumulations which form the greater mass of the projection are easily brushed off, so that the size of the growth is much greater where it is protected from mechanical disturbances.

FIG. 265



Hyperkeratosis, showing the typical appearance under low power. The horny mass is growing from a comparatively small area of the cryptal epithelium, and the plug shows the ordinary fraying of its edges: *a*, cryptal epithelium; *b*, horny material; *c*, masses of bacteria; *d*, follicles. (Wood.)

The symptoms caused by this condition of the throat are either entirely wanting or very slight, and are due for the greater part to the local irritation caused by the hard, horny plug. If they project from the base of the tongue so as to come in contact with the epiglottis, there is an irritating tickling sensation which causes a hacking cough. If they are so placed as to be compressed during the act of swallowing, they may give rise to a slight pricking pain.

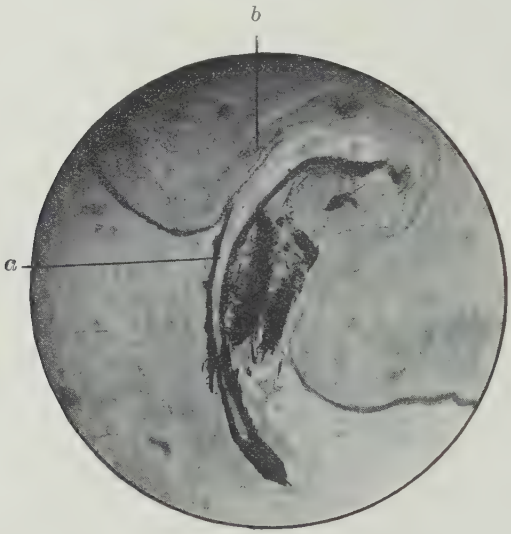
Occasionally among the rich and various bacterial flora which grow in such luxuriance on this horny material, there may lurk a germ possessed of more or less pathogenic power, which may set up an accom-

panying inflammatory reaction in the tonsil or surrounding structures. Hence, the relation which some observers have noticed between acute tonsillitis and this disease.

Dr. Wood also says that to understand correctly the pathology and the etiology of lacunar hyperkeratosis, we must turn our attention for a few moments to the anatomy of the normal active tonsil. The tonsil consists of four chief elements: the connective tissue, the germinating follicles, the interfollicular tissue, and the crypts.

1. The connective tissue, that is, the trabecula or reticulum, acts as a supporting framework to the tonsil substance proper. The trabeculæ carry bloodvessels, nerves, and lymphatics.

Fig. 266



Hyperkeratosis, faucial tonsils. This specimen is from a case which had been vigorously treated with antiseptics. There are practically no microörganisms. The black staining is due to nitrate of silver which has been used in treating the patient: *a*, keratoid plug; *b*, intact cryptal epithelium. (Wood.)

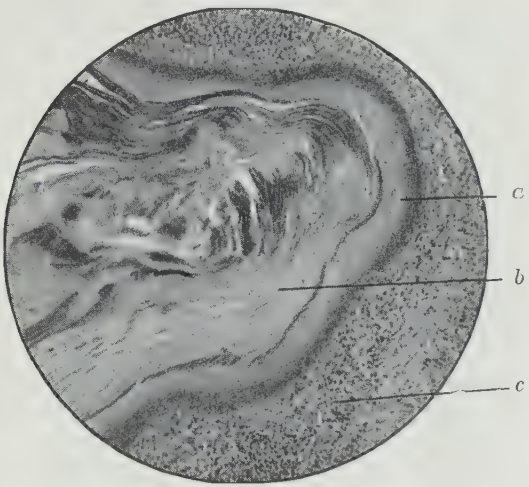
2. The germinating follicles (Fig. 265) are the centres wherein the larger mother cells of the leukocytic group undergo karyokinesis and form young lymphoid cells.

3. The interfollicular tissue is made up of lymphoid cells in various stages of development. The cells making up this interfollicular tissue differ in size and shape according to their location. They are greater in number around the follicles, and show greater difference in their anatomical construction in the immediate neighborhood of the crypts.

4. The crypt of the tonsil is its peculiar and most characteristic structure. It consists of an invagination of the epithelium from the surface of the tonsil, which has undergone a very interesting anatomical change. In the first place, the subepithelial connective tissue which is present in a

marked degree beneath the surface epithelium disappears as soon as the epithelium starts to form the crypts. This permits the epithelial cells to come in direct contact with the lymphatic structures of the tonsil, and very frequently it is impossible to distinguish a dividing line between the epithelium of the crypt and the interfollicular tissue. The epithelium of the crypt, unlike its progenitor, which covers the surface of the tonsil, does not form a compact unbroken barrier or protection. For the greater part of its extent it presents an intact line only one or two or possibly three cells in thickness. Toward the parenchyma the epithelial cells show a peculiar condition. They are separated from each other by interposed cells varying in type from slightly changed epithelial cells to well-formed lymphocytes. The epithelial cells may also extend from the crypt into the tonsillar substance, suggesting the ramifications of a malignant

Fig. 267



Hyperkeratosis. Cross-section of a crypt filled with keratoid material and bacteria: *a*, intact epithelium; *b*, hornified cells; *c*, lymphoid tissue. (Wood.)

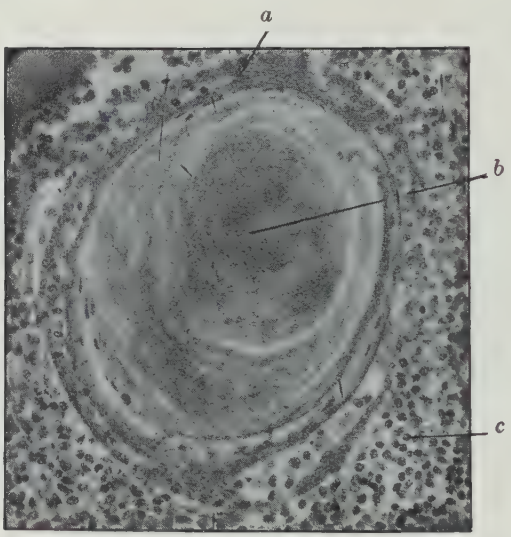
epithelioma. The smaller terminal invaginations of the cryptal epithelium are usually solid sprouts, frequently with central keratosed cores. The lumen of the crypt is formed by the subsequent exfoliation of the keratosed cells.

"Turning now to hyperkeratosis, we find the epithelium of the crypts showing characteristic changes. In hyperkeratosis the epithelium loses its rarefied condition and becomes ordinary pavement squamous epithelium similar to that covering the surfaces of the tonsil, except that generally it does not show the connective-tissue papillæ. The crypt of the tonsil is markedly dilated and filled with a horny mass (Fig. 267), which merges at various points into the epithelium, though in sections stained with eosin and thionin there seems to be a more or less distinct line where the epithelial cells become keratosed. The living cell has a nucleus which stains

with thionin, and its protoplasm is of a purplish color, due to the mixed staining with eosin and thionin. The keratosed material stains only with eosin, and is, therefore, of a bright pink color. Occasionally in the keratoid mass a very faintly stained nucleus is found, indicating that the material of which the mass consists has been originally derived from epithelial cells.

"According to the mechanical circumstances by which the tonsil is surrounded, the horny mass becomes sooner or later broken up into layers, between which multiply and grow organisms of all varieties. This fraying of the cryptal plug may take place within the crypt itself, so that the resulting fissures permit the bacteria at times to penetrate almost

FIG. 268



Hyperkeratosis. Cross-section of the terminal portion of a crypt showing the concentric arrangement of the layers of horny material and the epithelium, which is still somewhat disintegrated: *a*, epithelium; *b*, horny material in crypts; *c*, lymphoid tissue. (Wood.)

but not quite to the living epithelium. Mytotic figures may be seen in the epithelium at different places, but especially along the border toward the parenchyma of the tonsil. The epithelium is, therefore, in a state of active growth. This eccentric growth, however, which results in the formation of the keratoid plug, is not equally distributed to all parts of the epithelial lining of the tonsillar crypts. Take, for instance, a single individual crypt: a portion of the epithelium may still persist in its normal condition of partial disintegration without a discernible border line between it and the tonsil parenchyma; in another part the epithelium may exist simply as a barrier of cells with a very thin layer of subepithelial connective tissue, and again in the same crypt we may see the hyperkeratosis in its most beautiful and characteristic appearance.

"This change in the epithelium of the crypts is the characteristic pathological feature of hyperkeratosis. Besides this there are generally other changes in the tonsil. The connective tissue extends from the surface epithelium for some distance down along the crypt. The follicles are small and much less numerous, and the surrounding zone of lymphocytes has become comparatively insignificant. The mitotic figures in the follicles, though present, are less numerous, and the whole aspect of the organ is one of suppressed activity. We sometimes find, however, signs of local irritation in the immediate neighborhood of the crypts, as evidenced by the outwandering of polymorphonuclear leukocytes from the capillaries and their penetration between the cells of the cryptal epithelium. This irritation is easy to understand when we consider that the crypts contain a large number of saprophytes and probably also pathogenic microörganisms growing actively and receiving their nutriment from the accumulated keratosed cells.

"The toxins elaborated by these organisms must be absorbed to a greater or less extent by the tonsillar tissue. It is probably due to the fact that the cryptal epithelium has become an impact protective barrier that a more noticeable reaction is not a common result."

Hyperkeratosis is a condition peculiar to young adults, and is self-limited, from two to three years being required for it to run its course. Treatment is unnecessary, though if the horny masses cause irritation they may be removed by cauterization. The electrocautery should be used to destroy them, and the surrounding tissues should be penetrated until only healthy tissue remains. From three to four masses may be thus treated at each sitting at intervals of one week.

CHAPTER XXIII

SURGERY OF THE TONSILS

It is being more and more recognized that the complete enucleation of the tonsil within its capsule is the most satisfactory method of dealing with diseased tonsils. It is true that in a certain number of cases the distressing symptoms yield to less radical measures, such as the application of the cautery to the crypts, the incision of the crypts, the removal of the retained debris from the crypts, and the partial removal of the tonsil. I believe that if these cases were observed for a period of five or more years it would be found that the tonsil is still the seat of diseased processes not unlike those present before the operations above named. In addition to the diseased conditions it would also be found that in some instances the tonsillar tissue had grown again, oftentimes in greater bulk than before the operation.

If, on the other hand, the tonsil is removed in its entirety, with its investing fibrous capsule, the diseased processes in the tonsillar fossa and the tonsillar tissue will never recur. F. E. Hopkins, in a review of the literature since 1856, found several recorded cases of recurrence, chiefly before the year 1870, though instances of recent date were also cited. His conclusion coincides with that of Sir Morrell Mackenzie, Sir Felix Semon, and the author, that recurrence is nearly always due to incomplete removal of the tonsil. D. Braden Kyle expresses the opinion that some cases of apparent recurrence after excision of the tonsil are, in reality, the regrowth of an adenoma, the tonsil having taken on that type of benign neoplastic development. N. L. Wilson says that the complete removal of the tonsil may be followed by an inflammatory process in the tonsillar fossa, but that such processes will not often be found after a period of two years subsequent to the operation. Tuberculous and specific taints no doubt cause some of the recurrences after tonsillotomy.

It seems to me, therefore, after considering all the data obtainable, including my own experience, that many of the conditions heretofore regarded as necessitating only cauterization, incision, partial removal, etc., should be operated on by the complete method, whereby the entire tonsil with its investing fibrous capsule is removed.

Indications for Operation.—In the following paragraphs it should be remembered that the indications stated have special reference to the complete operation technically known as tonsillectomy:

According to George B. Wood, it is impossible except in certain acute, well-marked pathologic lesions of the tonsil to determine clinically the exact condition of the tonsil parenchyma. Tonsils which appear

to be diseased were found microscopically to consist of normal tissue, while on the other hand innocent appearing tonsils presented various suppurative or tuberculous lesions in the deeper structures.

Much has been written, and but little determined, concerning the internal secretion of the tonsil. Of the more recent writers, Dr. J. G. Wilson says: "The tonsil does not develop like a lymphatic gland from a plexus of preëxisting lymph vessels in the mesothelium. It develops as an ingrowth of endothelium from the second branchial pouch, and in its origin comes into line with the thymus and the thyroid, for I need not remind you that the thymus originates from the third branchial pouch, the thyroid from the fourth and the parathyroid from the third and fourth, all by imbudding of the endothelial lining of the primitive pharynx."

Dr. George B. Wood says there has been a great deal of guesswork as to the function of the tonsil, but that the only physiologic property they have been proved to possess is the production of lymphocytes in the germ centres or follicles, and that therefore the removal of the tonsils after the first two or three years of life removes only a very small fraction of the normal supply of lymphocytes. He has never seen a case in which the absence of the tonsillar tissue was harmful to the individual. He advocates the complete enucleation of the tonsil.

(*a*) Nasal catarrh and (*b*) diseases of the ear are sometimes true indications for tonsillectomy. Pychon says: "In a goodly number of those cases applying for treatment for nasal catarrh, or for ear disease, in which a plainly apparent hypertrophy of the faucial tonsils does not exist, it will be found upon close inspection that there is present a certain degree of faucial fulness which is markedly increased by causing the patient to gag. Among the embellishments of this every-day picture an abnormal faucial redness is observed, gradually increasing in depth of color from the normal pale pink of the lowest point of the pharynx disclosed by the use of the tongue depressor. There will also be observed a tendency for frothy saliva to adhere to the parts." The relationship between nasal catarrh and tonsillar disease is not perfectly clear, while that between the tonsil and the ear is more apparent, as the palatopharyngeus muscle extends to the pharyngeal orifice of the Eustachian tube, and inflammation of the tonsils and pillars might readily extend along the pharyngopalatine fold to the mucosa of the tube, and thence to the middle ear. Repeated attacks of angina in this region may result in degeneration of the palatopharyngeal muscle fibers and thus impair the muscular mechanism that controls the patency of the tube. Again, infectious material in inflammation of the tonsil may gain entrance to the tube and middle ear, either during coughing or vomiting, or in intense inflammation by the destruction of the ciliæ of the epithelium of the tube. Ordinarily, the ciliæ with their wave-like motion carry the secretions from the middle ear to the epipharynx. When they are destroyed, or their action is inhibited by violent inflammation, the entrance of foreign matter, as bacteria, etc., into the middle ear is comparatively easy. Hence, in certain diseases of the ear which have their origin in tonsillar inflammations, the removal of the tonsil is indicated.

(*c*) Recurrent attacks of tonsillitis, which are independent of aural or pharyngeal complications, usually justify the enucleation of the tonsils. The operation should not, of course, be done during one of the acute manifestations, as to do so might give rise to severe infection of the wound and deeper structures.

(*d*) By referring to Fig. 262 it will be seen that the tonsils drain into the deep glands of the neck. When these glands are enlarged and tender, the tonsils are usually the source of the infection; and if there is a history of repeated glandular involvement, the tonsils should be excised. According to George B. Wood there are afferent lymph channels to the tonsil.

(*e*) When the crypts of the tonsils are examined and they are found more or less filled with debris and bacteria, tonsillectomy should be considered. If the debris is removed with a tonsil hook or with a tonsil syringe, the inflammation is temporarily relieved, but in most instances it returns. If after repeated trials the inflammation recurs, tonsillectomy is indicated.

(*f*) Laryngitis with attacks of hoarseness is often due to tonsillar disease, hence the tonsils should always be examined; and if the crypts are diseased or the tonsils are hypertrophied, the tonsils should be removed.

(*g*) Hypertrophy of the tonsils is an evidence of a disease process, for in a perfectly normal throat they are of small size. There is a divergence of opinion upon this point; some writers hold that the tonsil is an organ of the body, while others believe it to be a pathological entity. Bacterial infection, when long continued, causes either hypertrophy or hyperplasia. When thus changed, its function as a lymphatic gland is impaired or lost, and the physical economy is best served by its complete ablation.

(*h*) Chronic follicular tonsillitis is an indication for tonsillectomy, as there is little likelihood of curing it by simpler methods. Even if the crypts are closed by the use of the cautery, the low vitality of the tissue predisposes to infection and inflammation.

(*i*) Follicular pharyngitis is, according to George Troup Maxwell, often caused by a chronic suppurative follicular tonsillitis. He claims that after the tonsils are removed, the follicular pharyngitis disappears.

(*j*) Tuberculous infection often begins in the tonsils, and when such a process is demonstrated or strongly suspected, the tonsils should be enucleated.

(*k*) Recurrent acute articular rheumatism following acute tonsillitis is an indication for tonsillectomy.

OPERATIONS ON THE TONSILS

There are so many methods of operating upon the tonsils for the cure or relief of the morbid conditions affecting them and the neighboring structures and organs, that it is impracticable to attempt to describe all of them. I shall, therefore, select those methods which appeal to me as the most rational from a clinical and surgical standpoint, and

which have, after long trial, given the best results. Some of the procedures to be described are not recommended as the best, but under some circumstances they must be resorted to as preliminary or tentative measures. Hemophilia, reluctance or refusal of the patient to submit to what seems to be the best method, will occasionally lead the surgeon to elect the incomplete method of operating. Hence, both complete and incomplete operative procedures will be described and their comparative merits stated as fairly as possible.

Complete Operations.—By the term "complete operations," I mean those surgical procedures whereby the faucial tonsil is removed in its entirety, either *en masse* or piecemeal. Clinical observations have clearly shown that any procedure short of this is often followed by little or no permanent improvement in the conditions for which it was done. Numerous cases are on record, and doubtless many more are unrecorded, in which there was a continuation of the pathological processes, and recurrence of the tonsillar tissue after incomplete operations.

As has been stated in a preceding paragraph, even after the complete removal of the tonsil, the sinus tonsillaris is sometimes the seat of an inflammation, but it rarely persists for more than two years. I can say from a personal experience covering about 5000 cases, in which the tonsils were removed in their entirety with the investing capsule intact, that such subsequent inflammations have been exceedingly rare, while recurrence of the tonsillar tissue has never taken place.

On the other hand, I can refer to a large number of cases in which I performed an incomplete operation, or what is known as "clipping the tonsils," with a Mathieu's tonsillotome or other instrument, in which the subsequent tonsillar inflammation occurred comparatively frequently.

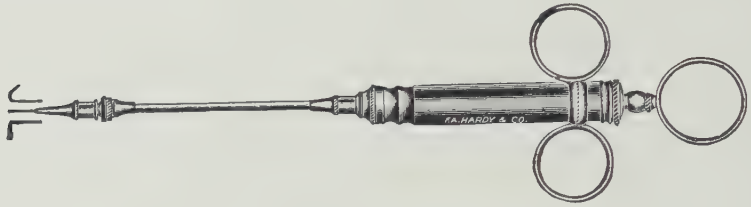
It seems, therefore, that the time has come when a text-book should clearly recommend the complete operations upon the tonsils as the ones that should be used if it is at all expedient to do so, and that the incomplete operations should be resorted to only when the peculiar conditions of the patient contra-indicate any of the complete methods, or when other circumstances prevent their adoption.

The Author's Complete Operation with Right-angle Knife and Ecraseur.—While every detail in the following technique is not original with me the operation as a whole has been my own creation, especially with reference to the removal of the entire tonsil with its capsule intact. In most cases the diseased tonsil is composed of three lobes, or masses, each with an investing capsule, the three lobes being held together by a fibrous envelope, or secondary enveloping capsule. For all practical purposes, the tonsil may be regarded as one mass with an investing capsule, and as such it may be removed in its entirety.

(*a*) Anesthesia may be either local or general. Personally, I prefer local anesthesia, except in those cases in which, for various reasons, the patient cannot be operated upon in the conscious state. This is a matter that must be decided by each surgeon, as the personal element enters so largely into its consideration.

Local anesthesia may be induced by swabbing the tonsils and the faucial arches at intervals of five minutes with an aqueous solution containing 10 per cent. of cocaine and 5 per cent. of carbolic acid. Both ingredients produce blanching and anesthesia. From five to ten applications are usually required to produce complete anesthesia. In some cases a single application of a 20 per cent. solution of cocaine should be applied. The frequent use of a 20 per cent. solution is quite likely to produce toxic results.

Fig. 269



Street's tonsil hypodermic syringe.

Thorough anesthesia may be produced by using the weakest preparation of Schleich's solution in much larger quantity. I now use this solution, injecting $\frac{1}{2}$ dram into the peritonsillar tissue, and waiting five minutes for its anesthetic effect. Adrenalin may be added to the solution to reduce the hemorrhage.

Fig. 270

The author's tonsil forceps.

The position of the patient is a matter of some importance. Under local anesthesia the upright position in the operating chair should be used. Under general anesthesia the patient is placed upon the operating table, with his head either over the end of the table in the Rose position, or upon his side (Fig. 236), according to the preference of the surgeon. A mouth gag (Fig. 237) should be used if a general anesthetic is given.

In the further description of the technique, I will assume that the patient is conscious and in the upright position.

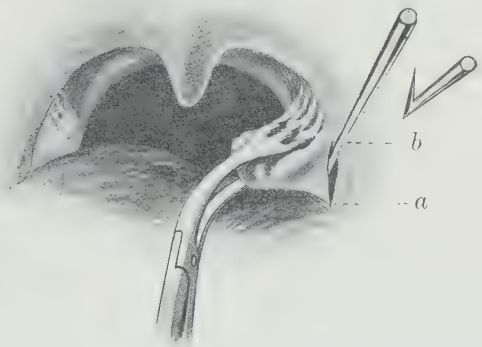
(*b*) Seize the tonsil with the vulsellum forceps (Fig. 270); the tip of one prong should be placed in the supratonsillar fossa and the other at the base of the tonsil. When they are thus placed, they should be pushed deep into the tissues, closed, and locked. In this way they engage the fibrous capsule or deep surface of the tonsil, and will not tear loose except in young children when traction is made.

When the blades are closed the bulk of the tonsil lies between the shanks of the instrument, as shown in Fig. 271. This has a distinct advantage over a superficial grasp of the tonsil, as it enables the surgeon

to dissect it with greater ease. It also enables the operator to bring the posterior pillar into easy reach of the tonsil knife.

(*c*) Dissect the anterior pillar from the tonsil and carry the incision above the margosupratonsillaris, or the supratonsillar space, to the posterior pillar (Fig. 271). The aim should be to dissect around the upper half of the tonsil, removing the mucous membrane forming the roof or dome of the supratonsillar fossa. These details are important if it is the intention to remove the tonsil with its fibrous capsule intact. The incision thus assumes the form of an inverted U. The instrument used is a right-angle knife. It should be hooked into the mucosa at the junction of the anterior pillar with the plica triangularis (Fig. 271). It is then pulled toward the median line of the throat, thus severing the pillar from the plica triangularis and the tonsil. Reintroduce the hook blade into the incision thus made and engage it as before, and pull toward the median line. Two or three such cuts are required to bring the incision above the supratonsillar fossa. While the foregoing incision is being made, the tonsil is in the grasp of the vulsellum forceps, and it is pulled forcibly toward the median line. This stretches the pillar and greatly facilitates its separation from the tonsil with the hook knife.

Fig. 271



The primary incision being made with the right-angle crypt knife. The knife is introduced through the mucous membrane at the junction of the anterior pillar, and the plica triangularis upon being pulled forward makes the incision *b;* the knife is again introduced through the incision as shown (*a*) in the illustration. The incision is thus completed by three or four cuts with the knife.

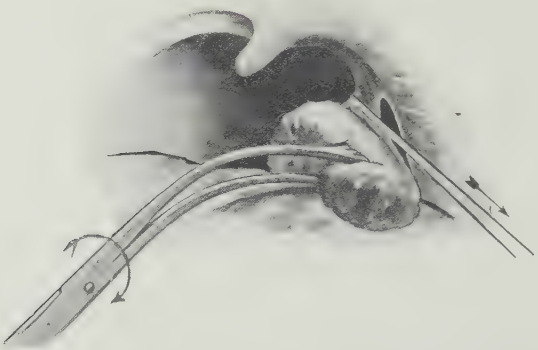
The posterior pillar should next be separated in much the same manner. This pillar is not as accessible as the anterior one, but it can be brought into view by rotating the handle of the vulsellum forceps, thereby turning the tonsil upon its lateral axis in such a way as to bring the posterior pillar forward and upward, where it is readily accessible to the hook knife (Fig. 272).

The two incisions should be united above the margosupratonsillaris. Observe carefully the margin of mucous membrane forming the roof of the supratonsillar space and make the incision just above it.

The combined incisions are thus converted into an inverted U-shaped incision.

(*d*) Again seize the tonsil with the vulsellum forceps, with the upper prong tip introduced into the supratonsillar portion of the incision, and

FIG. 272

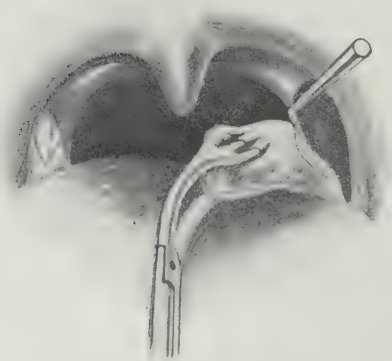


Showing the dissection of the posterior pillar from the tonsil with the right-angle knife. The tonsil is turned forward upon its lateral axis with the author's vulsellum forceps to bring the pillar upon the upper surface, where it is accessible to the knife.

the lower prong tip at the base of the tonsil. The tonsil is thus well within the grasp of the forceps and is ready for dissection with the hook knife.

(*e*) Pull the tonsil toward the median line, thereby putting the fibers attaching it to the superior constrictor muscle upon a tension. With the hook knife sever the fibrous bands (Fig. 273), following the external contour of the tonsil to its inferior portion. It may be necessary to dry the wound during the operation, even though cocaine-adrenalin solution has been injected. If anesthesia has been induced by brushing the tonsil with cocaine without adrenalin the hemorrhage may be considerable.

FIG. 273



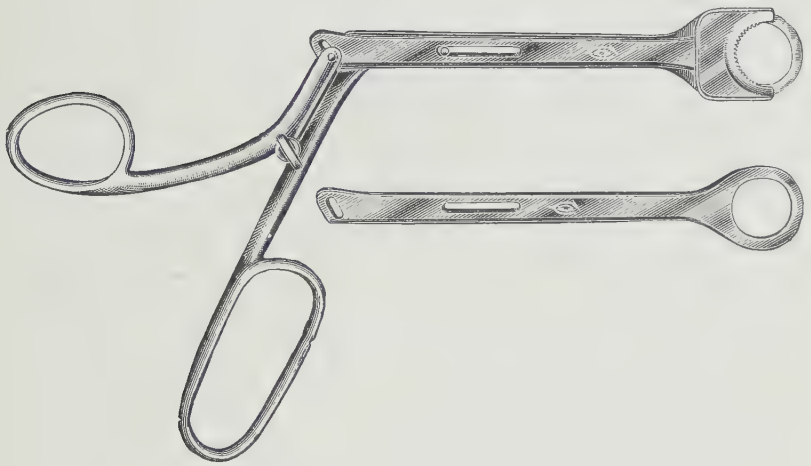
The tonsil in the process of dissection with Kyle's crypt knife. During the dissection the tonsil is forcibly drawn toward the median line of the fauces with the author's vulsellum tonsil forceps.

(*f*) At this stage of the operation the use of the knife may be abandoned and the author's ecraseur tonsillotome substituted (Fig. 274) to complete the operation. This shortens the time of operation, though it may be completed with the knife.

(*g*) Pass the forceps through the ring blade of the ecraseur and seize the tonsil, then pass the ecraseur over the tonsil as shown in Fig. 276.

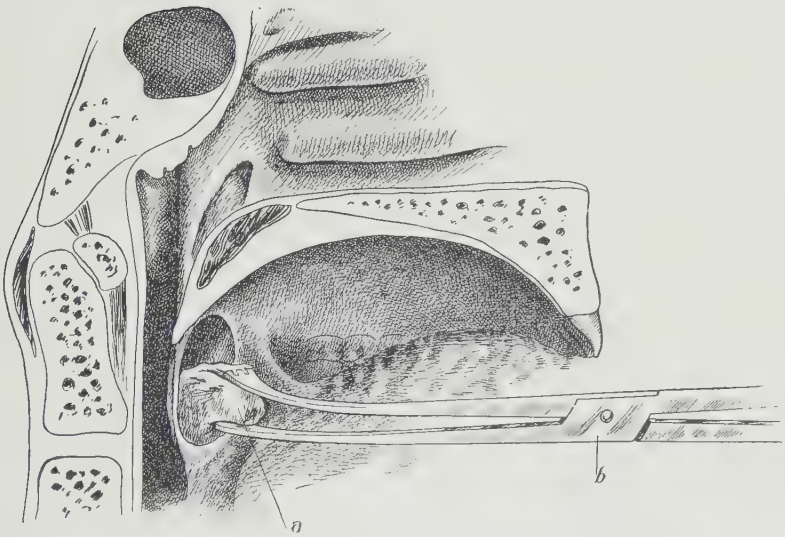
Close the instrument and thus complete the operation. The dull ring blade of the ecraseur readily passes behind the tough fibrous capsule of the tonsil and makes a clean dissection of its lower portion.

FIG. 274



The author's tonsil ecraseur, a substitute for the snare.

FIG. 275

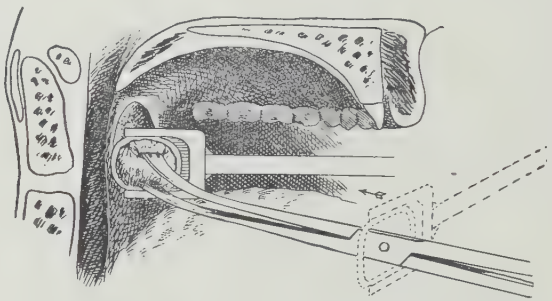


a, the tonsil in the grasp of the author's tonsil forceps; *b*, the upper half of the tonsil *a* has been enucleated by dissection with its capsule intact.

The wire snare, on the contrary, tends to cut through the capsule and leave the lower portion of the tonsil *in situ*.

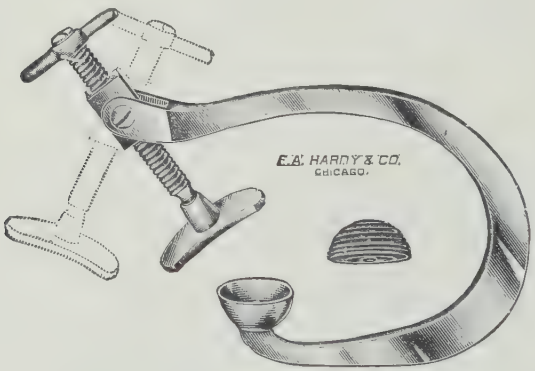
If hemorrhage follows the operation, it may be controlled by swabbing the sinus tonsillaris with a solution of permanganate of potash, $\frac{1}{8}$ to 1 grain to the ounce of water. Peroxide of hydrogen may also be

FIG. 276



The final step of the tonsillectomy as performed with the author's tonsil ecraseur, a substitute for the tonsil snare.

FIG. 277



Pynchon's tonsil hemostat.

FIG. 278

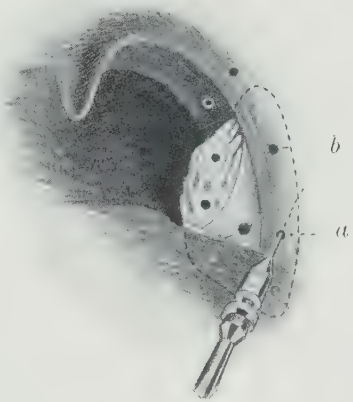
Boetcher's tonsil hemostat.

used for the same purpose. Stronger remedies are rarely required. Continuous gargling with iced water often controls it. Tonsil clamp forceps (Figs. 277 and 278) need rarely be used.

The advantage of the author's tonsil ecraseur over the tonsil snare is, that it is always ready for use, whereas the wire of the snare needs adjustment each time it is used. When two tonsils are to be removed, the wire for the snare must either be straightened or another one inserted before the second tonsil can be removed. This is not true of the ecraseur, as it is always ready for use, like an ordinary tonsillotome. The edge of the fenestrated blade is round, thus conforming to the cutting surface of a wire. (Sharp blades are also furnished with the instrument.) If, as claimed, little hemorrhage follows dull dissection, the ecraseur meets this requirement. The same is true of the cold-wire snare. After many dissections with the ecraseur, I have rarely known it to fail to complete the dissection of the tonsil with its capsule intact.

This method of removing the tonsil with its capsule intact, while not based upon as good surgical technique as the author's method with a scalpel, is easier for the average operator to perform than the dissection with the scalpel. I prefer dissection by means of the scalpel because I can do it in much less time, with less hemorrhage, and less discomfort to the patient. I also prefer this method, because I believe the wound after a clean dissection with a sharp knife heals more kindly and quickly than the wound after dull dissection.

Fig. 279



Schema showing the points of injection of adrenalin and cocaine solution preliminary to the removal of the tonsil with its capsule intact. About 2 minims of the solution is injected at each point. If a $\frac{1}{8}$ gr. Schleich's solution is used (infiltration anesthesia) $\frac{1}{2}$ to 2 drams may be injected at *a* and *b*.

Tonsillectomy with a Scalpel.—The Author's Operation.—After having tried almost every known method of removing tonsils in the adult, the simplest of all instruments was found to be the best adapted for the purpose. A small scalpel (Fig. 280) is the instrument used in all cases. The only other instrument required is the vulsellum forceps (Fig. 270). A tongue depressor is not used, as the forceps crosses the tongue and keeps it out of the way.

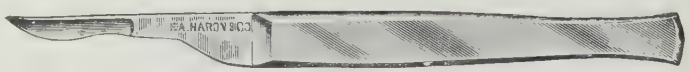
Technique.—(*a*) Induce anesthesia by the injection of the cocaine-adrenalin or the infiltration solution in the peritonsillar tissue. If the cocaine-adrenalin solution is used, only 8 or 10 minims should be injected. If Schleich's No. 3 solution is used, 1 to 2 drams should be injected, and a period of from one to five minutes allowed to intervene between the injections and the operation (Fig. 279).

(*b*) Seize the tonsil with vulsellum forceps, one blade in the supratonsillar fossa, the other at its base, as in the preceding method. Pull the tonsil medianward and forward to dislodge the anterior shoulder

from beneath the anterior pillar. This pulls the posterior margin of the pillar forward and facilitates the introduction of the scalpel between it and the tonsil.

(*c*) Introduce the blade of the scalpel to a depth of about one-half inch between the anterior pillar and the tonsil at the junction of the pillar and plica tonsillaris (Fig. 281). Sweep the blade upward to the margosupratonsillaris, and thence over the margosupratonsillaris to the posterior pillar (Fig. 282). The knife should be very sharp for this purpose. This completely severs the tonsil from the anterior pillar and

FIG. 280

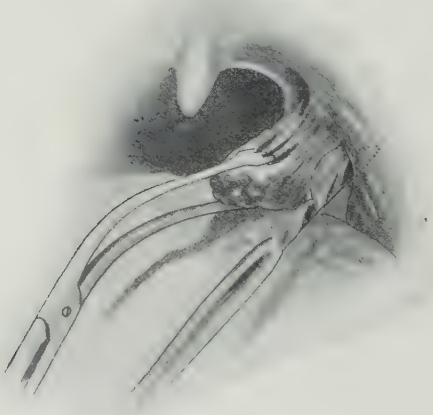


The author's tonsil knife.

exposes the outer aspect of it to further dissection. By including the margosupratonsillaris in the incision, the upper portion of the tonsil concealed in the supratonsillar fossa is freed from its attachments. If this step of the operation is not observed, the dissection is more difficult.

Casselberry called attention to the advantage of dividing the mucous membrane along the margosupratonsillaris. He claimed that this

FIG. 281

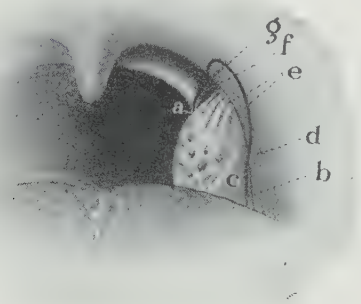


The first incision in the removal of the tonsil with its capsule intact. The tonsil is drawn forward and medianward from the sinus tonsillaris. The incision is extended upward over the margosupratonsillaris to the posterior pillar.

procedure rendered the liberation of the velar lobe, or supratonsillar portion of the tonsil, much easier and more certain. Without knowing of Casselberry's recommendation, I arrived at the same conclusion, though my technique is quite different from his.

By my method, the mucous membrane is divided at the junction of the plica tonsillaris and the anterior pillar, and the incision is then extended along the margosupratonsillaris to the posterior pillar, as

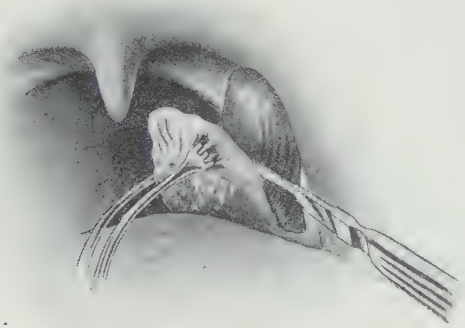
Fig. 282



Anatomical landmarks of the fauces: *a*, *b*, the incision liberating the pillars in the removal of the tonsil; *c*, plica tonsillaris; *d*, anterior pillar; *e*, supratonsillar slit-like crypts, or hilum of the tonsil; *f*, supratonsillar fossa; *g*, margosupratonsillaris.

shown in Fig. 282. If this preliminary incision is thus made, the subsequent steps of the operation will be more easily accomplished; indeed, the dissection of the tonsil is nearly consummated by this procedure alone.

Fig. 283

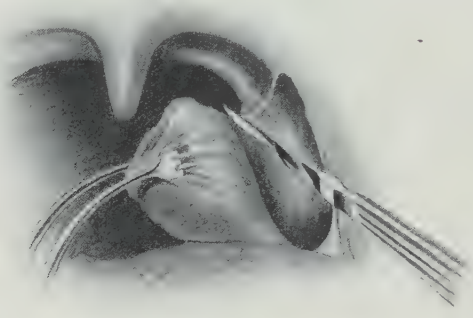


The tonsil being separated from the bed of the sinus tonsillaris, to which it is loosely attached, the capsule is followed closely with the author's scalpel, care being exercised to avoid injuring the superior constrictor muscle, which forms the bed of the sinus tonsillaris.

(*d*) Continue to pull upon the tonsil with the forceps. Then introduce the knife through the upper part of the incision, follow closely the capsule of the tonsil, and sever it from its attachment to the superior constrictor muscle, as shown in Fig. 283. The branches of the tonsillar artery are severed in this step of the operation. They are small and do

not often give rise to hemorrhage. If, however, some of the fibers of the superior constrictor muscle are accidentally removed the main stem of the artery is severed and the hemorrhage may be severe. If the hemorrhage is severe, the bleeding points should be seized and twisted with artery forceps. The edge of the blade should be slightly turned to the tonsil, as this will avoid injuring the superior constrictor muscle of the pharynx.

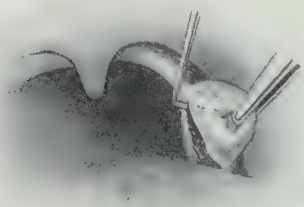
FIG. 284



The tonsil is drawn toward the median line of the throat to expose the posterior pillar to the knife. The pillar is incised to the bottom of the tonsil at its junction with the tonsil.

(*e*) Disengage the vulsellum forceps from the tonsil and place the tip of one prong in the anterior aspect of the wound, the other over the inner aspect of the tonsil, and close them upon the tonsil (Fig. 284). Tract the anterior border of the tonsil toward the median line of the throat, using the posterior pillar as a hinge.

FIG. 285



The Beck-West method of beginning the enucleation of the tonsil, *i. e.*, by separating the posterior pillar.

(*f*) Then, having rendered the posterior pillar accessible, shave it free from the posterior border of the tonsil with the scalpel (Fig. 284). Great care should be taken to avoid injuring the muscular tissue of either the anterior or posterior pillars during the dissection. If the muscles are not injured, there is little chance of hemorrhage from these regions, as the artery is within the muscular substance of the pillars.

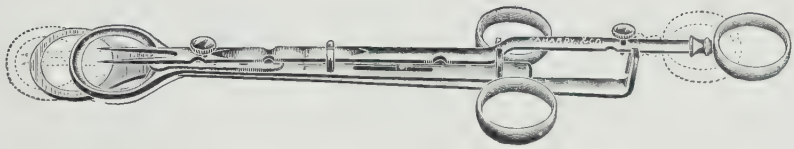
(*g*) The tonsil is now only attached at its inferior portion. While still pulling the tonsil toward the median line of the throat, complete the dissection by cutting downward and medianward. The tonsil is thus removed with its capsule intact. The first incision separates the anterior pillar

and the plica supratonsillaris from the anterior and superior surfaces of the tonsil. The second separates the outer surface of the tonsil from the superior constrictor muscle of the pharynx. The third separates the posterior pillar from the corresponding border of the tonsil. The fourth incision completes the dissection by freeing the inferior attachment of the tonsil from the pharyngeal wall.

Since adopting this method of operating, I have seen no alarming hemorrhages except in a few instances, in which some fibers of the superior constrictor muscle of the pharynx were injured. The hemorrhage was primary, and was easily controlled by a solution of permanganate of potash ($\frac{1}{8}$ grain to the ounce of water) or with a hemostat.

The Complete Removal of the Tonsil with a Tonsillotome and Punch Forceps.—This method of operating is the simplest way to remove the entire tonsil, and is especially recommended for children with large protruding nostrils. It is also recommended to general practitioners and inexperienced throat surgeons in both children and adults on account of its simplicity and thoroughness. I have used it in hundreds of cases with complete satisfaction.

FIG. 286



Tonsillotome.

Technique.—(*a*) Induce cocaine anesthesia.

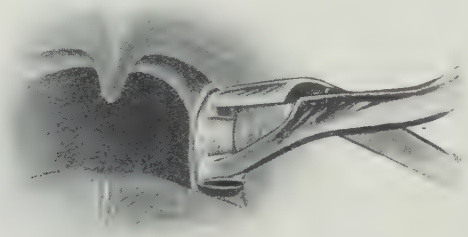
(*b*) Remove as much of the tonsil with the tonsillotome (Fig. 286) as possible. (See Tonsillotomy.)

(*c*) Remove the remaining substance of the tonsil with the Ruault, Rhodes, or Farlow punch forceps. The forceps should have a heavy female blade, with a wide flange, to push the pillars away from the male or punch blade (Fig. 287). The closed forceps should be introduced between the pillars with the cutting surfaces at right angles to the pillars, as in this position they may be opened and closed without cutting the pillars. If introduced with the cutting surface of the blades parallel with the pillars, the pillars may be injured or cut away. When properly placed the forceps should be *forced* into the sinus tonsillaris and opened and closed until the remainder of the tonsil is completely removed. I use the Ruault forceps and exert from five to twenty pounds' pressure upon the shank of the instrument with the left hand while it is in action. I have never injured the superior constrictor muscle with it, nor have I failed to remove all the remaining tonsillar tissue with it.

(*d*) When the punch forceps are removed the index finger should be introduced into the wound to search for fragments of the tonsil.

These fragments feel firm to the touch, and in sharp contrast to the smooth and soft bed of the sinus. If fragments of the tonsil still remain *in situ*, introduce the punch forceps and remove them.

Fig. 287



The removal of the tonsil with the Ruault tonsil punch forceps after the preliminary separation of the pillars.

(*e*) Having completed the operation, mop the sinus tonsillaris free of blood and search for bleeding points. If found, seize them with an artery forceps and twist them.

Fig. 288

Robertson's tonsil scissors. The scissors are made in pairs.

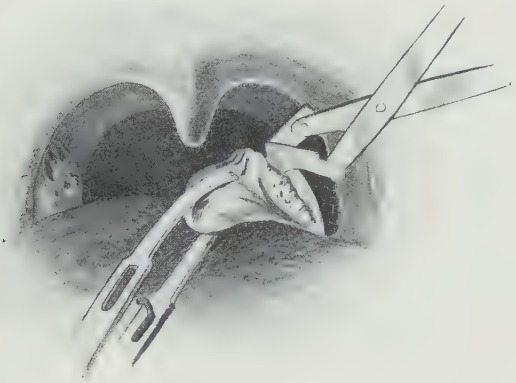
Robertson's Operation.—Robertson's method of removing the tonsil is as follows: (*a*) A general or local anesthetic may be used.

(*b*) The anterior and posterior pillars are first separated from the tonsil with a curved double-edged knife, or, if the pillar is adherent, with his pillar scissors.

(*c*) The tonsil is then grasped with forceps and pulled forward and

inward, the scissors pushing the pillars back out of the way. The scissors are then closed and the tonsil removed by a series of cuts (Figs. 288 and 289). The tonsil upon the opposite side shows the position of the tonsil before it was pulled from its sinus.

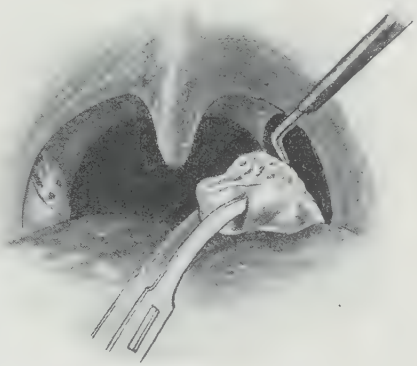
Fig. 289



The removal of the tonsil with Robertson's scissors.

This operation may also be performed under local anesthesia, as in the author's method. The tonsil may also be removed in its entirety with its capsule intact by this method, though Robertson did not advocate this until recently. The tonsil scissors are made in pairs to adapt them to either side. This method of removing the tonsils is thorough and commendable. The prime question in reference to any operation on the tonsils is that of its completeness.

Fig. 290



Pynchon's cautery dissection.

Pynchon's Cautery Dissection Operation.—According to Pynchon, this method of removing the tonsil in its entirety possesses the advantages of (*a*) but slight or no primary hemorrhage, and (*b*) the sealing of the wound by the eschar, thus preventing severe infection of the wound. Dr. Pynchon was the first to systematically remove the tonsil in its entirety, he having done this for twenty-five years. He did not, however, attempt to remove it with its capsule intact, as I have done for fifteen years.

Technique.—(*a*) Induce local anesthesia by repeated swabbings, first with a 10 per cent. solution of cocaine, and then with a 20 per cent.

solution. To each solution of cocaine should be added one-half as muc carbolic acid as cocaine. If preferred, the anesthesia may be induced by injecting cocaine and adrenalin or the infiltration solution.

(*b*) Seize the tonsil with mouse-toothed forceps at about its central portion and pull it inward and backward, thus putting the plica tonsillaris and the anterior pillar upon a tension. This renders the anterior border of the tonsil easily discernible.

(*c*) With a nearly straight cautery electrode at a cherry-red heat, puncture the membrane at the junction of the anterior pillar and the plica tonsillaris about one-third the distance from the top of the tonsil, and dissect downward to the tongue. Then dissect upward over the margosupratonsillaris and a little way down the posterior junction of the tonsil and pillar (Fig. 290). In other words, make the incision shown in Fig. 282.

(*d*) With a nearly right-angle electrode (Fig. 290), complete the dissection of the posterior pillar from the tonsil.

(*e*) Pull the top of the tonsil inward and downward, and dissect it, with the electrode, from its attachment to the superior constrictor muscle, thus freeing it from the sinus tonsillaris.

(*f*) The remaining pedicle, at the base of the tonsil, is severed by stretching it over the heated electrode.

(*g*) Only one tonsil is removed at a sitting, the remaining tonsil being removed in about two weeks, or after the first wound has healed.

(*h*) Applications of a 20 to 30 per cent. aqueous solution of the nitrate of silver may be made from time to time during the operation, to check oozing hemorrhage.

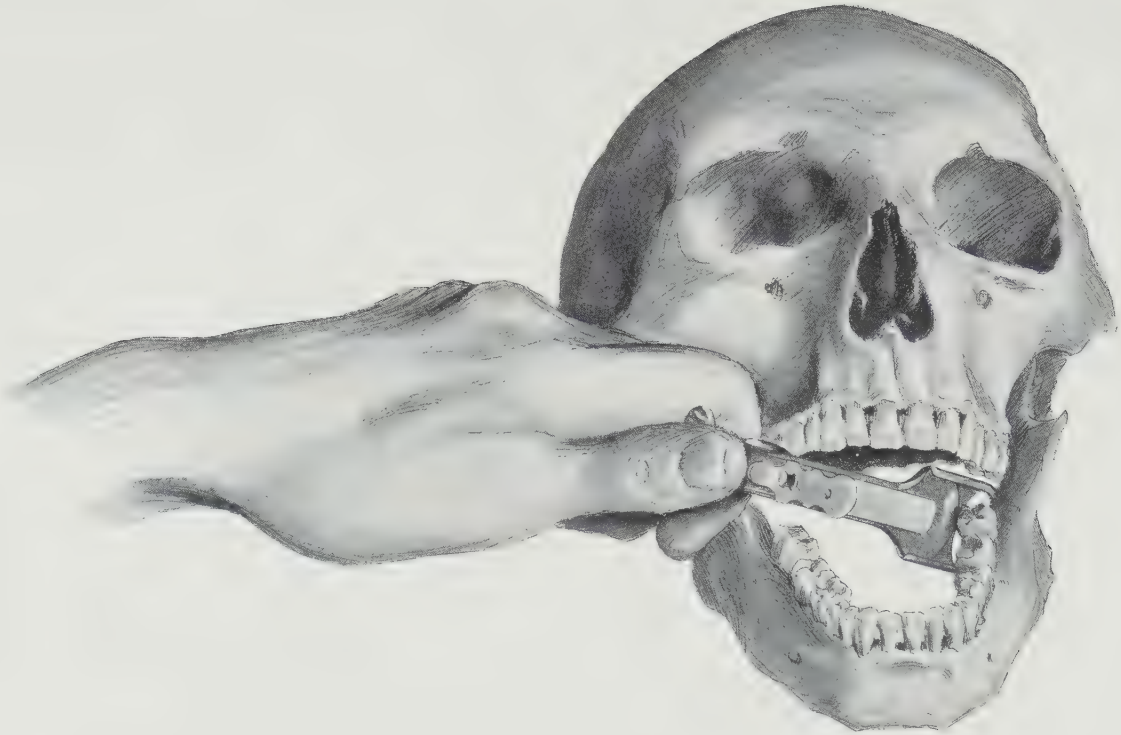
(*i*) The after-treatment should consist in the use of alkaline and aromatic gargles and the daily application of the following mixture:

℞—Tr. iron,
Glycerin . . . . . āā ℥j

The above mixture should be rubbed into the wound with a cotton-wound applicator, to prevent infection and exuberant granulations. The wound should heal with a smooth surface and without the formation of cicatricial bands. If the muscular tissue of the pillars is injured, contracture and disagreeable deformity of the fauces may result.

The Sluder Guillotine Operation.—Sluder's method of removing tonsils is unique in its simplicity, and because it reverts to the use of an old, and, in America, almost obsolete instrument, namely, the guillotine. The guillotine and tonsillotome have in America been supplanted by various dissecting knives and scissors, some blunt-tipped, curved, angular, etc., in an endeavor to adapt them to the peculiar anatomical requirements (real and imaginary, mostly the latter) for the complete enucleation of the tonsil. Personally, I have heretofore used the straight bistoury from the beginning to the end of the operation, with great satisfaction. I must, however, confess that Sluder's method is simpler, safer, and better in every way, though it cannot be used

PLATE XII



Showing the Method of Using the Guillotine in Position for the Sluder Operation on the Tonsil.

PLATE XIII

Showing the Method of Using the Guillotine in Position for the Sluder Operation on the Tonsil.

in more than 75 per cent. of the cases. Most laryngologists separate the pillars with scissors or peculiar angular knives, and then place a wire snare over the tonsil and pull it through the loop and complete the operation with the snare.

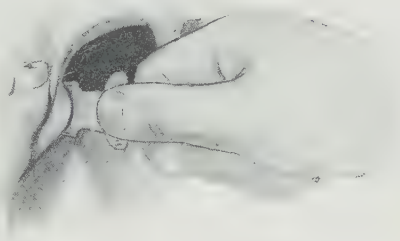
All of these methods are practical, and are to be commended, because they accomplish the necessary result—namely, the *complete removal of the tonsil with its capsule intact*, as first advocated by me in 1898. Until five years ago there was much opposition to my contention for complete tonsillectomy, whereas it is now almost universally adopted by American laryngologists.

FIG. 291

Sluder's tonsil operation. First step.

It must be admitted that tonsillectomy has more "terrors" for the laryngologist than tonsillotomy ever had, and the reasons are not difficult to discover. Tonsillotomy was formerly performed chiefly upon young children, rarely upon adults. Both tonsillotomy and tonsillectomy are more serious procedures in adults—hence, one reason for the increasing dread of tonsil operations. Another and more important reason is, that, whereas, formerly the operator depended almost exclusively upon the guillotine and tonsillotome (semi-automatic instruments), he now depends upon knives and scissors which require much more personal dexterity and so-called surgical skill for their successful use. With these instruments, the muscular bed of the tonsil is often injured and the bleeding more profuse in consequence.

FIG. 292



Second step: the tonsil dislocated forward over the tubercle alveolaris.

Dr. Greenfield Sluder has attempted to restore order where chaos existed, confidence where fear reigned, and simplicity in place of complexity—all, by the return to the use of the guillotine in the removal of tonsils. By his method, the guillotine no longer merely decapitates the tonsil, but *removes it with its capsule intact.*

Rationale.—Heretofore the objection to the removal of the tonsil with the guillotine and tonsillotome was that neither would remove the entire gland—it was only decapitated or partially removed. The simplicity of the technique required in the use of these instruments made them very popular, and they were almost universally adopted. When the modern movement for the "*complete removal of the tonsil with its capsule intact*" became the rule of practice, the guillotine and tonsillotome were relegated to the vast heap of discarded instruments. Most laryngologists will gladly restore the guillotine to the list of useful

Fig. 293

Third step of Sluder's operation: pushing the tonsil through the fenestra of the guillotine.

instruments when they learn that with it they can remove the tonsil in its entirety in as few seconds as was formerly required to decapitate it. The complete enucleation of the tonsil will have lost many of the modern terrors, and will assume its former place as one of the simpler and comparatively safe surgical procedures if Sluder's operative technique is adopted. The *hemorrhage* in Sluder's operation under *ether anesthesia* is considerable, though it is oozing in character, rather than spurting. This feature can no doubt be overcome by the preliminary injection of adrenalin. In all my cases operated by his method, *not*

a shred of muscular tissue has been removed, a point of great importance in operating upon *singers*. Apparently the only tissue cut was the mucous membrane of the two pillars at their junction with the tonsil. The muscular bed upon which the tonsils rested seemed to have been separated from the capsule of the tonsil without the slightest injury to the muscular tissue. Another fact has impressed me, namely, that not infrequently little or no soreness in the throat follows the operation in children. The reason is obvious; the only tissue incised was the mucous membrane, while the tonsil capsule was separated from the muscular tissue without trauma.

The *fundamental facts* underlying Sluder's technique are three in number, namely: (*a*) The guillotine will remove the tonsil with its capsule intact, provided the tonsil is pushed through the fenestra of the instrument. The advisability of pushing the tonsil through the fenestra of the guillotine has long been recognized, as is exemplified by the oft-repeated advice to exert pressure under the angle of the jaw during the removal of the tonsil. This manipulation was, however, rarely attended with success.

(*b*) The sinus tonsillaris (bed of the tonsil) is freely movable, allowing the tonsil to be dislocated forward and upward a distance of about one and one-half inches.

(*c*) At a distance of one and one-half inches anterior and superior to the tonsil is located a bony prominence on the inferior maxilla, called by Sluder the eminentia alveolaris. This eminence corresponds to the location of the last molar tooth (Fig. 296, *a*).

These facts are put to practical use in the Sluder operation. The tonsil is displaced forward and upward over the tubercle, which in turn pushes it through the fenestra of the guillotine; the guillotine blade is then pushed home, removing the tonsil with its investing capsule.

Technique.—(*a*) In the removal of the *right tonsil*, the patient in the upright position, the Sluder guillotine is grasped by its handle with the operator's *right hand* and introduced through the *left angle of the mouth* until the distal portion of the fenestral margin is in contact with the inferior and posterior portion of the tonsil.

(*b*) The instrument is then pressed firmly against the tissues, which are drawn forward and upward for a distance of about one inch. The tonsil then rests over the eminentia alveolaris, which pushes the tonsil through the fenestra of the guillotine (Fig. 292), though it may not push it all through the opening. If the blade of the instrument were forced home at this stage of the procedure, the tonsil would not in all probability be removed in its entirety, with its capsule intact. Instead, only the superficial portion of the tonsil would be removed. To obviate this mishap, the tonsil is drawn a little farther forward until the distal margin of the fenestra rests almost upon the apex of the eminence. The handle of the instrument is then slightly depressed, to bring the inferior portion of the margin of the fenestra in firm contact with the inferior portion of the tonsil. This leaves the tonsil exposed to view (Fig. 291). The left index finger is then used to push the tonsil

through the fenestra (Fig. 292). The blade of the guillotine should, however, be gently pressed against the anterior portion of the tonsil, to hold it in position while the balance is being pushed through the fenestra with the tip of the left index finger.

(*c*) In the third step of the operation the remainder of the tonsil is pushed through the fenestra with the tip of the left index finger (Fig. 293). As the tonsil continues to pass through the fenestra, the blade of the instrument is advanced by gentle pressure with the thumb of the right hand.

The tonsil tissue is readily detected by the sense of touch, as it is firm and nodular, whereas, the mucous membrane is soft, thin, and smooth in texture. When the tonsil is completely through the fenestra of the instrument, only the smooth, thin mucous membranes of the anterior and posterior pillars lie between the tip of the finger and the distal margin of the fenestra. The blade is at this time advanced until only the two mucous membranes lie between its cutting edge and the distal margin of the fenestra.

(*d*) The blade is then forced home, with considerable power, both hands often being required for this purpose if the blade is dull.

This completes the removal of the right tonsil.

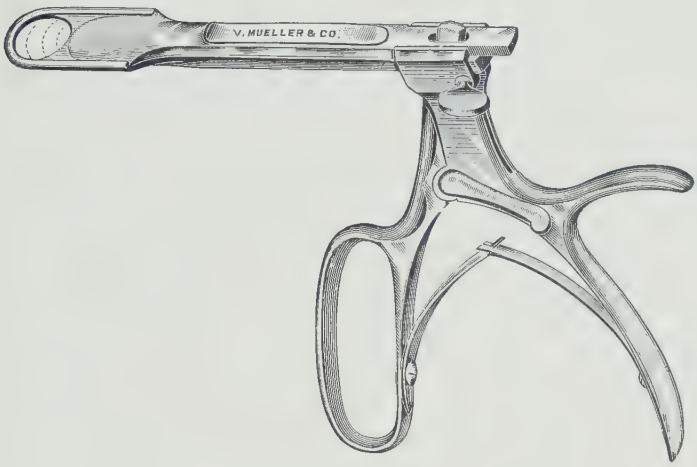
Position of the Surgeon in Relation to the Patient.—When the patient is in the upright position, the left tonsil is removed with the guillotine grasped in the left hand, the index finger of the right hand being used to force the tonsil through the fenestra of the guillotine. In all other respects the technique is the same if the operator is ambidextrous. If he is not ambidextrous, that is, can only use the instruments with one hand (the right), he will have to change his relative position to the patient in operating upon the left tonsil. If the patient is in the sitting posture, the operator will stand to the right and a little behind the patient, and lean forward over his head to remove the left tonsil.

When the patient is lying face upward upon a table and the operator is standing, he should stand on the right side of the patient, facing the head of the patient, to remove the right tonsil; to remove the left tonsil, the operator should stand at the head of the patient facing his feet. In this way the guillotine may be held in the right hand for the removal of both tonsils. The right tonsil is removed with the instrument in the right hand, the left index finger pushing the tonsil through the fenestra. The left tonsil is removed by the guillotine in the right hand, the left index finger pushing the tonsil through the fenestra. The surgeon stands on the right side of the patient and faces him.

Instrumentarium.—The only instrument required for this operation is the Sluder guillotine. The McKenzie or other models of the guillotine are not properly constructed for this operation, as the distal face of the fenestra must be applied to the tonsil in order to keep the handle of the instrument and the operator's hands from obstructing the view of the field of operation. A casual examination of the other models of the guillotine will readily demonstrate their unfitness to stand the pressure exerted in dislocating the tonsil forward and upward over the

eminentia alveolaris. Sluder's instrument and my modification of it have shorter and heavier shanks, especially constructed to withstand the pressure necessarily exerted in this operation.

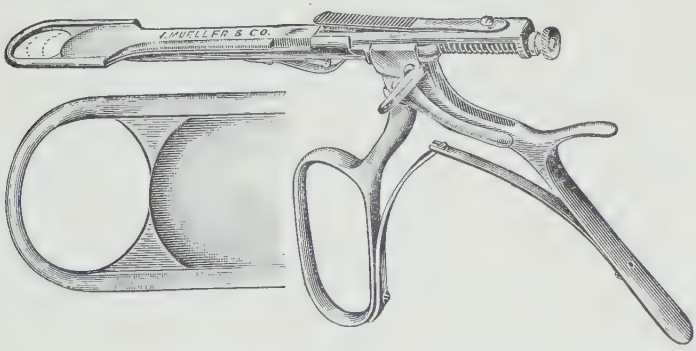
FIG. 294



The author's tonsillectome with sharp blade and scissors-handle.

Since the third edition of this work I have removed many hundreds of tonsils by the Sluder method, though a somewhat different technique was employed. I have modified the guillotine by adding a scissors-handle (Figs. 294 and 295), which greatly facilitates the work and

FIG. 295



The author's tonsillectome with dull blade, equal to No. 1 piano wire. This instrument drives the blade home with three closures of the scissors handle, and is designed to replace the tonsil snare.

requires very much less muscular power to cut through the tissues. Fig. 294 is supplied in two sizes, and has a sharp blade. Fig. 295 is supplied with a dull blade of the thickness of a No. 1 wire, and is designed

to take the place of the wire snare. The use of the dull instrument is attended by less hemorrhage than the sharp one. The dull instrument works with a rachet device and has great power; the sharp instrument works with direct power from the scissors-handle. I now pull the tonsil against the tip of my finger instead of the alveolar eminence, suggested by Dr. Sluder. This accomplishes the same purpose and simplifies the whole procedure.

Present Technique.—Introduce the tonsillectome (either Fig. 294 or 295) as heretofore described (Plates XII, XIII and Fig. 291) and engage the tonsil with the distal rim of the fenestral ring. Then introduce the index finger of the disengaged hand into the mouth of the patient, against the anterior pillar. Drag the tonsil forward and above the inferior molar teeth against the ball of the tip of the index finger. The finger holds the tonsil in the fenestra of the instrument, and at the same time pushes it through it. When the tonsil is through the fenestra close the scissors handle and drive the blade home. If the dull-bladed instrument is used three movements of the scissors-handle are required to send the blade home. As the blade "goes home," the two pillars are brought together external to the tonsil and severed from their attachment to it. The whole procedure need require but a few seconds or a minute at the most in a suitably selected case. Fully 75 per cent. of all cases, including adults, are operable by this method. Sluder claims 99 per cent. of successes by it.

Contraindications to the Sluder Operation.—1. Deep adhesions of the tonsil to its muscular bed, especially found in cases previously subject to quinsy or repeated severe anginas. When such adhesions are present it is difficult or impossible to drag the tonsil from the sinus tonsillaris against the finger.

2. Very thin flat tonsils are not suited for removal by this method, as there is not enough substance or bulk to them to allow the instrument to readily engage them.

Tonsillotomy.—The author has elsewhere expressed his views as to the inadvisability of removing a portion of the tonsil, but inasmuch as it is a time-honored procedure, and is likely, for various reasons, to be practised in the future, it will be described in this chapter.

Technique.—(*a*) The operation may be done under either local cocaine, infiltration, or general anesthesia.

(*b*) If the subject is an infant or a young child, and the operation is to be performed under either local or nitrous oxide gas or bromide of ethyl anesthesia, he should be held in the lap of an assistant. He should be wrapped in a sheet tightly pinned around his body and one arm, while his head should be grasped by the assistant's left arm and hand. The legs of the assistant should be crossed over those of the child, to prevent struggling during the operation. If a general anesthetic is administered, one arm should be left exposed to test the pulse and the muscular reflexes.

(*c*) A mouth gag may or may not be used, according to the discretion of the operator.

(*d*) Depress the tongue with a tongue depressor, to expose the tonsil to full view.

(*e*) Introduce the tonsillotome into the mouth of the child, place the ring blade over the tonsil and forcibly push it outward, and at the same time bring the ring blade forward to engage the tonsil.

(*f*) When the tonsil protrudes through the ring blade, close the instrument and thus cut off as much of the tonsil as happens to protrude through it.

It occasionally happens that the entire tonsil with its capsule intact is removed by this method of operating. More often only a portion of it is removed. The upper portion is often quite inaccessible to the ring knife, and as this usually contains the more diseased crypts, the operation is but partially effective.

Complications and Sequelæ of Operations on the Tonsils.—Inasmuch as tonsillectomy is, or should be, performed as often in adults as in children, the question of postoperative hemorrhage and of infection becomes an important one. In children hemorrhage and infection of a severe type are rare, whereas in adults they are much more common on account of the larger development of the vessels and the greater abundance of fibrous connective tissue, which offers less resistance to microbic infection.

Hemorrhage.—(See page 392.)

Infection.—The infection following operations on the tonsils is usually more severe and prolonged in adults than in children. In children the temperature is elevated 0.5° to 2° for two or more days, whereas in adults it is often more highly elevated for from two days to a week or more. The soreness in children is usually limited to three or four days, while in adults it often continues longer. If the infection were only thus manifested, it would be a matter of small importance. Unfortunately, it is occasionally so severe as to be alarming, even to the point of actual danger to life itself. While I have never seen a case result in death, I have seen a few assume alarming symptoms. That is, I have seen two, in about 9000 cases, in which the hemorrhage was so prolonged that marked anemia and exhaustion resulted, and two of severe sepsis from streptococcus infection.

If the cases with secondary hemorrhage had been operated upon in the hospital, the bleeding could have been more quickly controlled and the danger averted, or, indeed, it might not have occurred, as the patients would have remained quiet in bed.

In one of the septic cases the tonsils were removed by partial dissection and completed with a snare, whereas in the other case the dissection was done with a sharp scalpel. In the latter case the infection was the more severe of the two, a fact which apparently controverts my previous statement that a clean-cut dissection is less apt to be followed by infection than a dull-cut or crushing dissection with a snare. In spite of the apparent discrepancy, I wish to reaffirm my previous statement

that dissection with a sharp instrument is less likely to be followed by severe secondary infection than one done with dull-cutting or crushing instruments. Another factor which must be taken into account is the virulence of the infective microörganism causing the infection. If a virulent type of streptococcus is the infective agent, the resulting infection and sepsis will be severe, no matter what method of dissection is used. Crushed tissue is less resistant than tissue cut with a sharp instrument, hence it is more readily infected, though either may be the seat of infection. The whole question is one of the microörganism on one side and of the tone or resistance of the tissues on the other. If the resistance of the tissue is normal and the virulence of the microörganisms are great, infection will follow. If the resistance of the tissue is low and the virulence of the microörganism is low, there may or may not be infection, according to the balance or lack of balance existing between the resistance of the tissues and the virulence of the infecting microörganisms. It follows, therefore, that the question of infection is not wholly dependent upon whether the dissection is performed with blunt or with sharp instruments, but that the general tone of the tissues previous to the operation, the local tone as affected by either blunt or sharp instruments, and the virulence of the invading microörganism each has its influence in determining the severity of the infection and the resulting sepsis.

Fig. 296

Right half of inferior jaw, showing (*a*) the eminentia alveolaris.

The practical deductions to be drawn from the foregoing statements are as follows:

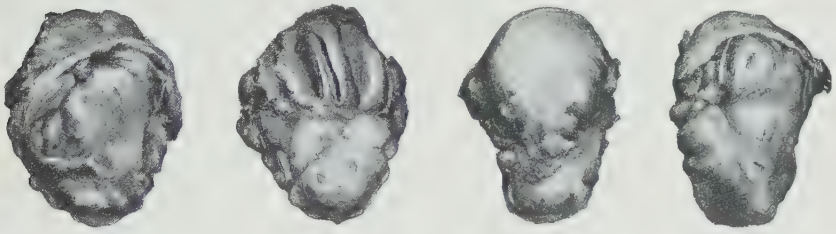
1. If the patient's vital forces are low, tonics and fresh air should be prescribed for some time before the operation. It is true that it is not often advisable to delay the removal of the tonsil until the general tone of the system is elevated, as the tonsils may be the direct cause of the lowered vitality of the patient, and should be removed to stop the toxemia. Under such circumstances the risk of the infection and sepsis must be assumed, and such measures adopted as will avert or minimize the intensity of the two processes.

2. The resistance of the tissues is influenced by the previous local disease and by the character of the dissection. The local changes due to previous disease of the tonsil cannot, perhaps, be eliminated,

and, in so far as this factor is concerned, the operation must be performed in spite of them. In so far as the tone of the local structures is affected by the character of the dissection, this is entirely under the control of the operator. He can avoid the use of crushing instruments by substituting sharp ones. While this precaution will not always prevent infection and sepsis, it will reduce the number and severity of the infections.

3. The virulence of the microörganisms in the throat may be determined before the operation by the adoption of the routine practice of making cultures from the tonsils. This is not always practicable, but when it is, it should be done. Another way of arriving at much the same result is to carefully inspect the tonsil, especially the crypts in the supratonsillar fossa and those covered by the plica tonsillaris, and note the local signs of irritation and inflammation, especially redness of the mucous membrane. Still further information may be obtained by questioning the patient as to the presence of soreness or pricking upon swallowing. If these signs are present, it is wise to defer the operation until the crypts are cleaned out and the local irritation and inflammation have subsided.

Fig. 297



Tonsils removed by Sluder's method. (Sluder.)

There is a possibility that severe infection may follow the removal of the tonsil, even in cases in which there is no apparent inflammation. Virulent germs may be lodged in the bottom of the crypts without giving rise to obvious symptoms. Close inquiry may elicit the statement that the patient has a slight soreness upon swallowing, a sensation of pricking. In one such case in the author's practice a most violent and obstinate infection occurred. The patient, a rhinologist, came for the removal of his tonsils, and inasmuch as I presumed that he knew whether his throat was in a proper condition for the operation, the tonsils were removed. After the occurrence of the infection he told me that he had been suffering for a week from a slight soreness or pricking in the throat. These facts show that the surgeon should not presume anything, even though the patient is supposedly well informed concerning his condition. All cases should be subjected to close scrutiny by the surgeon before performing an operation.

Should the examination show such soreness to be present, the operation should not be performed. The crypts of the tonsils should be

cleansed of all debris by syringing (Fig. 299) with warm normal salt solution. A curved cotton applicator moistened with the tincture of iodine should be introduced into each crypt to allay any infection and inflammation in them. Treatment thus carried out for one week will usually prepare the tonsils, so that the operation may be performed

FIG. 298

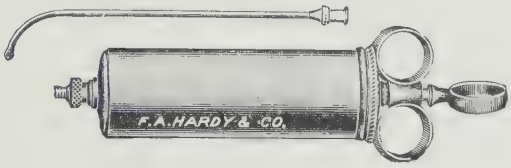
Reverse Trendelenburg position for the removal of tonsils.

without the danger of infection of tonsillar origin. It is urged, therefore, that the surgeon should always prepare the tonsils for operation, just as he would any other part of the body. The same rule should be applied to the nose, throat, and larynx, even though these regions are not susceptible to absolute surgical cleanliness. The breeding or incubating foci can at least be eradicated.

Is Tonsillectomy a Hospital Operation?—In young children it is not necessarily a hospital operation, because it is rarely followed by either severe hemorrhage or sepsis. In adults it should be a hospital operation, on account of the possible hemorrhage and sepsis.

A prominent surgeon has said that the tonsil is of greater clinical importance than the appendix; that it causes more suffering and more deaths. If this is true, and I believe it is, the tonsil is worthy of the most serious and painstaking study.

Fig. 299



The author's tonsil syringe.

The technique of its removal should receive the same careful and patient attention that has been devoted to the removal of the vermiform appendix. In view of the importance of the tonsil from a clinical standpoint, and in view of the possible complications and sequelæ following its removal, tonsillectomy should be regarded as a hospital operation. If performed in a hospital, the danger from primary or secondary hemorrhage is largely eliminated, and infection and sepsis may be diminished in severity and in the frequency of their occurrence.

George L. Richards and Charles Richardson advocate the complete removal of the tonsil by finger dissection. The pillars are partially separated with a knife of some description, the finger inserted into the incision and the tonsil separated from the sinus tonsillaris. The fibrous pedicle at the root of the tongue is then severed with a snare or tonsillotome. While this method of enucleation is old, it has awakened new interest on account of the enthusiastic indorsement of these eminent and practical laryngologists.

CHAPTER XXIV

NEOPLASMS OF THE TONSIL

BENIGN NEOPLASMS OF THE TONSILS

Benign tumors do not occur as often in the tonsils as they do elsewhere in the pharynx. Of the variety found in this region, papilloma is the most common.

Papilloma.—Papilloma is more often multiple than single, and presents the general outlines of a bunch of grapes. If single and large, it may be mistaken for a supernumerary tonsil. Like all papillomata, it has a tendency to return, and is sometimes apparently converted into a malignant growth. It should, therefore, be removed by clean surgical excision, rather than by a crude crushing method, as with a snare or dull forceps. It should be borne in mind that the transition from a benign papilloma to a malignant epithelioma is, histologically, rather easy. The epithelial growth in the papilloma is outward, whereas in epithelioma it is inward. There are, of course, other histological differences. The structural arrangements are, however, so similar as to warrant a certain amount of caution and discretion in their diagnosis and surgical treatment.

In some instances there may be one pedicle with many papillomata attached, whereas in others there may be many pedicles.

The growths, as a rule, give rise to no marked symptoms. A slight hacking cough, a tickling sensation, and the feeling of a foreign body in the faucial region are complained of. The only change noted in the surrounding tonsillar tissue is an increased hyperemia around the attachment of the tumor. Pain is never present. The tumors vary in size from that of a pea to a large walnut.

Lipoma.—Lipoma of the tonsil is rare, though Atkinson, Farlow, Ingals and others have reported cases. They are benign fatty tumors.

Angioma.—Angioma of the tonsil is also quite rare. Flatau, Phillips, Bosworth, Keimer and others have reported a few cases.

Treatment.—The treatment is preferably by electrolysis. The positive pole should be applied by means of gold-plated needles thrust into the neoplasm. The strength of the current should vary from 5 to 25 ma., and should be applied for from two to twenty-five minutes at each seance. Repeat the applications once or twice a week until the vascular growth is obliterated.

Fibroma.—Fibroma of the tonsil is a benign neoplasm next in frequency of occurrence to papilloma. It very rarely becomes malignant. Its growth is very slow, and is usually limited to one tonsil. Delevan and others have suggested that fibrous tumors of the tonsils may be

mistaken for supernumerary tonsils. This is especially true if the supernumerary tonsil acquires its fibrous tissue from the degenerative changes due to a constant irritation from its exposed position in the fauces. Technically, it is a fibroplastic fibroma. Some claim that it is only a fibroma which incorporates some of the lymphoid tissue of the tonsil.

Etiology.—Fibroma of the tonsil occurs equally often in each sex, and perhaps more often in the young than in middle and advanced life.

Pathology.—Fibroma is usually somewhat pedunculated, though it may be sessile. The larger the fibroma, the larger the pedicle. It is more often single than multiple. Being of connective tissue of mesoblastic origin, it must of necessity have its origin from the trabeculæ of the tonsil. Occasionally it undergoes cystic degeneration. Usually it is firm and scantily supplied with bloodvessels. It is composed of white fibrous tissue, the cells often being matted together, closely simulating embryonic connective-tissue cells.

Symptoms.—Annoying symptoms are seldom present, except in the large pedunculated type, in which it produces mechanical obstruction. Its presence is not accompanied by discharge. It is characterized by symptoms similar to those of enlarged or hypertrophied tonsils.

Diagnosis.—The diagnosis is usually easily made, and in case of doubt a portion should be excised and submitted to microscopic examination.

Treatment.—The treatment is purely surgical, and consists in its removal, a procedure easily accomplished if the growth is pedunculated. Occasionally it may be adherent to the tonsil or to the neighboring structures as a result of repeated inflammations of the tonsil.

Surgical Technique.—(*a*) Cocainize the growth and the area around the point of attachment with a 10 per cent. solution of cocaine by repeated swabbings.

(*b*) Separate the points of adhesion with a scalpel or scissors.

(*c*) Pass a cold-wire snare around the tumor, engaging it at its pedicle, or point of attachment.

(*d*) Sever the pedicle by closing the wire loop.

(*e*) Cauterize the stump of the pedicle, and if it penetrates the tonsillar tissue, dissect it to its point of origin.

(*f*) Frequent cleansing with some antiseptic gargle should be practised for about one week, or until healing takes place.

(*g*) Instead of using the wire snare as given in (*c*), the growth may be seized with the vulsellum or other toothed forceps and dissected with a scalpel from its attachment to the tonsil, or the tonsil may also be removed.

Fibro-enchondroma.—A few cases have been described, and notable among them is that of Cosolini, in which the growth was as large as an orange, and was readily enucleated. Grosvenor also reported one case.

Cystoma.—Cystoma of the tonsil is rare. It may be either superficially or deeply situated. Virchow reports having found them postmortem. I have occasionally found them of small size when enucleating hypertrophied tonsils. They vary in size, and may contain a quantity of fluid or a mass of inspissated secretions and epithelial debris.

They give rise to no peculiar symptoms other than those usually present in enlarged tonsils.

They may be eradicated by freely incising them with a bistoury and curetting the lining membrane, and then swabbing the cavity with pure carbolic acid to excite reactionary inflammation and agglutination of the opposed walls. A still better method of treatment is to enucleate the tonsil as described under Tonsillectomy.

Lymphadenoma in Hodgkin's Disease.—In every case of Hodgkin's disease, it is advisable to examine the tonsils, as they may be the seat of a lymphadenoma such as is present in other parts of the body. In the early stage of the disease it may be impossible to assert positively that the tonsils are involved, though they may appear abnormally enlarged. In the author's case the tonsils did not appear to be enlarged. By keeping the case under observation their growth may become apparent, and when it occurs is quite significant. Lymphadenoma of the tonsil is only a local expression of a disseminated lesion of a similar nature throughout the general lymphatic system. In my case the tonsils were not apparently involved, though the neck glands were enormously enlarged. The case improved markedly under the application of the Röntgen rays.

MALIGNANT NEOPLASMS OF THE TONSILS

Carcinoma of the Throat.—According to some authorities carcinoma is more frequently found in the tonsils than sarcoma, while others hold the reverse opinion. More than 100 cases have been recorded, and, according to Bosworth, it occurs once in every 2000 cases of carcinoma in all parts of the body. It is a disease of middle and advanced age, though J. D. Bryant reports a case in a patient aged seventeen years. Sarcoma may occur at any age, but more often in early life. The youngest case coming under my observation occurred at the eighteenth month. Cases of sarcoma have been reported as late as the eightieth year. The average age at which carcinoma develops is about the fifty-second year.

Carcinoma of the tonsil is more malignant than sarcoma because of the histopathological predominance of glandular epithelium. It is rarely primary, but is usually secondary to carcinoma of the tongue or pillars of the fauces. It is usually characterized by a squamous and spindle-cell epithelium. It does not attain the large size of sarcoma of the tonsils, but it involves the neighboring lymphatic glands at an earlier period.

Symptoms of Carcinoma.—Early ulceration, a fetid breath, more or less pain of a lancinating character, emaciation, and cachexia are the usual symptoms. Before ulceration the secretions are of a heavy mucous nature, while after ulceration they are often purulent in character. Slight hemorrhage is a frequent symptom. It may, however, in exceptional cases, be very profuse and cause death. Edema of the glottis is frequently present; indeed, one might say it is an almost constant concomitant complication of carcinoma of the tonsil in the advanced stage.

Pain is always aggravated during the act of swallowing, and the voice is either hoarse or aphonic. Secondary glandular involvement is an early feature. The subjective symptoms are very little different from those of sarcoma of the same region, except in the advanced stage, when ulceration and pain are present.

Diagnosis.—Carcinoma of the tonsil is a disease of middle and advanced life, while sarcoma more often occurs in the young. Ulceration occurs early in carcinoma and later in sarcoma; carcinoma is nodular, while sarcoma is smooth and round. Carcinoma has a fleshy pink hue and is often fungoid, while sarcoma is blue in color and is crossed by rather large arteries.

When in a state of ulceration carcinoma may be mistaken for syphilis, particularly if the adjacent glands are not much involved.

The progress of the case and the administration of the iodides will soon clear the diagnosis.

The pain in carcinoma is lancinating and sharp, while it is dull and periodic in sarcoma.

Papilloma is painless, pedunculated, seldom ulcerates, and secondary involvements by direct extension of metastases do not occur. There are no constitutional symptoms, and the growth is multiple and presents the appearance of a bunch of grapes.

Fibroma of the tonsil has a constricted base, grows very slowly, is free from pain and glandular involvement, and does not recur when removed.

A microscopic examination of the tissue should be made in differentiating the various types of tumors.

Differential Diagnosis of Sarcoma and Carcinoma of the Faucial Tonsils

Sarcoma	*Carcinoma*
1. Any age, most often after fifteen.	1. Not in early life, usually after forty.
2. Frequently primary.	2. Rarely primary.
3. Glandular involvement late.	3. Glandular involvement early.
4. Frequently encapsulated.	4. Not encapsulated.
5. Vascular; hemorrhages; ulcerates late.	5. Not so vascular; scant hemorrhage; ulcerates early.
	6. Frequent in males.

Treatment.—The treatment of carcinoma and sarcoma of the tonsil is palliative and surgical, though in most cases the latter affords little encouragement.

EXTIRPATION OF THE TONSIL BY THE EXTERNAL ROUTE

In malignant disease of the tonsils where the surrounding tissues have become involved, it may become necessary to remove the tonsil by the external route by von Langenbeck's method.

Technique.—(*a*) A general anesthetic should be given.

(*b*) The external incision is in the form of a U, thus making a tongue-shaped flap (Fig. 300). The flap thus made lies immediately over the

ascending ramus of the lower jaw. This portion of the jaw is to be temporarily resected, so as to expose the tonsillar region to operation.

(*c*) The external maxillary artery (facial) is ligated to control the hemorrhage.

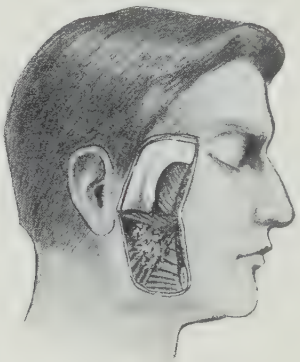
(*d*) The periosteum corresponding to the anterior incision should be divided preparatory to sawing through the bone.

(*e*) The jaw bone is sawn through along the line of the periosteal incision just in front of the insertion of the masseter muscle.

(*f*) The connective-tissue attachments of the ascending ramus of the jaw on its inner surface are then carefully dissected from the bone, care being exercised to avoid injuring the muscles of mastication.

(*g*) The ascending ramus of the jaw is then lifted outward and upward, thereby exposing the region of the tumor to view (Fig. 300).

FIG. 300



Temporary resection of the ramus of the inferior maxilla to expose the fauces in the removal of malignant tumor of the tonsil.

(*h*) The tumor is then exposed by dissection. The external carotid artery lies externally and posteriorly.

(*i*) The tumor should be removed with knife and scissors, care being exercised to avoid opening into the cavity of the mouth until the last moment, so as to keep the secretions from entering the wound.

(*j*) The ascending ramus of the jaw is then returned to its normal position and sutured with wire.

(*k*) The skin is then sutured with horsehair or with Harris' buried suture.

(*l*) The wound is dressed through the mouth, healing taking place by granulation, as after an ordinary tonsillectomy.

PART III

DISEASES OF THE LARYNX

CHAPTER XXV

INFLAMMATORY DISEASES OF THE LARYNX AND EPIGLOTTIS

ACUTE INFECTIOUS EPIGLOTTITIS

Synonyms.—Angina epiglottidea anterior (Michel); acute infectious epiglottitis (Theisen).

The disease is often primary, and is an acute infectious process. Clement F. Theisen reports three cases, and gives a most admirable review of the literature on the subject. Michel, in 1878, first described an inflammatory process, involving the anterior surface of the epiglottis, under this name. It is usually accompanied by more or less circumscribed edema. While the larynx may be somewhat involved in some cases, Theisen claims that true angina epiglottidea occurs quite often as a primary, separate, distinct condition.

Etiology.—In the diffuse type of inflammation the epiglottis may become inflamed by an extension from acute tonsillitis, pharyngitis, or lingual tonsillitis. In the true primary type its origin is not thus explained. In the cases reported by Theisen there was no history of coryza, or other acute infectious condition of the upper respiratory tract. The larynx was but slightly involved. The ages of the patients were thirty-six, forty, and fifty-nine years respectively, one male and two females. Hajek's experiments show that the submucosa of the anterior surface of the epiglottis is abundant and the mucosa loosely adherent, while on the laryngeal surface it is tightly adherent to the cartilage except at the nodules, where there is some loose submucous tissue. These anatomical facts explain why the edema does not extend to the larynx, as one might at first expect it would do. In excessive edema it may, however, extend to the larynx by way of the submucous tissue of the pharyngo-epiglottic ligament, thence to the arycpiglottic folds. Injury to the epiglottis or the neighboring tissue by swallowing foreign bodies or irritating substances may cause the condition. Hot drinks, raw spirits, or highly spiced liquids may also be regarded as possible predisposing etiological factors. In edema of the fauces due to large doses of

the iodide of potash, the epiglottis may become involved. The infectious fevers are also likely to give rise to this distressing condition.

Perichondritis, carcinoma, and ulcerative conditions due to syphilis or tuberculosis may suddenly become complicated by it.

Bacteriological examinations made in two of Theisen's cases showed Streptococcus aureus and pneumococcus in one and Staphylococcus albus and pneumococcus in the other. The atrium of infection in some instances seems to be a traumatic wound, in others it is an extension of an acute inflammation from contiguous anatomical parts, and in a third class it is a malignant tuberculous or syphilitic ulcer. The chief cause, then, is a mixed infection, which may or may not be preceded by a gross lesion of the anterior surface of the epiglottis.

Pathology.—From what has been given under Etiology and Symptomatology, it may be inferred that the pathology is such as is common to acute inflammation of mucous membranes covering loose submucous tissue. This consists of inflammatory congestion, exudation, and edema, which processes, in typical cases, are limited to the anterior surface of the epiglottis. The bacteriological infection is usually the pneumococcus with the Streptococcus aureus or the Staphylococcus albus.

Symptoms.—The onset is sudden, and attended with fever, painful deglutition, stiff, swollen tongue, and dyspnea, especially upon lying down. In one case reported by Theisen, the latter symptom was so severe as to necessitate propping the patient up in bed.

The febrile symptoms are similar to infectious fevers in general.

Upon examination, the anterior surface of the epiglottis is red and swollen, while the adjacent tissues are usually but little, if at all, involved. These symptoms continue with more or less severity for five or six days, when they abate in intensity; the epiglottis, however, remains red and swollen a few days longer.

Diagnosis.—If certain characteristic symptoms are borne in mind, there need be but little difficulty in arriving at a correct diagnosis. These symptoms are: (*a*) Sudden onset. (*b*) A febrile movement. (*c*) Redness and swelling limited to the anterior or lingual surface of the epiglottis. (*d*) More or less painful deglutition.

Acute angioneurotic edema is unattended by fever, and the edematous tissue is pearly gray instead of red.

It should be differentiated from acute miasmatic epiglottitis, which follows exposure to salt marshes, as in hunting for ducks on the mud flats of the California coast. Arnold has described this condition in Burnett's system on the *Nose, Throat, and Ear.* (See Acute Miasmatic Epiglottitis.)

Prognosis.—The prognosis in most cases is good, although deaths have been reported by Tompkins, Louis, Gibb, Crisp, and Fredet. Proper treatment exerts a favorable influence upon its course.

Treatment.—Early scarification of the edematous parts gives prompt relief in some instances. It should be done freely. Meyjer recommends the use of iced ichthyol sprays, which are prepared by putting cracked ice into the spray tube containing the ichthyol solution. Theisen speaks

of using a 0.5 per cent. solution of ichthyol every twenty to thirty minutes while the acute symptoms continue, and at longer intervals afterward. It is important to give early relief, as the patient may not be able to swallow even liquid food until this is done. Calomel and salines may be given advantageously at the onset.

The physician should be prepared to do tracheotomy at any moment, as suffocative symptoms may suddenly develop.

MIASMATIC EPIGLOTTITIS

Arnold, in Burnett's *System*, describes an acute inflammatory process which chiefly involves the epiglottis. It is attended by marked edema of the epiglottis, painful swallowing (odynophagia), and dyspnea.

Etiology.—He attributes the cause "to some animal, vegetable, or chemical poison in the exhalations of the salt marshes." He describes six cases, all of which were men who had returned from hunting ducks on the mud flats of the salt marshes on the California coast. It is probable that the cases were due to a mixed infection from some nidus of propagation in the marsh country along the coast. Whether the cases should stand apart as illustrative of a separate and distinct disease is perhaps doubtful.

Symptoms.—Epiglottic edema and inflammation may be severe, and the adjacent structures somewhat involved. There is odynophagia and dyspnea. In one case the suffocative symptoms became so alarming that tracheotomy was performed. Pyrexia is more or less marked.

ACUTE CATARRHAL LARYNGITIS

Synonyms.—Catarrhal laryngitis; acute catarrh of the larynx; simple laryngitis; laryngitis catarrhalis acuta.

Acute catarrhal laryngitis is an acute catarrhal inflammation of the laryngeal mucosa and of the vocal cords. It is characterized by hoarseness or aphonia, and pain upon phonation.

Etiology.—The etiology of acute catarrhal laryngitis may be studied under: (1) Systemic disturbances and diseases; (2) preëxisting diseases of the upper respiratory tract; (3) hygienic conditions and environment; (4) traumatism; (5) age; (6) climate; (7) idiopathic causes.

1. **Systemic Disturbances.**—Systemic disturbances, such as "catching cold," arthritis, the eruptive specific fevers, syphilis, and tuberculosis, play an important role in the causation of catarrhal inflammations of the larynx. "Catching cold" is a complex process difficult to explain, but in general it may be said to include a lack of balance of the vasomotor nerves whereby the capillary vessels are erratically controlled. Increased vascularity, or congestion, is thus a common disturbance. According to Woakes and J. A. Stucky, the phenomena of "catching cold" are due to digestive disturbances and the final results thereof, *e. g.*, toxic products

in the circulation, which irritate the vasomotor nerves, thus establishing a predisposition to "catching cold." Clinical observation seems to support the above theory in that acute laryngitis quite often follows or accompanies digestive disorders. Arthritis also seems to have a causative relation to laryngitis, and, inasmuch as it is an inflammatory disease of infectious origin, it is easy to appreciate the fact that certain toxins are in the circulation and affect the tonicity of the vasomotor system, very much as in acute coryza, or "catching cold." The toxins of syphilis and tuberculosis likewise irritate and disturb the vocal apparatus. In addition, the pathological lesions are often localized in the larynx, and are specific in character. The exanthematous or eruptive fevers are often accompanied or followed by laryngitis. The specific microörganisms peculiar to these diseases are especially profuse in the upper respiratory tract; indeed, they probably gain entrance to the system through the mucosa of the nose and throat when the resistance is lowered, especially through the tonsils and adenoids; hence, the mucosa of the larynx is subjected to the direct irritation from their presence, as well as to the toxins in the blood.

2. **Pre-existing Diseases.**—Pre-existing diseases of the upper respiratory tract are important predisposing etiological factors in laryngitis. This is especially true in reference to diseases of the sinuses, nasal stenosis, and infectious inflammations of the tonsils. It may be stated as an axiom that *inflammatory processes in one part of the upper respiratory tract tend to extend to contiguous parts.* This is in part explained by the extension by continuity of tissue, and in part by the simultaneous exposure of the various structures to microbic and toxic irritation. The most vulnerable area is first affected, the contiguous parts becoming involved later. The tendency is for the inflammatory process to extend downward rather than upward, probably because the flow of the lymph streams is in that direction. It is true, however, that there is a marked hesitancy in the downward extension from the nose to the larynx. This is explained by the difference in the character of the epithelium covering the mesopharynx. Nearly the whole of the mucosa of the upper respiratory tract, except the mesopharynx, is covered with ciliated columnar epithelium, whereas the mesopharynx is covered with squamous epithelium. Inflammatory processes do not readily extend from one kind of tissue to another, hence the hesitancy. If, however, the nasal inflammation is severe and prolonged, or often repeated, the inflammation finally reaches the larynx. Indeed, the "dropping" into the hypopharynx often leads to catarrhal inflammation of the larynx by lowering the resistance of the laryngeal mucosa, which subsequently becomes infected. In sphenoidal and posterior ethmoidal sinuitis, the secretion and the exudate are discharged into the epipharynx and drop or trickle down the walls of the mesopharynx to the upper surface of the larynx, thus irritating its mucosa. The mucous membrane of the larynx becomes lowered in resistance, and infection and inflammation follow. In obstructive deflections of the septum the respiratory functions of the nose, namely moistening, warming, and filtering the air, are impaired. The pharyngeal and the laryn-

geal mucous membranes are, therefore, subjected to air that is irritating to them. This in time causes lowered resistance, infection, and laryngitis.

We may say, then, in a general way, that diseases of the respiratory tract above the larynx often predispose to catarrhal inflammations of the larynx by (*a*) extension or continuity of tissue; (*b*) by contiguity of tissue; (*c*) by lymphatic communication; (*d*) by irritation and lowered resistance from secretions from the nose and accessory sinuses; (*e*) by simultaneous exposure of the entire upper respiratory tract to microbic infection; and (*f*) by the irritation from the toxins evolved by the bacteria in the nose, the accessory sinuses, the epipharynx, and the tonsils. *The chief barrier to the downward inflammatory extension is in the squamous epithelium of the mesopharynx.*

3. **Hygienic Conditions and Environment.**—Under hygienic conditions and environment as causative agents in catarrhal laryngitis are included (*a*) the inhalation of noxious gases; (*b*) poor ventilation; (*c*) undue exposure of feet and body; (*d*) improper bathing; and (*e*) the abuse of the voice.

The inhalation of noxious gases, as in chemical laboratories, factories, etc., may cause laryngitis by direct irritation, or it may lower the resistance of the tissues and predispose to infection. Poor ventilation likewise causes laryngitis, though not by direct irritation. In the latter instance the vital energy is lowered by breathing impure air. Then, too, the oxygen in the air is diminished in quantity. The vitiated atmosphere irritates the endothelial lining of the air vesicles, and thereby causes changes which interfere with the absorption of oxygen into the blood and the expulsion of carbonic acid gas from the blood. These factors combine to deprive the patient of the normal amount of oxygen, and lead to an overaccumulation of carbonic acid gas. The processes of metabolism are thus deranged, and toxemia results. The vital energies are lowered, and the patient is in prime condition to be affected by bacterial infection and inflammation. Undue exposure of the body, especially the feet, is a prolific exciting cause of laryngeal inflammation. The large vessels of the feet give off large quantities of heat when the soles are insufficiently protected from the cold ground. When this occurs there is a shock to the terminal vascular system, which causes a lack of balance of the physiological functions of the more delicate structures of the body. The larynx in some cases is the vulnerable point, and reacts in the form of a catarrhal laryngitis. The question of clothing is discussed more fully under the etiology of the nasal inflammations. Suffice it to say, therefore, that there is danger in an excessive amount of clothing, as well as in too little. One accustomed to living in an open, poorly constructed residence, and changing to a well-built city residence, which is overheated and poorly ventilated, is especially subject to catarrhal inflammations of the upper air passages.

Bathing, when judiciously practised, is a healthful and invigorating procedure. When, on the contrary, it is injudiciously practised, it may cause considerable mischief to the upper respiratory tract. What is good practice for one may be bad for another. Hard-and-fast rules cannot be

laid down. For some a cold plunge or shower bath after a warm bath is invigorating, while for others it throws them into a mild state of shock from which they do not quickly react. A Turkish bath is often a harmful procedure unless the bather remains for some hours in rooms of gradually diminished temperature. Hyperemia of the superficial vessels is induced, and if the bather goes out into the open air before the circulatory balance is restored, he is likely to "catch cold." The abuse of the vocal apparatus in singing and speaking disturbs the circulatory poise, and by mechanical irritation excites inflammation of the cords and the mucous membrane.

4. **Traumatism.**—Chemical or mechanical injury of the cords or adjacent mucous membrane may cause laryngitis.

5. **Age.**—Laryngitis is more common in young adults.

6. **Climate.**—Laryngitis is more common in the temperate zones, especially during the early spring and late autumn months, as the weather conditions are very changeable.

7. **Idiopathic.**—In some cases the cause is unknown. In such cases it is probable that certain cachexia are present though not well defined. The iodides are usually beneficial in these cases.

Pathology.—The histological changes in acute catarrhal laryngitis are the same as in inflammations of the mucosa of other portions of the upper respiratory tract. The peripheral vessels are congested and the tissues are infiltrated with round cells and leukocytes. If the inflammation runs a short course the infiltration disappears, leaving little or no trace of its occurrence. Should the inflammation be phlegmonous, the tissues become edematous and the surface epithelium eroded in patches. The secretions at first thin and scanty, later become heavier and more profuse. In severe cases they may become purulent and streaked with blood from the superficial follicular ulcers. The pathology of laryngitis secondary to the exanthematous fevers does not differ from ordinary laryngitis except as to the microörganisms causing the disease and the greater tendency to phlegmonous inflammation. The greatest swelling in laryngitis is naturally in the most lax parts, namely, in the ventricles, though the true cords are sometimes red and swollen like sausages. In children the swelling is sometimes below the cords, and is a source of extreme danger.

Symptoms.—**Objective Symptoms.**—The objective symptoms are a change in the appearance of the cords, the mucosa, the secretions, the exudate, and the presence of pathogenic bacteria. With the laryngeal mirror and reflected light an inverted image of the larynx is shown. The mucosa is red and more or less swollen from hyperemia and infiltration, or edema, according to the virulency of the inflammatory process. The cords are pinkish red, or even as red as the mucosa. Sometimes ecchymotic spots of extravasated blood may be seen on their upper surfaces, or free borders. The secretions at first thin and scanty later become thick, semitranslucent, or opaque, according to the amount of lymphocytes thrown out. They have a tendency to accumulate at the anterior commissure and to some extent along the cords. They appear as opaque plugs rather than as thin, diffused, glairy masses.

When follicular ulcers are present, the denuded areas appear as slightly roughened red spots, or, if covered with secretions, as whitish opaque ones. In some cases there is a cloudy swelling of the epithelium in isolated areas. These areas are the beginnings of ulcerations. They appear as slightly elevated patches, with a grayish semitranslucent covering. Hemorrhages may occur at the commissure of the cords, or on the ventricular bands. At first the site of the hemorrhage is red, later almost black. When the inflammation is severe the venous flow may be blocked so that the parts are edematous. This condition is sometimes termed hydrops laryngis. The temperature varies from a slight elevation to one of several degrees, according to the severity of the inflammation and the virulency of the microörganisms contributing to the phenomena. The paralysis or paresis of the intrinsic muscles of the larynx, which sometimes occurs, may be due to a neurosis, though it is more often due to a mechanical interference by infiltration and degeneration of the muscles and the tissues immediately surrounding the nerve endings.

Subjective Symptoms.—The subjective symptoms are changes in voice and respiration, and pain and cough. The voice may be hoarse in any degree, or aphonia may be present. The hoarseness is due to the swelling and infiltration of the cords and adjacent mucous membrane, and to the paresis or paralysis of the muscles. The respiratory effort may be slightly labored, on account of the diminished lumen of the chink of the glottis, or to the paresis or paralysis of the abductor muscles.

In cases complicated by excessive edema, the respiration may be labored because of the edematous swelling. The respiration is shallow because the cough is excited by deep breathing. The character of the cough depends largely upon the individual, though it bears some relationship to the stage and intensity of the disease. In the early stage it is usually soft and husky, whereas later it is more heavy and harsh. In those cases in which there is extensive infiltration and edema it is spasmodic, hoarse, and wheezy, but with little tonal quality. If the inflammation is limited to the interarytenoid space, hoarseness may be absent.

Prognosis.—The prognosis depends somewhat upon the primary cause, that is, whether the laryngitis is due to a chronic constitutional disease, like syphilis, or to a simple exposure which causes temporary lowered resistance of the tissues. If due to the former, the prognosis as to the voice is bad. If to the latter, it is good. If the attack is primary, it is good. If it is one of a series of acute attacks, the chances are in favor of its recurrence, as the etiology is evidently a fixed factor. Again, the prognosis depends largely upon the character of treatment administered. It is obvious that if the cause is a nasal obstruction from septal malformation, the prognosis will depend upon the treatment instituted. If due to nasal disease, and sprays, lozenges, and only medicated nebulæ are used, the prognosis is bad. If the nasal disease is corrected by suitable treatment or an operation, the prognosis is good. Finally, and perhaps of more importance than all other considerations, the prognosis depends upon whether complete rest of the vocal apparatus is observed. If this

is done for from three to ten days, simple catarrhal inflammation will subside, leaving the voice clear.

Treatment.—The successful treatment of the immediate symptoms consists largely in *giving the voice complete rest.* Without this all other methods are usually futile and the inflammation runs its full course. The patient should be confined to his room, the temperature of which should be maintained at from 67° to 70° F. The atmosphere should be surcharged with steam from boiling water to which turpentine and creosote have been added. The bowels should be kept open with calomel and salines. The feet should be placed in a hot mustard bath, after which hot lemonade should be administered. The patient should then be wrapped in a woollen blanket and put to bed. Still further relaxation may be induced by the administration of effervescing tablets of pilocarpine, $\frac{1}{100}$ of a grain. One tablet should be given every hour until three or four are taken. The inhalation of steam impregnated with the compound tincture of benzoin, one teaspoonful to the pint of boiling water, from the spout of a croup kettle, affords relief, and should be used every two to three hours. Kyle recommends the following prescription:

℞.—Acidi nitrici . . . . . ♏iij (0.18)
Tr. opii deodorati . . . . . ♏iij (0.18)
Cocaine phenati . . . . . gr. $\frac{1}{10}$ (0.006)—M.
Sig.—Give every hour until three or four doses are taken.

The application of an ice-bag to the neck exerts a favorable influence in the phlegmonous variety, though it should not be applied longer than a few minutes at a time. A compress of cold water applied over the larynx beneath a flannel bandage also relieves the laryngitis, as it induces hyperemia and leukocytosis just as heat does. It is an open question as to whether the relief is due to the compress *per se* or to the constriction of the bandage, according to Bier's principle. The constriction also increases the local leukocytosis and thus frees the inflamed tissues of the infectious agents and dead tissue cells. Whether the good results are due to the water compress or to the constriction, the effects are favorable. An oily spray of menthol, 1 to 2 grs. to the ounce, is a pleasant application, affording temporary relief. Its frequent use, however, irritates the mucous membrane, hence it should not be used more often than twice a day.

In severe cases in which there is considerable obstruction to the breathing, it may be necessary to puncture the swollen laryngeal mucosa with a laryngeal knife (Fig. 301). The serous fluid in the edematous membrane is thus let out without serious damage to the parts, and in addition the reaction of inflammation is promoted and the bacteria more rapidly destroyed. In extreme cases it may become necessary to intubate or to perform tracheotomy. (See Intubation and Tracheotomy.)

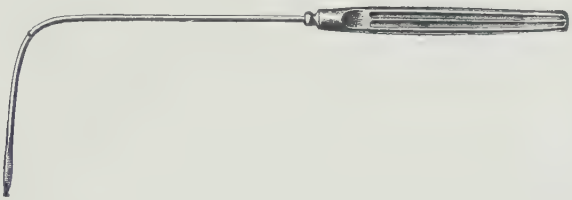
In infants the danger in acute laryngitis is much greater than in adults, on account of the relatively smaller and more easily occluded chink of the glottis. Then, too, the mucosa is much more richly supplied with lymphatics and bloodvessels and is more loosely attached to the

deeper structures. For these reasons the mucosa is more likely to become swollen or edematous and cause suffocation. A fatal issue is possible.

For the relief of the cough, codeine sulphate, gr. $\frac{1}{12}$ to $\frac{1}{8}$, may be administered every three hours until relief is afforded.

After the second week it may be advisable to touch the inflamed cords with the solid stick of nitrate of silver. This should be done but once. In the milder cases the larynx may be painted with a 2 to 4 per cent. solution of the nitrate of silver.

Fig. 301



Laryngeal lancet.

The principles of treatment are: (*a*) Absolute rest of the voice, the patient remaining in a warm room containing steam vapor. (*b*) Free purgation to promote the elimination of the toxins and ferments, and (*c*) relaxation of the peripheral vessels of the body by the administration of pilocarpine and hot drinks. (*d*) Diaphoresis, aided by wrapping in warm blankets. (*e*) The relief of cough by the use of codeine or other sedatives. (*f*) Scarification, intubation, or tracheotomy in threatened suffocation. (*g*) Caustic and astringent applications in the late stage.

ACUTE LARYNGITIS IN CHILDREN

Synonyms.—Pseudocroup; false croup; Miller's asthma; laryngitis stridulosa.

In children, acute laryngitis is often characterized by a spasmodic, croupy, or barking cough and suffocative fits. The subjective symptoms are quite like those of tracheal diphtheria, hence the name pseudocroup. Histologically it is a true catarrhal process.

Etiology.—The etiology of catarrhal laryngitis in children is in general like that of catarrhal laryngitis in adults, though many of the exciting causes may be absent, on account of the different habits of the child or infant. The special etiology in children consists of the presence of adenoids and the epipharyngitis which accompanies them, and in the different anatomical construction of the larynx. In children the chink of the glottis is both relatively and absolutely smaller, the lymphatic and vascular structures are more abundant, and the mucosa is more loosely attached to the underlying tissues. All these factors predispose the larynx of the child to attacks of laryngitis; they also render the disease a

much more serious one on account of the tendency to suffocation. To the foregoing facts should be added the greater susceptibility of children on account of the unstable condition of the nervous system and glandular tissues. A moderate amount of swelling of the mucosa, either above or below the true cords, to which is added an irritation of the terminal motor nerve filaments, is often sufficient to bring on severe and alarming fits of dyspnea and suffocation, even to the point of death.

The disease in children may be divided into two varieties, namely, (*a*) acute supraglottic laryngitis, and (*b*) subglottic laryngitis, or Miller's asthma.

The symptoms of acute supraglottic laryngitis more nearly resemble those of the adult type, though in many cases the spasmodic suffocative fits are present on account of the extreme swelling and edema of the mucosa and the paresis of the abductor muscles.

The subglottic variety is more dangerous because the swollen mucous membrane is confined at its circumference by the cartilaginous rings of the trachea. The swelling must, perforce, encroach upon the lumen of the trachea, and close the breathway.

Symptoms.—The objective symptoms are about the same as in the adult. (See Acute Catarrhal Laryngitis.) The subjective symptoms are somewhat different on account of the greater swelling and the smaller lumen of the chink of the glottis. The prodromal symptoms are those of cold, the respiration becoming embarrassed toward evening. A dry cough develops before bedtime, but is not severe enough to prevent sleep. Toward midnight the child is suddenly seized with a laryngeal spasm, and breathing becomes difficult. The cough is loud and harsh. Inspiration is difficult, and is accompanied by stridor. The child becomes cyanotic, and death is imminent. After a few minutes, the symptoms disappear and the child falls asleep. The following night, and perhaps for two nights, the attack returns with diminishing severity, until after a few days all signs of the disease disappear. In these cases there is a true spasm of the muscles of the larynx, probably due to the natural hypersensitiveness of the nervous system in infants and growing children. In the subglottic variety the swollen mucosa beneath the true cords may be seen through the chink of the glottis as beefy-red bands. These cases closely resemble tracheal diphtheria in their subjective symptoms, though an inspection of the larynx and a microscopic examination of the secretion and exudate will clear the diagnosis.

Diagnosis.—Acute laryngitis in children should be differentiated from diphtheria, pseudomembranous croup, laryngismus stridulus, foreign bodies, and perichondritis.

Diphtheria is characterized objectively by a membranous deposit, which may be seen upon laryngoscopic examination. It may be either on the laryngeal mucosa or in the trachea, or both. Cultures show the diphtheria bacilli. In acute laryngitis there is an absence of the false membrane and the bacilli, while the mucosa is greatly swollen and reddened. If it is of the subglottic variety, the swollen red mucous membrane may appear as round, reddened cords, parallel with and below

the true cords. The temperature is usually higher in acute laryngitis in children than in true diphtheria, while the prostration is not so great.

Pseudomembranous croup has a sudden onset, while acute laryngitis begins with the symptoms of a cold. In pseudomembranous croup the suffocative symptoms make steady progress with little or no remission. The laryngoscopic image in pseudomembranous croup shows the presence of the membrane, whereas in acute laryngitis the mucosa is red and swollen. The Klebs-Loeffler bacilli are absent in both diseases. The systemic disturbance is less marked and not so severe. There are no nocturnal exacerbations, as there are in acute laryngitis with the laryngismus stridulus phenomena superimposed.

Foreign bodies in the larynx are differentiated by the history of the accident, the sudden onset of the suffocative symptoms with no prodromal history, and the image of the foreign body in the larynx.

Perichondritis of the cricoid cartilage is characterized by irregular nodules in this region and the chronicity of the case. It is usually associated with a tuberculous process in the lungs.

Prognosis.—The prognosis of acute laryngitis in children is favorable in most cases, though a fatal termination is possible, especially in the subglottic variety. The disease runs its course in from six to twelve days.

Treatment.—Prophylactic measures should be instituted in those cases in which there is a history of recurrent attacks. A child subject to laryngitis with pulmonary complications, as bronchitis, should have the tone of the system built up by daily cold sponge baths, followed by brisk rubbing with a towel until the skin glows. During the summer he should be kept in the open air as much as possible. At night the room should be well ventilated. The food should be nutritious, easily digested, and liberal in quantity. The clothing should be of linen mesh next to the skin all the year round. In the winter light woollen underwear should be worn over the linen mesh. If there are adenoids or diseased tonsils, they should be removed. If suppurative rhinitis is present, it should receive appropriate treatment. All other ailments should be corrected as early as possible. In short, all disorders should receive attention and a healthful vigor be established as soon as possible. In this way laryngeal inflammation may be prevented.

In the beginning of the acute attack, the bowels should be moved by the administration of broken doses of calomel, followed by a saline cathartic. During the acute stage the child should be confined in a room kept at a temperature of about 70°, and the atmosphere surcharged with steam. The feet should be placed in hot mustard water for fifteen minutes, after which the patient should be wrapped in a woollen blanket and put to bed, to promote diaphoresis. If there is much mucus in the throat and trachea, an emetic should be administered. If the secretions are scanty or tenacious, the inhalation of menthol vapor from a nebulizer, or from the crystals in boiling water, stimulates the secretions and gives marked relief.

The external application of an ice-bag or a cold compress to the neck

often affords relief. The ice-bag should be covered with woolen cloth and left in position for only a few minutes at a time. Counterirritation to the neck with iodine, camphorated oil, kerosene, etc., is used to relieve the swelling when it is great, and to promote the reaction of inflammation. (See Chapter VII.)

In the *later stage*, paregoric, Dover's powder, codeine, etc., may be administered in small doses to relieve the cough. If the secretion is heavy and accumulates in the larynx and trachea, an emetic should be given to clear it away.

Surgical interference may be necessary when the symptoms become alarming. If, upon laryngoscopic examination, the mucous membrane above the cords is found to be greatly swollen, it should be punctured with a laryngeal lancet (Fig. 301). Or if the cyanosis is marked and does not yield to other methods of treatment, intubation or tracheotomy should be performed to save the child's life. (See Intubation and Tracheotomy.) These extreme measures are rarely necessary, but it is well to recognize that in children this disease is sometimes attended with death unless the breathing is maintained by medicinal, hygienic, or surgical interference.

ACUTE PHLEGMONOUS LARYNGITIS

Definition.—Acute phlegmonous laryngitis is a catarrhal inflammation of the laryngeal mucosa, to which is added an edematous effusion which runs an inflammatory course, for example, serous, seropurulent, and purulent stages. The mucous membrane becomes undermined with purulent secretion.

Etiology.—The causes of this variety of laryngitis are about the same as in acute catarrhal laryngitis, except that the infection is more virulent. The disease is common among hospital attendants, on account of their exposure to erysipelas and other infectious diseases. It is rarely primary, but is usually secondary to some other infectious disease. It occurs most frequently between the twentieth and the fortieth years of life.

Pathology.—The pathology is the same as in inflammatory edema of mucous membranes elsewhere in the body. The mucous and submucous tissue are infiltrated with round cells, and there is an effusion of serum and pus corpuscles. On account of the loose texture of the mucous membrane in the aryepiglottic region, the ventricular bands, and the subglottic region, there is great swelling and respiratory obstruction, as in acute laryngitis of children. There is at first a vascular engorgement, followed by a serous effusion. Later, the effusion takes on a seropurulent and finally a purulent character. General sepsis may follow, and prove to be a serious complication.

Symptoms.—The symptoms during the first twenty-four hours are about the same as in the acute catarrhal variety. A chill and elevation of temperature are often the initial ones. The symptoms gradually grow worse, and dyspnea often occurs within the first twenty-four hours. Pain and soreness are usually complained of. Cough may or may not be present.

Objectively, the laryngoscopic mirror shows the mucous membrane to be red, tense, and glassy, with three rounded, swollen masses above the chink of the glottis. If the subglottic region is involved, the swollen membrane may be seen projecting from below the true cords.

Prognosis.—The prognosis is grave on account of the rapid development and the septic infection. If, however, the dyspnea persists longer than thirty-six hours without severe sepsis or other untoward complication, the case will probably end in spontaneous resolution. The cases should be closely watched during the first thirty-six hours.

Treatment.—The treatment consists in local depletion with ice-bags, followed by the use of leeches and scarification. The ice-bag should be applied for forty minutes, after which three or four leeches, two on either side, should be applied to the skin over the larynx. The cold reduces the swelling and thus establishes a more rapid flow of blood through the inflamed tissues, and the leeches bring about an increased leukocytosis. The cellular resistance is increased by the greater amount of blood flowing through the tissues. The various reactions produced by the cold and leeches establish ideal conditions for the destruction of the infectious micröorganisms. The administration of calomel and salines promote the elimination of the toxins. The atmosphere of the room should be kept surcharged with steam. If scarification is resorted to, the laryngeal lancet (Fig. 301) should be used by the aid of the laryngeal mirror and reflected light, or by direct laryngoscopy. The swollen mucous membrane should be repeatedly punctured rather than scarified, as the damage to the parts is less and the relief is equally great. The chief benefit of scarification is in the increased leukocytosis excited by it. It may be necessary to resort to tracheotomy if suffocation becomes imminent. If sepsis is a severe complication, the administration of alcoholic beverages and strychnine are indicated to support the system.

MEMBRANOUS LARYNGITIS

Synonyms.—Croup; croupous laryngitis; häutige bräune; diphtheritic laryngitis; pseudomembranous croup; idiopathic membranous croup.

Definition.—Membranous laryngitis is characterized by an inflammation of the larynx, attended with the formation of a false membrane of non-diphtheritic origin. Opinions differ as to the unity or duality of this disease and true diphtheria. The evidence, however, seems to show that they are two diseases, the latter being due to an infection from the Klebs-Loeffler bacillus, while the former (croup) is due to an infection from other microörganisms, usually the cocci, or to a caustic irritant. When due to the latter, the membrane is not of microbic origin, though it may become infected secondarily. Under the microscope it presents the same appearance as that due to cocci.

Etiology.—The causes of membranous laryngitis are microbic, chemical, and mechanical irritants. Exposure to damp and cold and neuroses are predisposing causes in young children. The cases of microbic origin usually follow or attend scarlet fever, measles, smallpox, etc. Exposure

to damp and cold seems to precipitate attacks by lowering the vital resistance, and thus establishing a suitable soil for the bacterial growth. It appears that chemical and mechanical irritants cause the membranous formation without bacterial influence, although this is not certain. Some children seem to have a predisposition to a membranous inflammation of the larynx, though in these cases I suspect that adenoids and epipharyngitis may cause the susceptibility. It is essentially a disease of young childhood, occurring chiefly between the ages of two and eight. It is most prevalent in the winter season.

Pathology.—The membrane is in two layers, a superficial or epithelial, and a deeper or fibrous layer. It is comparatively loosely attached to the mucous membrane, whereas in diphtheria it is firmly attached. The epithelial layer of the mucosa is rapidly proliferated, and enters into the composition of the pseudomembrane. The mucous membrane is hyperemic and red, and in places is denuded of its epithelium. The bacteria causing the inflammation are chiefly cocci, for example, pneumococcus, streptococcus, and staphylococcus, though other bacteria, as the spirillum and the Bacillus pyocyaneus, are found and probably contribute to the etiology. The membrane is not grayish white, as it usually is in diphtheria, but is yellowish and of a soft, friable consistency. It is more easily removed, and does not leave an ulcerated or bleeding surface, as in diphtheria.

Symptoms.—The laryngoscope shows a free fauces, a coated tongue, and hyperemia of the fauces and the larynx. The membranous formation appears on the aryepiglottic folds, on the ventricles, and occasionally on the vocal cords. It is usually primary in the larynx, though it may originate in the fauces and pharynx, and spread to the larynx. The laryngoscopic image, therefore, shows a yellowish, friable membrane in one or more of these regions. The temperature rapidly rises to 102° or 103°.

The *onset* of the disease may be the same as in acute catarrhal laryngitis, but in the course of an hour or two a loud, brassy cough develops, which steadily increases until toward midnight, when it reaches its climax. There is loss of appetite, and the patient complains of thirst. The pulse is full and the skin is hot and dry. Deglutition becomes painful. The cough, at first infrequent, becomes more and more frequent, and is finally followed by laryngeal spasm. Great dyspnea then comes on, and the child, in his endeavors to cough out the obstructing membrane, clutches at his throat and tosses about in his bed. These symptoms increase in severity as the membrane is formed in the larynx, until the voice is aphonic (silent croup) and the inspiration through the narrowed glottis gives rise to a peculiar crowing sound. The next morning the symptoms are lessened in severity, only to be increased again in the evening. Sometimes the climax is delayed until the third night. The disease is progressive, whereas in laryngitis the obstructive symptoms are spasmodic and are not steadily progressive. In case of marked glottic obstruction the inspiratory and expiratory dyspnea and asphyxia may necessitate intubation or tracheotomy.

If the dyspnea continues, the pulse becomes weak, the temperature falls, and the general strength rapidly ebbs away on account of the diminished oxygenation of the blood and the increased amount of carbon dioxide in the blood. When the membrane is thick in the region of the soft palate there may be a regurgitation of fluid food through the nose. This is not due to paresis of the palatal muscles, as in true diphtheria, but is due to the mechanical interference of the false membrane with the action of the muscles.

Laryngismus stridulus sometimes appears in the course of the disease, and is to be regarded as a neurotic phenomenon.

Diagnosis.—Membranous croup resembles in some respects spasmodic laryngitis, diphtheria, laryngismus stridulus, and retropharyngeal abscess.

In *spasmodic laryngitis* there is a catarrhal inflammation with spasms of the laryngeal muscles, which cause suffocative symptoms. They disappear, however, in a few minutes and the child rests comfortably. In membranous croup the suffocative symptoms come on gradually and disappear as gradually.

In *diphtheria* the temperature does not rise so high or so rapidly. The chief diagnostic points, however, are the culture of the Klebs-Loeffler bacilli and the ashen-gray and firmly adherent pseudomembrane. After its removal the mucous membrane is ulcerated and bleeding, whereas in membranous croup it is smooth and does not bleed.

Laryngismus stridulus is a neurosis and not an inflammatory disease, hence the laryngoscopic examination shows the absence of inflammation. Then, too, there is a history of a healthy child who suddenly has a fit of suffocation. In membranous croup there is a history of inflammation and progressive dyspnea.

Retropharyngeal abscess may simulate membranous laryngitis in its suffocative symptoms; otherwise there is little similarity. An examination of the throat reveals a fluctuating tumor on the posterior wall of the hypopharynx, whereas in membranous laryngitis the tumefaction is within the laryngeal zone.

Prognosis.—The prognosis is grave. Some author's report a mortality of from 50 to 60 per cent. of the cases, while other report as low as 10 per cent. This discrepancy in the reported death rate is probably due to the difference in the diagnosis. Those who figure the death rate at 50 to 60 per cent. probably include cases of true diphtheria. The prognosis is grave in inverse ratio to the age of the patients. The younger the patient the more serious the prognosis. In adults the danger is greatly diminished as the lumen of the larynx is relatively and actually greater, and the mucous membrane is more firmly attached.

Complications.—Membranous laryngitis may become complicated with rapid edema of the bronchial mucous membrane or with cardiac infection. In either event the case becomes one of great gravity.

Treatment.—The treatment consists in the administration of broken doses of calomel until free catharsis is produced, and in the inhalation of steam vapor charged with lime and turpentine. The child should be

put into a tent-bed and a pound of lime should be placed in a bucket of water, to which has been added a tablespoonful of the spirit of turpentine. The tent-bed is thus filled with the vapor, which is inhaled by the child. The lime and turpentine seem to aid in loosening and expelling the false membrane. The steam-tent seances should last about fifteen minutes, and should be repeated every four or five hours. The efficiency of the steam-tent baths is increased by the administration of ipecacuanha wine or powder, which is a non-depressing emetic.

Calomel fumigations, as advocated by Corlin, have proved an efficient method of treatment. He recommends the administration of one or two grains of calomel before the fumigation begins. The patient should then be placed in a completely closed tent-bed. It requires about ten minutes to volatilize the calomel, and the patient should be exposed to the fumes in the closed tent for about fifteen minutes. It is recommended that fifteen grains be volatilized every two hours for two days and nights, after which the intervals should be prolonged to three hours on the third day, four hours on the fourth day, and three times daily thereafter as long as indicated. Pure calomel thus used does not produce ptyalism, though anemia may occur and should be combated by the administration of iron.

EDEMA OF THE LARYNX

Synonym.—Edema glottidis.

Edema of the larynx is an inflammatory process attended with an edematous infiltration of the loose submucous tissue of the larynx, which is usually due to a more serious general disease of the heart, kidneys, or the liver, though it may be caused by local conditions.

Etiology.—The local causes are mainly traumatic from the injudicious use of caustics, laryngeal injections of creosote in tuberculous inflammations, operations, foreign bodies in the supraglottic region of the larynx, the swallowing of hot liquids and the inhalation of hot steam, or the inspiration of alcoholic or other irritating liquids into the larynx. The prolonged or violent use of the voice, as in shouting, may bring on edema of the larynx. Local diseases of the larynx, as tuberculosis, syphilis, abscesses, neoplasms, perichondritis, and peritonsillitis, may also cause it. Abscess of the larynx may be accompanied by a non-inflammatory edema.

The constitutional causes of simple edema of the larynx are Bright's disease, diabetes, valvular lesions of the heart, sclerosis of the liver, and Ludwig's angina. In the latter disease there is a neurotic paresis of the bloodvessels of the neck, which causes engorgement and edema. Certain drugs, as the iodide of potassium and the fumes of ammonia and bromine, may cause it.

Pathology.—There is an effusion of clear serum into the laryngeal submucous tissue, producing swelling of the aryepiglottic folds and of the anterior and superior parts of the epiglottis. Sometimes the loose subglottic tissue becomes edematous. In associated ulcerative processes the serous infiltration may become seropurulent.

Symptoms.—The onset is sudden and is characterized by the loss of the voice and rapidly increasing dyspnea. In severe cases a fatal issue may occur in from two to three hours by asphyxiation. There is little or no pain or cough. The laryngoscopic image shows the mucosa in the region of the aryepiglottic folds, the anterior and upper surface of the epiglottis, and sometimes the subglottic region to be tumefied. The surface of the mucous membrane is of a pale gray color, in marked contrast to the tumefaction in phlegmonous or inflammatory edema of the larynx, in which it is red.

Prognosis.—The prognosis is grave on account of the sudden development of the edema, and the serious nature of the constitutional disease back of it. If it is due to an extraneous irritation, the danger is less, and the chance of recurrence is less.

Treatment.—If the disease is secondary to a serious constitutional disorder, this should, of course, receive appropriate treatment. For the immediate relief of the symptoms, cracked ice should be dissolved in the mouth, and the patient should be assured by the attending physician that the dyspnea will disappear, as the sense of impending death only aggravates the distress. Astringent applications of cocaine and adrenalin should be made. Diaphoresis and catharsis should be induced by the administration of Dover's powder, hot lemonade, etc., followed by a twelve-ounce bottle of the citrate of magnesia. In addition to the above, it may be necessary to puncture the edematous tissue with the laryngeal lancet (Fig. 301). If suffocation is imminent, the patient should be tracheotomized (see Tracheotomy), to prevent a fatal issue. The surgeon should not hesitate to perform tracheotomy on a deeply cyanotic case because he does not have with him the instruments usually used for this purpose. A pocket knife, or a paring knife from the kitchen, may be quickly sterilized and used to open the trachea. A needle and thread may be used to retract the parts until a tracheotomy tube is secured. In the meantime, the patient's life has been saved, whereas to have waited for suitable instruments would have jeopardized his life.

ABSCESS OF THE LARYNX

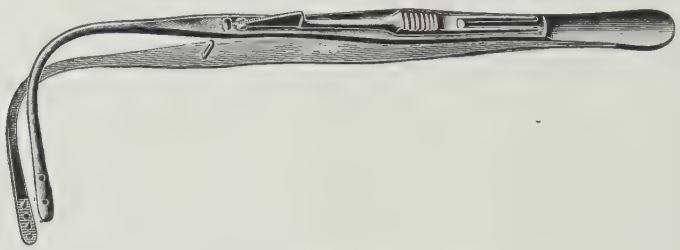
Etiology.—Abscess of the larynx is usually a complication of tuberculous perichondritis. Perichondritis of the laryngeal cartilages is attended with ulceration of the mucous membrane. Infectious bacteria gain entrance beneath the perichondrium and cause the formation of pus. The accumulated pus causes a rounded tumor-like mass. This is a laryngeal abscess. It has also been known to follow erysipelas of the larynx, and it may be of traumatic origin.

Symptoms.—The abscess swelling encroaches upon the glottis, hence there are loss of voice and intense suffocative symptoms. It is an infectious inflammatory process, and causes fibrile phenomena. There is retention and pressure, hence pain in the larynx. The laryngoscopic image shows a greatly swollen and reddened mucous membrane at the

site of the abscess. Upon puncturing it with the laryngeal lancet there is a free flow of pus.

Treatment.—It is obvious that there is but one method of treatment, namely, the evacuation of the pus with a laryngeal lancet (Fig. 301). This may be done under cocaine anesthesia with the patient in the sitting

Fig. 302



Sajous' laryngeal forceps applicator.

posture. The anesthesia is induced with a 10 to 20 per cent. solution of cocaine applied repeatedly with Sajous' forceps (Fig. 302). The curved laryngeal lancet should then be used with the aid of reflected light and the laryngoscopic mirror, or by direct laryngoscopy and the tumor-like mass freely incised. The relief is immediate. If suffocation threatens, tracheotomy may be necessary. (See Tracheotomy.)

CHRONIC LARYNGITIS

Definition.—Chronic inflammation of the mucous membrane of the larynx includes the glandular, vascular, and connective-tissue layers. It is usually secondary to acute attacks, or to inflammation in the nose, epipharynx, and tonsils, though it occasionally seems to occur as a primary affection.

The following classification meets both the clinical and the pathological requirements:

1. Chronic hypertrophic laryngitis.
   - (*a*) Diffused hypertrophic laryngitis, sometimes called chronic hyperemic laryngitis.
   - (*b*) Discrete or localized hypertrophy of the mucous membrane, either supraglottic or subglottic.
   - (*c*) Chorditis nodosa, or trachoma of the vocal cords.
2. Atrophic laryngitis.
3. Hemorrhagic laryngitis.

Chronic Hypertrophic Laryngitis.—(*a*) **Chronic Diffused Laryngitis.**—Each of the three varieties of chronic hypertrophic laryngitis presents a distinct clinical and pathological picture, hence they will be described separately.

Synonym.—It is sometimes called hyperemic laryngitis.

It is characterized by a diffused infiltration throughout the laryngeal

mucosa, no one part being affected more than another. As it is due to irritations of a general character, rather than to those directed to one part, it is easy to understand the diffusion of the hypertrophy and hyperemia.

Etiology.—It is extremely doubtful if there is a *primary* chronic laryngitis, except from the improper use of the voice. It is always, or nearly always, secondary to a preceding disease of the nose, epipharynx, or the faucial tonsils. It is possible to conceive of a chronic laryngitis following the excessive use of tobacco or alcohol, or even following digestive disturbances. Clinically, however, it is rare to see cases in which there is not an associated or a preceding disease higher up in the respiratory tract. The diffused hypertrophic variety arises from obstructed nasal breathing and from the discharge of secretion from the sinuses into the pharynx. Other sources of irritation may also be present, but they are generally incidental and of secondary importance.

The etiology may be classified under the following headings:

1. Improper preparation of the inspired air on account of disease of the nose and sinuses.
2. Hematogenous irritation of the larynx in mouth breathing, hepatic and digestive disorders.
3. Passive hyperemia in cardiac disease, thoracic tumors, and enlarged glands.
4. Smoking, inhalation of dust-laden air, excessive use of alcohol, and violent use of the voice.
5. Climatic conditions.
6. Age and sex.

Mouth breathing, adenoids, deflections of the septum, turbinal hypertrophy, sinuitis, and polypi, also improper breathing by public speakers and singers, lead to a diffused irritation of the laryngeal mucous membrane. As the improperly prepared air and secretions pass over the whole laryngeal mucosa, there is a diffused hypertrophy. As the air in damp cold weather is more irritating than it is in warm and bright weather, it follows that the symptoms are aggravated during the winter and early spring months in the higher latitudes. This is especially true in the region of the Great Lakes and on the northern Atlantic coast of the United States.

The breathing of improperly prepared air results in deficient oxygenation of the tissues and an excess of carbon dioxide in the blood. This in turn disturbs the metabolic processes, and still further loads the blood with deleterious material. This blood in circulating through the laryngeal mucosa irritates all its parts, and causes a diffused hyperemia and hypertrophy. The excessive use of alcohol and tobacco similarly affects the larynx. Smoking does it by direct irritation, and indirectly through the blood. The ingestion of alcohol affects the larynx by direct irritation of neighboring parts, and through the circulation, to say nothing of the digestive and metabolic disturbances thus aroused. The foregoing etiological factors predispose the larynx to acute attacks, and the chronic state is usually a sequel or a continuation of repeated acute inflammations.

I am of the opinion that through disease and obstruction in the nose the laryngeal mucosa is kept in a state of irritability, and is made susceptible to chronic inflammation by the inspiration of the improperly prepared air and by the toxins in the blood. At the age of puberty, boys are subject to attacks of chronic laryngitis on account of the unstable condition of the vasomotor nervous system, the rapid development of the larynx, and the consequent instability of the same. Any disease of the heart, wherein there is an interference with the return circulation, may cause huskiness of the voice and perhaps diffused hypertrophy of the mucous membrane. Thoracic tumors, or enlarged thoracic and cervical glands, also interfere with the return circulation, and lead to hypertrophic changes. Stonecutters, tobacconists, metalworkers, and workers with certain chemicals are often affected by chronic laryngitis from the inhalation of the contaminated air. Men are more often affected than women, for obvious reasons. The aged are more subject to it on account of the vascular and glandular changes accompanying senility. Indeed, many old people living in the northern part of the United States are more or less afflicted with chronic laryngitis.

Pathology. —There is a diffused hypertrophy of the laryngeal mucous membrane, including the glandular and the connective tissue. The bloodvessels are but little affected excepting a few small arteries on the surface of the epiglottis and the vocal cords, where they may be enlarged.

Symptoms.—The objective symptoms of diffused hypertrophic laryngitis, if carefully studied, are somewhat different from those of the other two varieties of hypertrophic laryngitis, and are as follows:

Diffused hyperemia of the laryngeal mucous membrane, including that of the epiglottis, is usually present. It may be more marked in the ventricular pouches, on the epiglottis, the aryepiglottic folds, or on the vocal and the ventricular bands. Indeed, it often spreads from one part to another in the order given above, until in the later stages it is general. In singers and speakers the hyperemia is generally greater in, or is entirely limited to, the true cords. The color varies in different individuals, and, indeed, in the same case at different times. The cords may be the normal ivory white, or pinkish red, or they may be streaked with red, or they may be of a pale, mottled brown or slaty gray color. Enlarged bloodvessels are rarely seen, except upon the epiglottis and the vocal cords.

The secretions are increased but little, indeed, in some cases they are apparently decreased. The image may present, therefore, either a moist or a dry membrane. The hyperemia is rarely demonstrable by laryngoscopic examination. The mobility of the cords is usually unaffected, though in some cases there is a tardy action from the infiltration of the intrinsic muscles.

The subjective symptoms have reference to the voice, the sense of accumulated secretions, and the ease with which the vocal apparatus becomes tired. The voice upon rising is often quite husky, or even aphonic. During the day it becomes nearly or entirely clear, unless it is

used excessively. In this event it remains husky, and its use is attended with aching in the larynx.

The diffused hyperemia and hypertrophy give rise to the sense of accumulated secretions and the desire to clear the throat.

Diagnosis.—The diagnosis is based upon the hoarseness or aphonia, the diffused hyperemia in the later stage, the absence of discrete hypertrophy, and the small amount of expectoration, except when complicated by bronchitis.

Prognosis.—The prognosis in the early stage is good, but when the hyperemia has extended over the entire mucosa, it is not so favorable. If the laryngitis is due to the excessive use of alcohol or tobacco, or to an excessive or violent use of the voice, the excesses should be corrected. If it is due to nasal obstruction or to adenoids these conditions should be corrected. No matter what the cause, the prognosis as to the voice is bad if the hypertrophy is great. In these cases there may be an infiltration of the thyro-arytenoidei interni muscles, thus giving rise to a sluggish action of the cords.

Treatment.—From the foregoing description of the disease it is apparent that the treatment must be addressed to (*a*) the correction of the preexisting disease of the nose and sinuses; (*b*) the removal of adenoids; (*c*) the discontinuance of the use of tobacco and alcohol; (*d*) the correction of digestive and hepatic disorders; and (*e*) the avoidance of excessive use of the vocal organs.

When the nose and accessory sinuses are the seat of a catarrhal or a suppurative inflammation, they should receive appropriate attention. Deflections of the septum, turbinal hypertrophies, sinuitis, polypi, etc., should be corrected or removed by surgical procedures. Adenoids, if present, even though they are somewhat reduced by atrophy in adults, should be removed, and the associated epipharyngitis treated with silver applications. The faucial tonsils when enlarged or diseased should be removed in their entirety. The use of tobacco and alcoholic beverages should be forbidden, as but little benefit can be expected while the larynx is subjected to their deleterious effects. Singers who practise improper placement of the voice should either be forbidden to sing, or be taught proper methods of voice placement. (See the Singing Voice.) Violent use of the voice, either in singing or speaking, should be avoided.

The use of sprays, gargles, and oily nebulæ by the patient are of little value. These remedies, at most, can do no more than thin the secretions and thus facilitate their expulsion.

Local applications of a 2 to 10 per cent. solution of the nitrate of silver with Sajous' forceps should be made three times a week. The chloride of zinc in the same strength should be tried, although I have found nothing as efficacious as the nitrate of silver. Other preparations of silver in my hands have proved disappointing. In making applications to the larynx the excess of fluid should be squeezed from the cotton to prevent it trickling between the cords, where it excites spasm of the laryngeal muscles. Should a spasm occur, instruct the patient to take a number of deep breaths in rapid succession. Sustained efforts of this

sort quickly stop the spasms. Spasms of the larynx excited by an excess of silver solution may be so violent as to cause cyanosis and extreme apprehension on the part of the patient.

Constitutional remedies, as saline cathartics, calomel, and the iodide of potash, should be given if syphilis is suspected. They are often of value in small doses when syphilis is not present, as the cathartics improve the elimination, while the iodide of potash stimulates the glands.

The improvement following the correction of digestive and hepatic disorders is often very gratifying. To this end I advise the daily use of one of the bitter salines in small doses, and a five grain dose of the iodide of potash three times a day. In addition to these remedies, it may be necessary to use others, according to the needs of the case. If chronic bronchitis is present, the administration of a ferruginous tonic, with five grains of the iodide of potash three times daily, for from three to six months, will often effect a cure of both the laryngitis and the bronchitis. One of my patients gained twenty pounds in five months under this treatment.

The hygienic conditions should be good, the living and the sleeping rooms ventilated, and proper clothing worn. Even with all these precautions it is often impossible to greatly improve the quality of the voice.

(*b*) **Discrete or Localized Hypertrophic Laryngitis.**—*Synonyms.*—Chronic subjective laryngitis; laryngitis hypoglottica; chorditis vocalis hypertrophica inferior; Stoerk's blennorrhea.

Discrete or localized hypertrophic laryngitis is characterized by hoarseness or aphonia, dyspnea, a brassy cough, and an infiltration of the tissues in the subglottic space.

Etiology and Pathology.—The pathological changes are the same as those given under the diffuse form, except that they are more localized.

Symptoms.—The subjective symptoms are about the same as those given under the diffuse form, but are greatly exaggerated. The hoarseness usually amounts to aphonia. The hypertrophic tissue in the subglottic space and the infiltration of the laryngeal muscles, interfere with the normal movements of the cords to such an extent that approximation is often impossible. The dyspnea, or suffocative symptoms, are due to obstruction below the glottis. The brassy cough is characteristic of obstructive swelling and hypertrophy in the subglottic region.

The objective signs of this variety of laryngitis are quite characteristic. The hypertrophied tissue below the cords appears in the form of two sausage-like masses, nearly parallel with and beneath the true cords. Their color varies from a pale grayish pink to the deep red of active inflammation. The epiglottis is also congested, and enlarged blood-vessels pass over its posterior surface. In some cases there is more or less edema. In these cases deglutition is difficult, owing to the imperfect closure of the glottis. The dyspnea in discrete hypertrophic laryngitis is increased upon exertion. Patients sometimes complain of a sense of stuffiness, or of a foreign body in the larynx. After the disease is well

advanced, the above symptoms are fairly persistent, as the hypertrophic swelling is a fixed factor. Upon attempted phonation the cords fail to approximate, and instead of the free edges presenting straight lines they are slightly concave or wavy, owing to the weakness of the abductor and tensor muscles and infiltration. No doubt the hypertrophic masses in the subglottic region also interfere with the movements of the cords. The secretions are thick and whitish in color and are often accumulated in the interarytenoid space, and over the sluggishly moving cords.

Diagnosis.—Rhinoscleroma presents some points of similarity, but in view of the fact that it is a very rare disease in this country, and that if the subglottic swelling is touched, under cocaine anesthesia, with a probe, it is yielding, whereas in rhinoscleroma it is hard and resistant, there is little difficulty in excluding rhinoscleroma. The removal of a piece of the growth for microscopic examination may be practised in case of doubt. This, when stained by Gram's method (see Rhinoscleroma), shows the characteristic cell formation, and the bacillus of rhinoscleroma if that disease is present.

Prognosis.—On account of the hypertrophic swellings below the cords, the dyspnea may become so great as to require the performance of tracheotomy (see Tracheotomy) and the wearing of a tube throughout the remainder of life. The danger from suffocation and the pulmonary complications incident to the wearing of the tracheal tube render it a grave disease.

Treatment.—Before undertaking the treatment, the cause or causes of the affection should be carefully studied. When the etiology has been definitely determined, an endeavor should be made to overcome the predisposing causes of the disease. If rheumatism, gout, dyspepsia, anemia, or constipation (Watson Williams) are present, appropriate remedies should be given. The iodide of potash and the protoiodide of mercury should be given whether or not syphilis is suspected, as they often promote more or less absorption of the deposit. Tonic remedies, as iron, arsenic, quinine, gentian, and strychnine, should be given to promote the general tone of the system and to innervate the laryngeal muscles. Obstructive lesions and inflammatory diseases of the nasal chambers and of the epipharynx should be remedied by appropriate medicinal and surgical measures. If the excessive use of tobacco and alcohol enter into the etiology, their use should be interdicted. The local application of astringents, as the chloride of zinc (10 to 30 grains to the ounce), nitrate of silver (10 to 30 grains to the ounce), alum (5 to 15 grains to the ounce), should be made with Sajous' laryngeal forceps or with an atomizer during phonation. A change of climate or a sea voyage is sometimes beneficial, though not curative. Last, but not of least importance, is the absolute rest of the vocal organs. Great improvement sometimes results when these precautions are faithfully observed for a few days.

(*c*) **Chorditis Nodosa.**—*Synonyms.*—Trachoma of the vocal cords; chorditis tuberosa; singers' nodules.

Chorditis nodosa is characterized by the formation of nodules along the free border of one or both of the vocal cords. Some authors claim that they are more often near the junction of the middle and posterior thirds of the cords, though others have observed them at the junction of the anterior and middle thirds. In my cases they have been in the former position.

Etiology.—The nodules usually complicate chronic hypertrophic laryngitis in singers and public speakers who use faulty methods of respiration and voice placement (Curtis). Curtis insists that his patients practice lower costal respiration with the upper ribs elevated, and that they practise voice placement by attacking the initial tone with the lips gently closed, as in humming, so that when they are plucked with the finger the tone flows therefrom. If the tone does not emit through the lips when plucked, but comes through the nasal chambers only, it is an evidence of faulty voice placement. When such is the case there is an overtension of the intrinsic and extrinsic muscles of the larynx. This causes attrition of the cords at the tips of the arytenoid processes, hence the nodules at this position. Singers' nodules of this type may be likened to corns due to ill-fitting shoes. Chiari claims that chorditis nodosa is a typical pachydermia laryngis. Hajek thinks the nodules are glandular hypertrophies. The term as herein used refers to nodules from improper voice placement.

Pathology.—The nodules consist of layers of stratified squamous epithelium surrounded by a circle of congested tissue.

Symptoms.—As the nodes accompany a diffused hypertrophic laryngitis, the symptoms are sometimes similar to those described under that condition. The special subjective symptoms are that the singer or the public speaker is unable to strike the tone he desires, especially in the middle register. When the cords are widely separated, as in the lower register, no difficulty is experienced, as the opposing nodes do not touch. When the higher register is attempted, the posterior thirds of the cords are necessarily closely approximated and not in use, and the voice is not greatly affected. When, however, the middle register is attempted, the cords vibrate their entire length, and as the nodes touch they interfere with voice production. Hence, a prominent symptom is the difficulty in tone placement experienced by singers in attempting to use the voice in the middle register. The laryngoscopic image shows a nodule on the free border of one or both cords, usually at the junction of the posterior and the middle thirds, though the nodules may occasionally form anywhere along their borders. If both cords are involved, the nodules are exactly opposite. A small area of hyperemia is often present at the base of the nodule. If diffused hypertrophic changes are present, they may not be apparent except as shown by the hyperemia.

Prognosis.—The prognosis in regard to the disappearance of the nodules is good, provided the patient faithfully follows the instructions contained in the chapter on the Singing Voice, or practises external massage of the larynx, as recommended by Miller.

Treatment.—The treatment consists in refraining from singing and loud speaking, and in practising proper methods of breathing and tone placement. This should be done under an intelligent and appreciative instructor, which, alas! is hard to find. I have treated a few cases of "singer's nodules," according to Curtis' suggestions, with most excellent results. In none of the cases did I resort to either local, medicinal, or surgical treatment, as the nodules were apparently the result of faulty methods of singing.

If advisable, the astringent remedies described under discrete hypertrophic laryngitis may be used. In extreme cases, it may be necessary to remove the nodules with an intralaryngeal cutting forceps introduced by the direct or indirect method. This should be done only after failure to cure by the other methods suggested. Miller recommends external massage of the larynx with a mechanical vibrator as an adjunct to proper training in tone building and voice placement. The massage improves the circulation and nutrition of the mucous membrane, increases the local migration of leukocytes, and relieves the associated laryngeal inflammation.

Atrophic Laryngitis.—**Synonym.**—Laryngitis sicca.

Atrophic laryngitis is characterized by a burning or pricking sensation after exercising the voice and by suffocative attacks (simulating spasmodic croup and asthma) during the night.

Etiology.—The atrophic changes in the larynx are usually secondary to the same process in the nose and pharynx. Bosworth believes that some influence is brought to bear upon the mucous glands of the laryngeal mucous membrane, which deprives them of their secretory power, and that this influence is often independent of intranasal or pharyngeal atrophy. According to my observation, atrophic laryngitis is often secondary to ethmoiditis and sphenoiditis, and I usually address therapeutic measures to these cavities as well as to the larynx.

Pathology.—The mucous membrane undergoes a retrograde change, and fibrous tissue finally replaces the normal elements constituting the mucous membrane and submucous tissue. The mucous glands and the bloodvessels disappear, or become greatly diminished in size. The ciliated columnar epithelium is gradually replaced by squamous epithelium. The secretions are diminished in quantity and changed in quality. They are thicker and admixed with white corpuscles and epithelial debris. The desiccated secretion appears as brownish, blackish, or grayish crusts on the cords, and in the interarytenoid space. Ulceration of the mucosa is not generally present, though it may be, especially on the posterior wall.

Symptoms.—After using the voice, there may be a burning or pricking sensation in the throat. Cough of a hoarse, spasmodic character is excited by the presence of, and the attempt to remove, the crusts from the larynx. The cough and hoarseness are more severe in the morning. Dyspnea, simulating spasmodic croup or asthma, may occur at night on account of the accumulation of the crusts over the vocal cords. Upon laryngoscopic examination the mucous membrane appears pale and dry, with discolored crusts on the cords, or in the interarytenoid space.

They may also be seen upon the posterior wall of the larynx in some cases, especially if there is ulceration in this region. The cords are dry and wrinkled and more or less covered with crusts. The trachea may be dry and glazed or covered with crusts.

Prognosis.—The prognosis is bad except in those cases in which the atrophic changes have progressed but little. In such cases the surgical exenteration of the ethmoid and sphenoid sinuses may effect a cure or an amelioration of the disease, provided, of course, the sinuses are affected.

Treatment.—The internal administration of the iodides occasionally stimulates glandular activity, and thus affords relief. Pilocarpine may also be given for the same purpose if the heart is strong. It should never be given unless an examination of this organ has first been made. The chloride of ammonium and cubebs stimulate the glands and thin the secretions, rendering them easier to dislodge. The inhalation of aromatics in solution in olive oil, thrown into the larynx with a nebulizer, is grateful and affords temporary relief. Medicated lozenges with a mucilaginous base may be used to protect the dry membrane. A warm, moist climate or a sea voyage will ameliorate the symptoms. Careful attention should be given to the condition of the nose, the accessory sinuses, and the pharynx. If the nose is kept free from crusts and the secretions are increased the larynx will undergo a corresponding improvement. In empyema of the posterior ethmoidal and the sphenoidal cells, the secretions discharge into the pharynx and trickle downward into the larynx, where they become dried and adherent to its posterior wall, or lodge upon the cords. In such cases great improvement follows the radical operative treatment of the sinuses.

Hemorrhagic Laryngitis.—**Synonyms.**—Spurious hemoptysis; laryngeal hemorrhage; bleeding in the throat; spitting blood.

By hemorrhagic laryngitis is meant a laryngeal inflammation accompanied by hemorrhage from the laryngeal mucous membrane. The spitting of blood, or hemoptysis, is not always of laryngeal origin. It may come from the nose, the pharynx, the trachea, the bronchi, or the lungs. The term hemoptysis, or spitting of blood, should be limited to hemorrhage from the lungs, and especially that which occurs in tuberculosis.

Etiology.—Hemorrhage which occurs in the course of laryngitis is due to ulcerations, acute inflammations, and to excessive use of the voice. Syphilis and tuberculosis of the larynx may be attended with laryngeal hemorrhage. Albuminuria, diabetes, variola, typhoid fever, yellow fever, leukemia, hemophilia, and malignant disease also predispose to hemorrhages.

Symptoms.—If chronic laryngitis is present, the usual symptoms of such a condition are also present. (See Chronic Laryngitis.) The patient also complains of a tickling sensation in the throat, followed by cough and the expectoration of blood. The quantity varies from a mere streak to a mouthful; usually, however, it is small.

The laryngoscopic examination shows one or more areas of extravasated blood in the cords or mucous membrane, and some fresh fluid blood may still cling to the surface of the laryngeal mucosa.

Treatment.—Ordinarily no treatment is required. Astringent sprays and the external application of ice may be tried. If the cough continues it should be quieted by the administration of morphine by hypodermic injection (Coakley). The act of coughing prevents coagulation and tends to prolong the bleeding.

General Diagnosis of Chronic Laryngitis.—The differential diagnosis of chronic laryngitis from other laryngeal disease is not always easily made. It may be confounded with laryngeal tuberculosis, syphilis, adenitis, carcinoma, and certain benign growths.

Tuberculosis is characterized by a rapid pulse, elevation of temperature, loss of appetite, emaciation, and a general lowered vitality. These symptoms are not present in chronic laryngitis. An examination of sputum for tubercle bacilli will still further aid in the diagnosis. A laryngoscopic examination does not always settle the diagnosis, unless the larynx is the seat of the tuberculous infiltration. In most cases of tuberculosis the laryngeal mucosa is ashen gray in contrast with the diffused hyperemia of chronic laryngitis. In the inflammatory type of laryngeal tuberculosis (mixed infection), the mucosa is red, but the swelling of the arytenoid cartilages is too great to be mistaken for catarrhal inflammation.

If the tuberculous process is well advanced, ulcerations may be present.

Syphilitic affections of the larynx may present much the same appearance as the edematous type of chronic laryngitis. Hyperplasia may be present in both diseases, but is more often present in syphilis. Careful inspection will often reveal small ulcers, which may lead to a mistaken diagnosis of syphilis. An accurate history of the case is, therefore, necessary in making the differential diagnosis. In the tertiary stage of syphilis the diagnosis is easily made. The ulcers in hypertrophic laryngitis are stationary, while those of syphilis and tuberculosis are deep and spread rapidly.

Carcinoma in the subglottic region is distinguished from discrete hypertrophic laryngitis by the nodular outline of the growth and the cachexia. Perichondritis in this region more nearly simulates carcinoma on account of the nodular outline of the tumor-like mass.

In *lupus* the surface of the membrane is markedly red and granular.

Sarcoma of the larynx presents a red and an uneven contour, whereas in all forms of hypertrophy the swelling and purulent discharge come before the perichondritis is well developed.

Enchondrosis of the laryngeal cartilages is differentiated from edematous laryngitis by the sense of hardness on probe pressure and the uneven contour of the swelling.

Paralysis of the posterior crico-arytenoid muscle may be mistaken for subglottic hypertrophy unless a careful examination is made. In paralysis the lagging movements of the cords reveal the nature of the lesion. The paralysis may also be mistaken for pachydermia laryngis.

Prolapse of the ventricles is differentiated from superior hypertrophy by marked pitting upon probe pressure.

Angina laryngis is differentiated from hemorrhagic laryngitis by the elevated whorl of bloodvessels and the absence of hemorrhage.

Papilloma is distinguished from chorditis nodosa by the point of attachment and the size and shape of the growth.

DIPHTHERIA; TRACHEOTOMY; INTUBATION

Definition.—Diphtheria is an acute infectious disease, characterized by the presence of the Klebs-Loeffler bacillus. It is still further characterized by a false membrane on a mucous surface or an abraded skin, and is communicable, either directly or indirectly, from one person to another. The lesion is usually located in the upper respiratory tract.

Etiology.—As to its geographical and racial distribution, it may be said to be well-nigh universal. No climate, season, country, or race is exempt from its ravages. It is, however, less prevalent in the summer season in temperate and northern latitudes, on account of the open-door life of the people at this season, and because, during the school vacations, the overcrowding and the close contact incident to school life are temporarily suspended. Statistics show that among the poor in crowded tenements, and in badly ventilated schoolrooms, the disease is more prevalent. A curious exception to this is shown by Walsh to exist among the negroes of Washington. The percentage of diphtheria among 10,000 negroes was 4.43 as against 15.25 per cent. among the same number of whites. This may be due to an antitoxic state of the blood in the negro race, or to a greater freedom from disease of the upper respiratory tract. (Nasal obstruction is comparatively rare among negroes.)

Sanitation is an important factor in the development of the disease. Sunshine and fresh air are twin sisters of charity in the prevention and the amelioration of infectious diseases. In one of the great children's hospitals of London, diphtheria was prevalent in one of the wards. As soon as they were convalescent, the patients were removed to another ward and no recurrences were reported. An adjacent building was torn down and the solid iron shutters of the convalescent ward were closed to exclude the dust. Incidentally the sunshine and the fresh air were also excluded, and there were many recurrences among the convalescents.

The overcrowded tenement districts in the great cities are usually poorly ventilated and the rooms little exposed to the sunshine. When many are in close contact, the opportunities for transmitting the infection are multiplied; hence, for these and other reasons, the poor of the cities are especially afflicted with diphtheria.

Defective plumbing, sewer gas, cesspools, etc., are thought to produce the disease. While these may indirectly influence the spread of the contagion, it should be remembered that the Klebs-Loeffler bacillus is absolutely essential to the production of the true disease. The presence of sewer gas may produce lessened resistance to the diphtheria bacilli, and thus predispose the patient to their ravages.

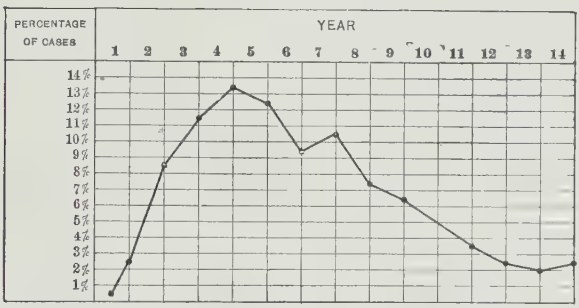
Bodily conditions have much to do with the susceptibility of the

individual exposed to the Klebs-Loeffler bacillus. The "scrofulous habit" lowers the tone of the cellular elements of the body, and thus renders it less fit to cope with the inroads of the disease-producing germ. Abraded or diseased surfaces in the upper respiratory tract also offer local areas of lowered resistance to the growth of the bacilli. Hence, enlarged and diseased tonsils, adenoids, glandular enlargements of the neck, and catarrhal diseases of the nose and throat favor the development of the diphtheritic process.

Rich and poor alike are affected, the only difference being the more favorable sanitary conditions surrounding the rich, who are, therefore, relatively less often affected.

Age has a great influence on the prevalence of the disease. The blood of nurslings is very antitoxic in its properties, hence children under one year of age are comparatively exempt from the disease. After the fourteenth year there is relatively slight predisposition to diphtheria. Babinski shows by the statistics of 2711 diphtheritic cases that under

FIG. 303



The above chart is arranged from the statistical data of Babinski, and shows at a glance the relative prevalence of diphtheria from birth to fourteen years of age.

six months the percentage of cases is 0.55; six months to one year, 2.5 per cent.; one to two years, 8.3 per cent.; two to three years, 11.6 per cent.; three to four years, 13.05 per cent.; four to five years, 12.4 per cent.; five to six years, 9.7 per cent.; six to seven years, 10.3 per cent.; seven to eight years, 7.7 per cent.; eight to nine years, 6.4 per cent.; nine to ten years, 5.5 per cent.; ten to eleven years, 3.7 per cent.; eleven to twelve years, 2.9 per cent.; twelve to thirteen years, 2.02 per cent.; thirteen to fourteen years, 2.6 per cent. (Fig. 303).

Modes of Infection, Direct and Indirect.—The direct infection is from the one affected to another, *i. e.*, by breathing the atmosphere immediately surrounding the patient, inhaling his breath, or receiving the mucus or the saliva into the mouth or the nose during an act of coughing, spitting, or sneezing on the part of the patient. Kissing is another mode of direct infection, and is to be condemned when diphtheria is known to exist in the family. All members of the family should refrain from this manifestation of affection during the term of diphtheritic infection, as there may

be a mild or an incipient infection without the knowledge of the individual. Without doubt the disease is often transmitted by persons who are not suspected of being infected.

The indirect mode of infection is not so easily traced as the direct; nevertheless, it is well established that the bacilli may be transmitted by domestic animals, as dogs, cats, chickens, rabbits, etc., which, being directly exposed to the contagion, convey it to persons removed from the direct source of infection. The author recalls a case which aptly illustrates this point. He was in the house of a minister when a member of the parish called to make the funeral arrangements for his child, who had just died of diphtheria. The man was accompanied by a collie, which was hugged and fondled by the four-year-old son of the minister. Within a few days the boy was ill with diphtheria, having no doubt received the infection from the collie.

It may also be conveyed by towels, table-linen and dishes, bedding, books, wall-paper, carpets, rugs, clothing, and all other articles bathed in the germ-laden atmosphere surrounding a diphtheritic patient. Food, especially milk, may be the source of infection.

The hands and the clothing of physicians, nurses, and parents should be mentioned as sources of infection.

The custom of serving the elements at communion services in churches from common cups is to be condemned as a possible mode of conveying contagious diseases. Individual cups should be used, thereby minimizing if not absolutely removing the danger. The church should be as cleanly in its table manners as its individual members are in their homes. There they do not think of drinking from a common vessel, each member and each guest being provided with one for his individual use. The same decent, cleanly, sanitary custom should prevail in ecclesiastical functions.

Diphtheria may be endemic, epidemic, or sporadic in its manifestations in a community. The mode of manifestation is largely due to the density and the numerical strength of the settlement. In large cities, where large numbers are congregated in small areas, diphtheria is epidemic, coming as a tidal wave of infection and carrying many away in its course. The community may then be free from the disease for months or years. The sporadic or isolated cases are more difficult to explain, but we know that the Klebs-Loeffler bacilli must be present. They may live under varying and peculiar conditions for a long time. The sporadic cases are often caused by the germs, which suddenly become virulent and give rise to the isolated attacks of the disease.

Bacteriology.—The Klebs-Loeffler bacillus being the specific cause of diphtheria, its characteristics and the method for its detection are important. The announcement of Klebs in 1883 that he had discovered a bacillus which was constantly present in the false membrane of diphtheritic patients, marked an epoch in the history of medicine, and soon revolutionized the methods of treating diphtheria. Loeffler in 1884 made pure cultures of the bacilli, and inoculated the mucous membranes of animals, getting the characteristic pseudomembrane of diphtheria. In 1888–89, Roux and Yersin reported the results of their experiments

relative to the toxins produced by this germ. Serumtherapy thus had its beginning.

The Klebs-Loeffler bacilli vary greatly in size, shape, and curvature, according to the medium in which they are grown, and often vary in the same medium. They also vary with the fluidity, the age, and the temperature of the medium, but they generally present the appearance of narrow rods, straight or curved, swollen at either extremity, and are found in groups with a tendency to parallelism. They are not always parallel, but may have a tangled, irregular arrangement, or be in broken chains.

The atypical forms may be thickened at one end only, or at the centre of the rod, the extremities being pointed. They may also be lance-, spindle-, or club-shaped, or even pear-shaped. One characteristic is always present, namely, segmentation.

The Klebs-Loeffler bacilli stain readily with alkaline methylene blue and many other aniline dyes.

Northrup gives the following directions for the preparation of Neisser's stain and its application to the differentiation of the diphtheritic germ:

"No. 1.—1 gm. methylene blue dissolved, 20 c.c. of 96 per cent. alcohol, 90 c.c. distilled water, 50 c.c. glacial acetic acid.

"No. 2.—2 gm. vesuvin to 1 liter of boiling distilled water.

"The culture is stained in No. 1 for one to three seconds, or even somewhat longer; washed off in water and stained with No. 2 for three to five seconds or longer; washed off and mounted. Colored in this way, a twenty-four-hour-old culture on blood serum or bouillon will show the body of the bacilli stained brownish yellow, while at one or both ends may be frequently seen the so-called polar granules (Neisser-Ernst bodies) as deeply colored blue, oval-shaped areas, the diameter of which is greater than that of the bacillus in which they are found. The outlines of these bodies are sharply defined, and they are not peculiar to true diphtheria bacilli, but are found occasionally in a slightly atypical form in certain forms of pseudodiphtheria bacilli, especially in older cultures."

The diphtheria bacilli may be grown upon blood serum, agar-agar, bouillon, milk, etc., and they are pathogenic for pigeons, rabbits, guinea-pigs, chickens, certain small birds, cattle, goats, and horses.

Bacteriological Diagnosis.—A portion of the pseudomembrane should be removed from the throat of the patient with an aseptic cotton-wound probe, wire loop, or other instrument, and smeared over a clean coverglass, dried and stained with Roux's double stain of dahlia violet and methyl green or with Loeffler's blue-staining solution.

The coverglass thus prepared should be mounted and examined with a microscope. The diphtheritic bacilli, if present, will be readily recognized by their typical appearance. If not found, a culture in blood serum should be made, which, in from twelve to twenty-four hours, in a temperature of 37° C., will develop grayish colonies, the size of a pinhead, with regular outline, the surface being dry. Held to the light, the periphery is translucent, the centre being somewhat opaque, on account of its greater thickness.

Upon the above appearances and reactions a fairly positive diagnosis of diphtheria may be made.

The development of the *streptococcus* is much slower (twenty-four to seventy-six hours), the colonies are white, and pinpoint in size.

The development of the *staphylococcus* is slower than that of the diphtheritic bacillus, but faster than that of the streptococcus. It presents the appearance of a flocculent or white colony much larger than a pinhead, and has a halo-like border. The areas are darker in the centre.

A negative result with the microscopic examination, or with the cultures, does not justify a positive statement that the case is not one of true diphtheria. The author knows of an instance in which seven different examinations were made by an expert bacteriologist and pathologist, before the Klebs-Loeffler bacilli were found.

Mixed infection generally occurs, hence a case of simple diphtheria is not commonly seen in practice. The Klebs-Loeffler bacilli are usually associated with streptococci, staphylococci, and diplococci, and the symptoms and the progress of the disease are modified accordingly. Again, virulent diphtheria bacilli may be present in a healthy throat without giving rise to any symptoms. Should, however, these same bacilli be lodged in a throat with enlarged, ragged tonsils, there is every probability that the person would be affected by true diphtheria. Mixed infections are more serious than simple ones, as the accessory germs may produce severe pathological changes, independent of the diphtheritic process.

The Systematic Distribution of the Bacilli.—Many investigators report the presence of Klebs-Loeffler bacilli in pneumonic areas and lymphatic glands, but they are generally associated with other germs. They have been found in the lungs, the spleen, the bone-marrow, the liver, the nasal accessory sinuses, the heart's blood, and they are probably in other tissues of the body.

Pseudodiphtheria Bacilli.—There are two schools of thought regarding the so-called pseudobacilli of diphtheria: (*a*) The larger school holds that the pseudodiphtheria bacillus is under no circumstances convertible into the true diphtheria bacillus. (*b*) The smaller school holds that the two germs are identical. The scope of this work will not permit of a presentation of the data upon which these two schools of thought rest their claims. Suffice it to say that the two germs are differentiated, according to the first or larger school, by their mode of development on various culture media, their morphology, and their pathogenicity.

Histopathology.—The distribution of the false membrane may involve the mucous membrane of the nose, pharynx, tonsils, hard and soft palate, mouth and lips, larynx, trachea, the bronchi from the largest to the smallest, the ear, and abraded surfaces of the skin. The vagina, the duodenum, the conjunctivæ, and other mucous membranes may also be involved.

In about 75 per cent. of the cases the membrane is above the larynx. In 15 per cent. of the cases the larynx is involved. Previous to the use of antitoxin, autopsies often showed the pseudomembrane extending

from the tip of the nose to the smallest bronchi; since the use of antitoxin it is rarely found so extensively distributed.

The *appearance of the pseudomembrane* varies from a grayish white through a dirty brown to a black color (in hemorrhagic diphtheria). Its consistency is usually tough and leathery, although it may be friable. It is *firmly attached* to the underlying tissues when found on the uvula or the pharyngeal wall, and *loosely* attached in the trachea.

The *formation of the pseudomembrane* begins with an exudation of lymphatic cells, which rapidly undergo coagulative necrosis, leaving a reticulated substance composed of fibrin from the broken-down cells.

If the *fibrin penetrates* the deeper layers of the mucosa, it is difficult to remove it, as the line of demarcation is not easily established between the living and the dead tissue. If, on the other hand, the fibrin remains superficially attached, it is easily removed, for obvious reasons. When the pseudomembrane is deeply attached, its removal is attended by some bleeding; if superficially attached, there is no bleeding.

Sloughing of the mucous membrane may occur when the bloodvessels supplying it become degenerated, thrombosed, or otherwise injured, so that the nutrition supplied to the parts is shut off. This is often spoken of as "gangrenous diphtheria."

It is seen by the foregoing statement of the varying appearances and conditions of the pseudomembrane of diphtheria that the picture presented is kaleidoscopic in character. Its appearance in the early stage is usually as a whitish or yellowish, circumscribed film, and, at a still later period, it may become yellowish or dirty brown in color. If hemorrhage takes place beneath or within the false membrane, it may become black.

According to Northrup, the pathological changes in various parts of the body have been shown by numerous writers, and only a brief mention of them can be made here.

The *nervous system* is involved in some cases with degeneration of the posterior roots (Bikeles and Kalisko), where they enter the gray matter of the posterior cornua, thus accounting for the ataxic symptoms which occur in diphtheritic paralysis. Manicatide reports his findings as follows:

(*a*) Purely muscular changes with no nerve involvement.

(*b*) Polyneuritis.

(*c*) Lesions of the spinal cord, which were either localized in the gray matter, leading to atrophy of muscles, or involving the white matter of the cord, in a similar way to that seen in locomotor ataxia or multiple sclerosis.

(*d*) Cerebral paralysis, chiefly due to circulatory changes.

The *heart* undergoes degeneration, chiefly fatty. This simple type of degeneration precedes the more destructive hyaline changes, which lead to the loss of the sarcous elements. The changes are due to toxins.

The *lungs* are, in about 60 per cent. of cases, affected by bronchopneumonia. True lobar pneumonia has not been found.

The *spleen* is affected by cell infiltration in the splenic follicles. In the centres of the follicles, masses of epithelial cells are sometimes found. There is local edema of the centre or the periphery of the follicles. Necrotic areas and hyaline changes are also present.

The *lymphatic glands* first undergo congestion and hemorrhage, and there is dilatation of the lymphatic sinuses. Later, foci very similar to miliary tubercles form, by a process of proliferation, phagocytosis, and degeneration. These changes are due to the toxins formed by the lymphatics and not to bacteria. The same changes, with minor modifications, take place in the tonsils.

The *thymus gland* undergoes the same changes as described under lymphatic glands.

The *skeletal muscles* undergo fatty degeneration.

The *bone marrow* undergoes hyperplastic changes.

The *pancreas* has not been found involved in autopsies following true diphtheria. Hibbard and Morrissy found *glycosuria* in 25 per cent. of 230 patients. Others have failed to find it so commonly present. *Examinations for sugar should be made in every case of diphtheria.*

The *alimentary canal* may be affected by true diphtheria of the stomach. The pseudomembrane has not been found in the intestine.

The *liver* undergoes degenerative changes, ranging from simple fatty to hyaline degeneration. Focal necrosis is the most characteristic change in this organ in diphtheria.

The *kidneys* undergo fatty and hyaline degeneration. Casts are present. There are also interstitial changes in about 25 per cent. of cases examined. There is an increase in the cells of the glomeruli, and sometimes necrosis with hemorrhage into the capsular space is present.

Types of Diphtheria.—Before considering the symptomatology, it will be well to consider briefly the various types of diphtheritic manifestations. It is often described, according to the seat of local manifestation, as angina, local or general; nasal diphtheria; bronchial diphtheria; broncholaryngeal (ascending) diphtheria; conjunctival diphtheria; aural diphtheria; vaginal and rectal diphtheria, etc.

Monti's classification, according to Northrup, in Nothnagel's *Encyclopedia of Practical Medicine,* is as follows:

Catarrhal Diphtheria (Bacteriological Diphtheria; Diphtheria Fruste).—This type is characterized by simple redness and swelling of the tonsils and the pharynx, with no false membrane. Microscopic examination shows the Klebs-Loeffler bacilli present. Spontaneous recovery occurs in a few days. The germs, transplanted into another throat, might give rise to a more severe type. Careful quarantine should be maintained to prevent the spread of the disease.

Fibrinous Diphtheria.—This type is due to the action of the Klebs-Loeffler bacilli uncomplicated by any other germ. It may be purely local in its character, the membrane and the slight redness surrounding it being the only symptoms; or it may be general, with a tendency for the false membrane to spread to other parts, with great toxemia and

severe complications. It is more often local in its manifestations. Microscopic findings: the Klebs-Loeffler bacilli.

Mixed, Phlegmonous, or Streptodiphtheria.—This type is characterized by great inflammatory reaction in the neighborhood of the pseudomembrane, and by the presence of the Klebs-Loeffler bacilli with some other pathogenic organism, usually the streptococcus, and their toxins. Mixed infections are more dangerous, and experiments on animals (Roux and Martin) show that antitoxin has little or no effect in checking the ravages of this type of infection.

Septic or Gangrenous Diphtheria (Septicemia).—In dealing with this type, we are essentially treating septicemia of diphtheritic or of mixed infectious origin. It is usually of mixed infection (Klebs-Loeffler, streptococci, and staphylococci) origin, although in rarer cases it seems to originate from the simple Klebs-Loeffler bacillus infection, which has assumed the so-called gangrenous diphtheria type. In other words, what started out as a simple diphtheria later became complicated by other germs and their toxins, a true septicemia resulting. It is doubtful if true septicemia ever results from pure Klebs-Loeffler bacillus infection.

General Symptomatology.—The disease is ushered in by a feeling of discomfort, lassitude, loss of appetite, constipation, slight sore throat, difficulty in swallowing, and more or less hoarseness.

The *temperature* varies with the type, but has certain characteristics which may be recognized. For instance, even in the fibrinous type, which is the least febrile, there is a rise of temperature with the beginning of the formation of the membrane. It is commonly said that this type is not attended with fever. Notwithstanding, it will be found that there will be a recurrence of elevated temperature with each extension of the pseudomembrane to a new part. In all types of diphtheria there is an increase of temperature with each extension of the local field of infection. There is a greater fluctuation of the temperature curve in the mixed infection and the septic type than there is in the catarrhal and the fibrinous varieties.

The *pulse* rate is invariably increased in uncomplicated cases in the beginning, in proportion to the toxic products eliminated. The pulse rate in infants is especially high.

Brachycardia (slowing of the pulse rate), if persistent, is a grave symptom.

Tachycardia (increased pulse rate), when reaching a rate of 140 or more, is a grave symptom. At 140 the death rate is about 20 per cent., increasing to 90 per cent. at a pulse rate of 180. Nasal diphtheria is usually the cause of the tachycardia, hence the occurrence of a rapid pulse should at once lead to a critical examination of the nasal fossæ. The nose is very richly supplied with lymphatic tissue, hence the rapid absorption and the toxic symptoms.

Reduced blood pressure, as shown by sphygmographic tracings, indicates an increased absorption of diphtheria toxins, and warrants a grave prognosis. The same is true of an intermittent pulse.

Partial angina is the most common anatomical form of the disease. Early there is a general redness of the pharynx and the pillars of the fauces. At the site of pseudomembrane formation, which is usually the tonsil, there is increased redness. It may form, however, on the posterior pillars, the uvula, or the walls of the pharynx. First one tonsil is involved, then the other. The cervical glands are somewhat swollen and tender. The temperature is elevated 1° to 2°, with frequent oscillations. The general health is good. There is transient albuminuria. The course is from six to eight days.

General or *toxic angina* is characterized by a thicker and more extensive pseudomembrane, gray or dirty yellow in color, or even brown or black. The whole, or nearly the whole, of the tonsils, the pillars (arch), the uvula, and the pharynx are covered by the membrane in from three to six days. Grave symptoms appear early, and are usually ushered in by a chill followed by fever. Delirium, restlessness, apathy, and vomiting are often present. Swallowing becomes difficult on account of the swollen and stiffened condition of the fauces and the pharynx. The epipharynx (nasopharynx) is filled with tenacious mucus. The cervical glands are swollen and tender. Albuminuria is severe. Without treatment, the pseudomembrane may be cast off and be reformed, continuing thus for three to six weeks. Under proper treatment the disease may be brought under control in from three to six days.

Phlegmonous or *streptodiphtheritic angina* involves the entire throat from the beginning. The mucous membrane is dark red, and the uvula swollen. Within a few hours a dirty gray or blackish membrane forms, and rapidly spreads. The cervical glands are much swollen and very tender. While the membrane is forming and spreading, the temperature is elevated. Toxic symptoms, as rapid pulse, delirium, restlessness, apathy, etc., set in after the membrane has reached its limit. The temperature usually drops at this time. Albuminuria often appears within forty-eight hours. Under antitoxin treatment the disease may be controlled in from five to six days. In obstinate cases the kidneys and the heart may become involved and thus complicate the case.

Septic angina is characteristic of certain epidemics, although it usually develops from the phlegmonous variety. The symptoms are most grave from the beginning. Vomiting is violent and attended with extreme prostration. The temperature curve rises very suddenly. The pulse is small, soft, and rapid. Respiration is increased proportionately. The tonsils and the fauces are swollen. They are a livid bluish white, with discolored spots. Bloody matter is mixed with the exudate. The cervical glands are very much swollen and tender on both sides. Death occurs usually on the second to the fourth day, from collapse and general sepsis.

Diphtheria of the nose may assume any one of the foregoing types, although it is probably more often of the simple fibrinous type. It may be primary or secondary. The upper lip is excoriated by the nasal discharge. The child "snuffles," sleeps a great deal, and takes food poorly on account of the nasal occlusion, and he may become cyanotic

in attempting to nurse the breast. The glands of the neck are swollen. Nasal hemorrhages occasionally take place. Many cases run a benign course, while others are malignant from the beginning, death occurring within a few days. In older children the disease runs a more favorable course. In scrofulous children it may be more chronic, often extending over many weeks.

The nasal occlusion is at first thought by the parent to be due to a foreign body in the nose. The membrane is usually situated on the septum, although it frequently involves the whole Schneiderian membrane, and may be removed with the forceps or the syringe, as a cast of the interior of the nose.

In *phlegmonous, mixed,* or *streptodiphtheria of the nose,* the symptoms are more severe from the beginning, the membrane is mixed with blood and appears black (black diphtheria). Toxic symptoms are marked, and the glands of the neck much swollen and tender. The patients are little inclined to take food. Early and vigorous treatment is often followed by recovery. The disease is, however, to be regarded as very grave in its nature. On account of the rich lymphatic supply of the nose, the septic form of nasal diphtheria is especially serious.

Laryngeal Diphtheria (True Croup; Membranous Croup; Diphtheritic Croup, etc.).—Laryngeal diphtheria may be primary, although it is usually secondary to diphtheria of the nose, the pharynx and tonsils, the trachea and the bronchi. On account of the great danger, and at the same time a possibility of a favorable issue under proper treatment, we will, according to Northrup, enter into a brief but careful analysis of this type of diphtheria. It should be studied under three headings, namely: (1) Stage of invasion; (2) stage of spasm—exudation; (3) stage of asphyxia.

Stage of Invasion.—This is characterized by a simple angina becoming suddenly complicated with hoarseness, and a cough characteristic of laryngeal irritation. The Klebs-Loeffler bacillus may or may not be found. A negative finding is not conclusive, however, as heretofore stated.

Stage of Spasm (Exudation).—The pseudomembrane may develop so rapidly that within twenty-four hours there is laryngeal stenosis. The cough is dry, short, and hoarse, becoming paroxysmal in character and often lasting for several minutes. It is attended with cyanosis, full veins, and a perspiring forehead. Aphonia, more or less complete, soon develops. The respiration is wheezing and noisy. As the stenosis becomes more advanced, the inspiratory act is prolonged and is attended with a whistling noise. There is pronounced depression of the supraclavicular region, the neck, and the epigastrium. The severe symptoms come in waves; extreme cyanosis, and harsh, difficult respiration, which gives way, temporarily, thus affording the sufferer a brief respite from the aggravated symptoms. The natural duration of the stage is from one-half to seven days.

Stage of Asphyxia.—This stage is characterized by greatly impeded respiration and toxic symptoms. The respiration becomes more rapid and irregular, the child sits up suddenly and falls back again exhausted.

The cyanosis and the retraction of the supraclavicular, the jugular, and the epigastric regions are more pronounced. The suffocative attacks occur more frequently. The head is thrown back, and all the accessory muscles of respiration are called into action. Even the abdominal muscles are retracted. The larynx rises with each respiratory effort. During one of the suffocative attacks, complicated with convulsions, death comes. According to Monti, in untreated cases the death rate is from 95 per cent. to 98 per cent. Under modern methods of treatment, the death rate is small in cases taken early.

Phlegmonous or Mixed Infection of the Larynx.—It is usually secondary to a similar process in the nose or the throat, and is characterized by great redness of the mucosa of the larynx and the trachea, with some grayish pseudomembrane scattered here and there in the larynx and the trachea. The stenosis of the larynx is not so marked as in the preceding type, nevertheless, death may occur suddenly from it. The toxic symptoms are also marked in this type, and no doubt contribute toward a fatal result.

Septic Diphtheria of the Larynx.—This is also secondary to a similar process in the nose or the throat, or both, and begins with fever, apathy, and marked weakness. The mucous membrane of the larynx and the nose is swollen, and covered with a grayish-yellow exudate. Toxic symptoms, as vomiting, delirium, suppression of urine, heavily coated tongue, rapid pulse, etc., are marked. The prognosis is quite grave.

Causes of Asphyxia in Diphtheria.—Four theories have been advanced: (*a*) Spasm of the glottis; (*b*) obstruction by pseudomembrane; (*c*) paralysis of the dilators of the glottis; (*d*) excitation of the respiratory centres by carbonic acid poisoning and reflex action of the pneumogastric nerve.

Autopsies have shown many instances of death from asphyxia when there was little or no false membrane to account for it. This leaves spasm of the glottis, paralysis of the dilators, and the irritation from carbonic acid as possible theoretical explanations. The latter two have but few supporters; hence, the probable explanation of the majority of cases is to be found in the first theory, namely, spasm of the muscles of the larynx.

Diphtheria of the Trachea and the Bronchi.—This is usually secondary to laryngeal diphtheria, although it may occur primarily in the bronchi or the trachea. Where it thus forms, and the larynx is secondarily involved, it is known as "ascending croup." If a cast of the bronchi is coughed up, it is a positive sign of bronchial involvement. Other signs, as respirations (50 to 60 per minute), continuous dyspnea (as contrasted with intermittent when the pseudomembrane is in the larynx and upper trachea), supraclavicular and epigastric depressions not so well marked, pale face, blue lips, and great physical depression, may aid in reaching a diagnosis of bronchial diphtheria. The prognosis is very grave.

Diphtheria of the Ear.—This is usually carried to the external ear by scratching (abrasion) with the infected fingers of the patient. Infection of the external auditory meatus is seen in rare instances in which

there is diphtheritic otitis media with extension through the tympanic membrane.

Otitis media as a complication of diphtheria, occurs in only about 4 to 6 per cent. of the cases. When present it is characterized by deafness and pain in the ear upon swallowing and coughing; these are followed by aural discharge, after which the pain subsides.

Diagnosis.—The differential diagnosis of diphtheria should be made between (*a*) peritonsillar abscess; (*b*) follicular tonsillitis; (*c*) pseudo-diphtheria; (*d*) pseudocroup; and (*e*) catarrhal rhinitis; the chief diagnostic point in each case are the microscopic and the culture findings.

Prognosis.—This may be summarized under the following headings:

(*a*) **The Age of the Patient.**—The mortality is the lowest in the first year and the tenth year, and the highest in the second to the sixth year of life.

(*b*) **The Site of the Local Lesion.**—Involvement of the larynx results in the highest mortality. Nasal diphtheria in infants is very fatal.

Treatment.—The administration of antitoxin has reduced the cases coming to operation one-half. The death rate in laryngeal cases under antitoxin has been reduced from 70 per cent. to 16 per cent. Intubation is attended with a lower rate of mortality than tracheotomy.

Time of Beginning Treatment.—Briggs and Guerard have compiled the following table:

	Cases.	Deaths.	Mortality. Per cent.
First day of disease	1415	5	3.5
Second day of disease	2640	213	8.0
Third day of disease	2340	300	12.8
Fourth day of disease	1458	346	23.6
Fifth day of disease	1912	671	35.0

It will be seen by the foregoing table that early treatment influences the prognosis very favorably.

Complications and Sequelæ of Diphtheria.—Adenopathy.—Swelling of the lymphatic glands in the region of the local diphtheritic lesion usually occurs. The cervical glands and the tonsils are accordingly most commonly affected. After these come the bronchial, the intestinal, and the mesenteric glands.

In the *pure diphtheria, i. e.*, the simple fibrinous type, the glands are swollen, slightly tender, and freely movable in the surrounding tissue.

In the *mixed forms* of infection there is greater swelling and tenderness, the glands being lost to the touch in the surrounding swollen and infiltrated tissue. In some cases the swelling is enormous, constituting the symptoms known as "le con proconsulair." Suppuration occurs only occasionally, and then only in the mixed type. In the *septic type*, gangrenous sloughing may occur. Treatment often results in recovery from even severe diphtheritic adenopathy.

Gastro-intestinal.—Vomiting, loss of appetite, diarrhea, and diphtheria of the esophagus and the stomach sometimes occur.

Urine.—The urine is variable in quantity and chemical proportions. Albuminuria is present in about one-half of all cases of diphtheria

and in nearly all cases of the toxic varieties. It is generally due to degenerative changes in the kidneys. Hyaline, granular, and epithelial casts may be found.

According to Simon, in diphtheria a well-marked increase of urine is the rule, with the exception of very mild or extremely severe cases, of constant occurrence. It is interesting to note that, barring a temporary diminution immediately after the injection, the leukocytosis is nowise influenced by the antitoxin treatment.

Hyperleukocytosis.—This exists in nearly all cases, and varies according to the toxemia and the sepsis. It may be so severe as to constitute a true leukemia.

Heart Lesions.—Endocarditis, myocarditis, waxy degeneration, nerve degeneration, heart clots, and dilatation have been found in certain cases which were examined post mortem.

Nervous Affections.—Degeneration of nerve tissue, paralysis, lessened functional activity, etc., sometimes attend, but more often follow, an attack of diphtheria.

Postdiphtheritic Paralysis.—Postdiphtheritic paralysis usually affects the velum palati (benign and discrete form) and the pharynx. The chief symptom is difficulty in swallowing and the return of liquids through the nose. Each act of swallowing is accompanied by a laryngeal cough. The voice is nasal, articulation is very much interfered with, and the patient snores during sleep. The paralysis disappears in from one to three weeks.

In the general or diffused postdiphtheritic paralysis, the palatal and the neighboring muscles are involved. The muscles of the eye are most frequently affected. Unequal pupils, diplopia, strabismus, or ptosis may be present. Complete recovery eventually takes place. The patellar reflex is impaired, or lost, and the muscles of the feet may be paralyzed. The patients shuffle their feet on the floor in walking. "Diphtheritic pseudotabes," or even complete paralysis of the lower extremities, may complicate some cases. The muscles of the upper extremities are less often affected. The muscles of the neck and the head are rarely involved. If they are, the child's head falls over on his shoulder. The facial expression may be lost, giving an idiotic cast to the countenance.

Diaphragmatic paralysis occurs in about 7 per cent. of cases, and may lead to a fatal termination. The chief sign of diaphragmatic paralysis is a sinking in of the abdomen during inspiration, and distention during expiration. Respiration is rapid and panting. Bronchitis or other slight lesion of the lower respiratory tubes may lead to asphyxiation and death.

Cardiac or **vagus paralysis** complicates about 1 per cent. of the cases.

Skin.—Erythema, papular eruption, brownish discolorations, and eruptions of the skin, like those of measles and scarlet fever, may complicate the disease.

Bronchopneumonia.—This is a serious complication, and often causes death after tracheotomy and intubation. It is ushered in by a rise of temperature, increased cyanosis (in laryngeal cases), change of the

respiration-pulse ratio from normal 1.4 to 1.3. At first the physical signs are those of diffuse bronchitis, later of consolidation over several areas.

Prophylaxis.—The following rules should be observed in preventing the spread of diphtheria. (Abstracted from the Rules of the Health Department, City of New York.)

1. No one but the attendant and the physician should be permitted to enter the sick chamber.

2. The discharge from the nose and mouth should be received on cloths provided for the purpose, and immersed for two or three hours in a solution composed of six ounces of carbolic acid dissolved in one to two gallons of hot water, and then boiled in soap-suds for one hour. All bed and personal clothing used about the patient should be similarly treated *inside the sick-room.*

3. The hands of the attendant and the physician should be washed in the same carbolic acid solution, and washed in soap-suds after making applications or handling the patient, and before eating.

4. Surfaces soiled by discharges should at once be flooded with carbolic acid solution.

5. Table utensils used by the patient should be *kept in the sick-room*, for his special use, and should be washed in carbolic acid solution and then in hot soap-suds. The vessel containing the soap-suds should then be washed in the carbolic acid solution.

6. The sick-room should be aired two or three times daily, and swept frequently after scattering sawdust, wet tea-leaves, etc., on the floor to prevent the dust from rising. The furniture and the woodwork should be wiped with damp cloths. The sweepings should be burned, and the cloths soaked in the carbolic acid solution.

7. All unnecessary articles of furniture, pictures, draperies, clothing, etc., should be removed from the room as soon as the nature of the malady is recognized.

8. When the patient has recovered, he should receive a hot soap-suds bath, including his hair; clean clothes should be put on, and he should be removed from the sick-room. He should be kept in quarantine as long as cultures of the diphtheria germ can be obtained from his throat.

In addition to the rules given in regard to the patient and the sick-room, the physician and the nurses should protect their clothing by wearing long gowns, which should be kept just outside the patient's room.

9. They should also be given immunizing doses of antitoxin.

10. The room should be scrubbed with bichloride of mercury solution, 1 to 1000, *all over*, the woodwork repainted or varnished, the walls cleaned and repapered, and the furniture sterilized with formaldehyde vapor. In the case of upholstered furniture, disinfection can be more thoroughly done by steam.

11. The periodical inspection of public schools by a corps of physicians will do much toward limiting the spread of the disease.

Immunization by Antitoxin.—An immunizing dose of antitoxin ranges from 100 to 500 units, according to the age of the patient and the length of time immunity is desired. In an average case 100 units will be effective for ten days, while 500 units will be so for twenty-eight days.

Treatment of Diphtheria.—The treatment may be divided into (1) local, (2) general, and (3) measures for the relief of the suffocation.

Local Treatment.—This consists in the use of an antiseptic solution, such as boracic acid, chloride of sodium, etc., at a temperature of 110°, with a fountain syringe. The patient should be wrapped tightly in a sheet fixed with safety pins. He should be placed upon his side and the glass or hard rubber nozzle of the syringe applied to one nostril, the fluid flowing out at the other, until it comes forth clean. The patient's mouth should be held open with a spool or a mouth gag, to prevent swallowing, as this act might force the solution into the middle ear and cause infection and mastoiditis. The pharynx should be treated in a similar manner. If it is desirable to combat pain and swelling, the temperature of the solution should be about 130°. The irrigations may be repeated at intervals of six hours.

General Treatment.—The general treatment of diphtheria consists in the administration of stimulants to overcome the depression, the weak action of the heart, the irregular pulse, and the septic condition. Alcohol in the form of whisky or brandy is the best for this purpose, and should be given to an infant in 10 to 15 drop doses, well diluted with water, three or four times a day. A child of three or four years may be given an ounce in twenty-four hours. In septic cases much more can and should be given. Strychnine is the second best stimulant. Dose, child one year old, $\frac{1}{100}$ grain every two or three hours. Child three to four years old, $\frac{1}{50}$ grain every two or three hours.

Sedatives should be given to relieve restlessness, cough, and spasm (second stage) in laryngeal cases. Morphine may be given in $\frac{1}{20}$ to $\frac{1}{12}$ gr. doses. Emetics may be given to overcome spasms and to remove mucus in laryngeal cases.

Antitoxin in Diphtheria.—The value of antitoxin is shown by a comparison of the following tables:

TABLE I.—By Briggs and Guerard

Treated with Antitoxin

Ages	Cases	Deaths	Per cent.
0-2 years	1494	469	31.4
2-5 years	3678	762	20.7
5-10 years	3184	473	14.8
Over 10 years	1444	99	6.0

TABLE II.—By Babinski

Not treated with Antitoxin

Ages	Per cent.
0-2 years	63.3
2-4 years	52.8
4-6 years	37.9
6-10 years	24.6
10-15 years	14.6

The advantages of the antitoxin over the other methods of treatment at the various ages is strikingly shown by a comparison of the foregoing tables, and needs no further comment.

Antitoxin in laryngeal cases is valuable in two ways, namely: (*a*) It prevents many cases coming to the operative stage, and (*b*) it affects favorably the intubated and tracheotomized cases. Statistics show that it affects the intubated cases more favorably than it does those upon which tracheotomy has been performed.

Antitoxin seems to increase *paralysis* rather than to decrease it. This is perhaps explained by the fact that cases treated with antitoxin live longer, and thus give more time for the paralysis to develop. Many more severe cases survive.

Injections of antitoxin often produce a *transient albuminuria.*

Dosage and Clinical Administration of Antitoxin.—The following dosage is recommended: (*a*) 2000 to 3000 units in ordinary diphtheria to a child over one year old; (*b*) 3000 to 5000 units in severe laryngeal cases of any age; (*c*) 1500 to 2000 units to an ordinary case in a child under one year old.

Repeat the dose in twelve hours, or less, if the symptoms are increasing, and in eighteen to twenty-four hours if there is no decided improvement.

A third dose may be given, if needed, in twenty-four hours.

An ordinary sterilized *hypodermic syringe* holding 5 c.c. is suitable for making the injections. The skin should be cleansed with an antiseptic solution.

Place of Injection.—The skin of the thigh, the posterior axillary line of the chest, or the abdomen are favorable locations.

Effects of Antitoxin on the Pseudomembrane.—In a few hours after the injection the pseudomembrane becomes blanched, the dirty color less marked, and the membrane more granular and swollen. Later it becomes loosened around its edges, rolls up, and detaches itself spontaneously or after irrigation. If the membrane returns repeat the dose of antitoxin at once.

Effects on the Temperature.—In pure or simple diphtheria the temperature rapidly returns to the normal, whereas in the mixed cases it comes down more slowly. If the temperature does not fall in the regular way, a second injection is indicated, provided the temperature cannot be accounted for by some complication.

Indications for Antitoxin.—1. If it is suspected that the child has a mild pharyngeal, nasal, buccal, conjunctival, or cutaneous case, give antitoxin if he is over one year of age and there is a distinct history of exposure.

2. If a laryngeal case is suspected, give antitoxin *at once,* and make microscopic and cultural examinations afterward.

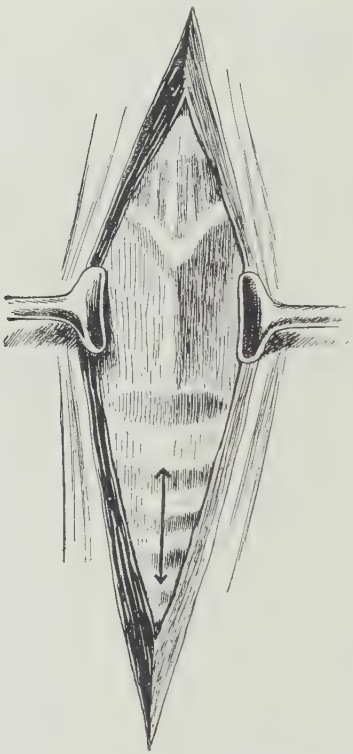
3. In all catarrhal cases antitoxin must be given.

4. In pseudodiphtheria, with repeated negative findings as regards the Klebs-Loeffler bacilli, antitoxin need not be given. If in doubt, however, give it.

Surgical Treatment.—*Tracheotomy.*—This operation is not now in vogue, relatively, as it was in former years. Intubation is usually elected in its stead, as it is a safer and surer means of tiding the patient over the suffocative period. Nevertheless, there are still cases in which tracheotomy is indicated.

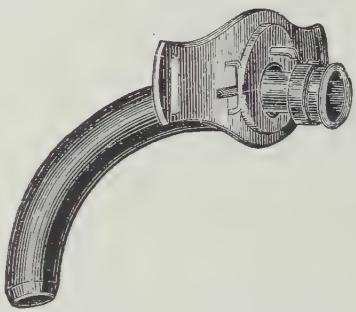
The *indications* for tracheotomy are: (*a*) When intubation tubes are not available, or if, for any reason, their use is not understood (Northrup);

Fig. 304



The line of incision in upper tracheotomy preparatory to laryngeal fissure or laryngectomy.

Fig. 305



Tracheotomy tube.

(*b*) in excessive edema of the larynx, where the intubation tube does not give relief; (*c*) when the membrane is in the lower tracheal tract, though these cases are favorable for tracheotomy.

The *method of performing tracheotomy* now in use is known as the high operation, in contradistinction to *tracheotome inférieure*, as first practised by Trousseau. In the low position of Trousseau, the blood-vessels passing over the field of operation render the operation difficult.

High tracheotomy is preferable. It should be done under antiseptic precautions, although this is not always practicable, on account of the urgency for immediate relief.

Steps.—(*a*) The cricoid cartilage should be located with the index finger of the left hand, while the larynx is held firmly but lightly between the thumb and the second finger.

(*b*) The skin and the subcutaneous tissue should now be incised, beginning with the location of the tip of the index finger, carrying it downward in the median line $\frac{1}{2}$ inch to 1 inch (Fig. 304).

(*c*) With the tip of the index finger in the superior angle of the wound, the bistoury should be passed under it into the trachea and the incision

carried downward in the median line far enough to admit the finger into the wound. With the finger thus placed blood cannot enter the trachea. A still better practice is first to check all bleeding with artery forceps or ligatures, and then open the trachea. If suffocation is imminent, the first method may be adopted.

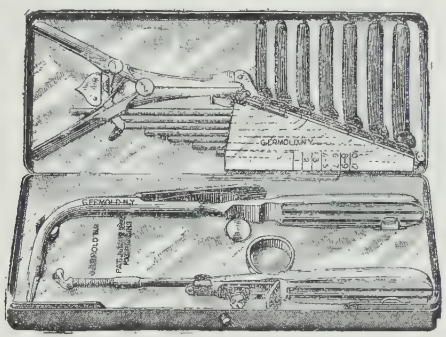
(*d*) The cannula (Fig. 305) should be next introduced as the finger is gradually withdrawn. If necessary, the dilator and the retractors may be used.

(*e*) The cannula should now be secured in its position by pieces of tape passed around the neck.

(*f*) If the suffocation is not relieved at once, there is either pseudomembrane still lower down in the trachea—perhaps a detached piece over the orifice of the cannula—or the cannula has become filled with mucus and shreds of pseudomembrane. In this event, the inner cannula should be removed and cleared of mucus, etc.

(*g*) If the removal of the inner cannula does not relieve the suffocation, there is probably membrane low down in the trachea.

Fig. 306



O'Dwyer's intubation instruments.

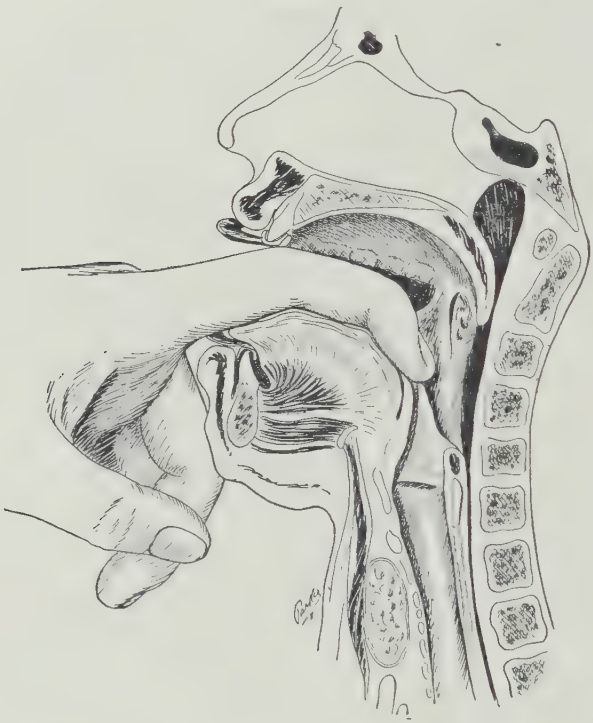
Mishaps or *accidents* which may attend the operation are: (*a*) Failure to open into the trachea, especially in very fat children; (*b*) hemorrhage when the incision is carried to either side or too far downward; (*c*) an irregular or too small incision, making the introduction of the cannula difficult; (*d*) secondary hemorrhage; (*e*) asphyxiation from dislodged membrane; (*f*) a too greatly retracted head, thus flattening the trachea and causing stenosis.

After-effects of tracheotomy may be summarized as follows: (*a*) Disappearance of the cyanosis and suffocation; (*b*) sleep; (*c*) coughing with expulsion of pieces of membrane and mucus through the cannula; (*d*) slight fever of two to three days' duration.

Complications which may arise are: (*a*) Infection of the tracheal wound, the bronchi, and the lungs; (*b*) ulceration of the trachea at the tip of the cannula; (*c*) erysipelas of the wound; (*d*) and most important of all, bronchopneumonia from the second to the seventh day after the operation. When this occurs the prognosis is grave.

After-treatment consists in: (*a*) The removal of the inner cannula every two or three hours for cleansing; (*b*) the external cannula should be removed and cleaned every twenty-four hours, the child being placed flat on his back, as in the operation—the wound should be cleansed each time the external cannula is removed; (*c*) under antitoxin it is not probable that the cannula will need to be worn after the third day, whereas under the older methods of treatment it was usually worn a week or more.

Fig. 307



Index finger of the left hand holding the epiglottis against the base of the tongue preparatory to intubation. (After Shurley.)

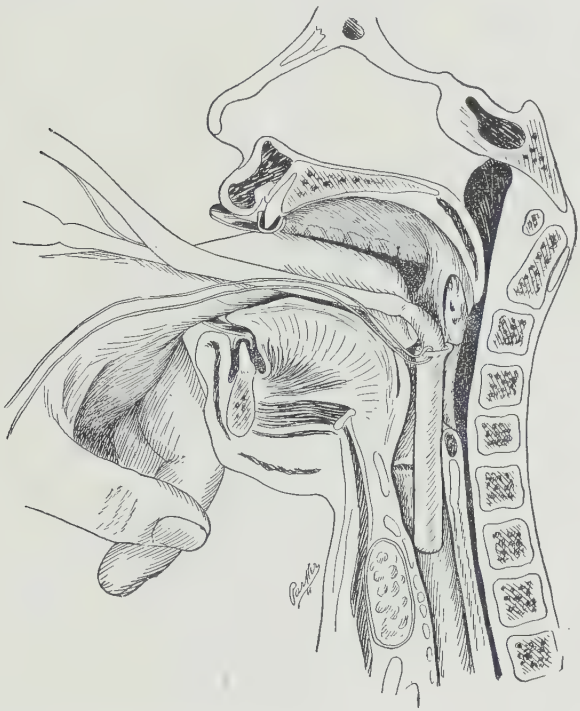
The author recently removed the cannula from a child who had worn it for four years. It was necessary first to dilate the glottis which curved Heryng bougies introduced through the tracheal opening. After a few treatments laryngeal respiration was sufficiently restored and the tube was removed. An attempt was afterward made to close the tracheal wound, but the anterior wall of the cartilaginous rings of the trachea had disappeared from pressure necrosis. The skin, when brought over the wound, acted as a valve closing the trachea, and asphyxia resulted.

Intubation.—To O'Dwyer is due the credit of first practising intubation upon his patients. The tubes used at first were straight and easily expelled. The tubes were gradually improved and their retention more sure (Fig. 306). At about this time Dr. F. E. Waxam successfully

intubated a patient in private practice. Dr. O'Dwyer was greatly encouraged by Dr. Waxham's success, and improvement in the tubes and instruments for their introduction and removal rapidly followed, and, though there was much opposition, intubation became one of the recognized therapeutic measures in stenosis from laryngeal diphtheria and immortalized O'Dwyer's name.

The introduction of antitoxin has very greatly reduced the necessity for intubation, though there are still many cases in which it is indicated.

FIG. 308



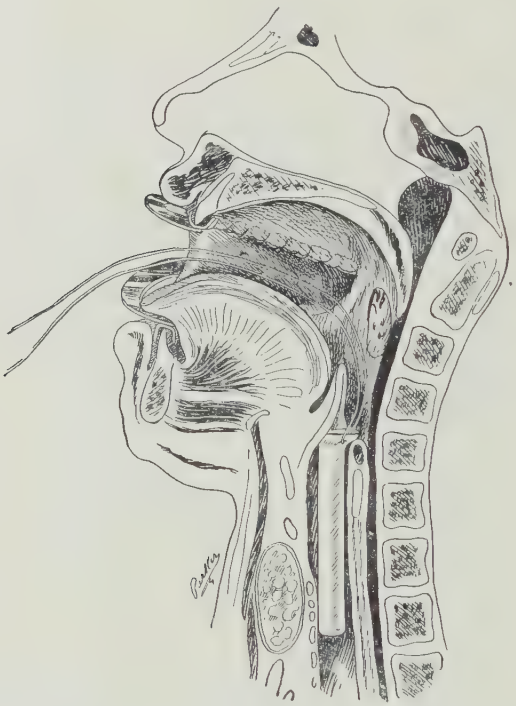
The tube passing through the chink of the glottis, the index finger still holding the epiglottis against the base of the tongue. A stout loop of thread is attached to the tube to provide for its speedy removal in case suffocative symptoms follow its introduction, and in case it is accidentally engaged in the esophagus.

Indications for Intubation.—(*a*) Great tracheal stenosis, as shown by much retracted supraclavicular and epigastric areas, necessitates the immediate resort to intubation, even though antitoxin has been given and sufficient time has not elapsed for its favorable influence. If milder suffocative symptoms are present, and antitoxin has been given, intubation may be delayed pending the results of the antitoxin. Since the use of antitoxin not one-half as many cases come to operation as formerly. (*b*) If not within easy call, the physician may intubate without waiting for marked suffocative symptoms.

Technique of Intubation.—The child is prepared for intubation by wrapping it in a sheet or a blanket from shoulders downward. The

sheet should be secured with strong safety pins, so as to bind the arms and legs of the child. This being done, the nurse should sit upright in a chair with the child upon her lap, his head resting against her left breast. His legs should be secured between hers, and her right hand should grasp his left, and her left hand his right. The assistant should stand behind the nurse and hold the child's head between his hands, as though suspending the child from the parietal walls of his cranium. A tube (Fig. 309) of proper size, threaded with silk through its eyelet, should be in readiness. The operator should stand or sit in front of the child,

FIG. 309

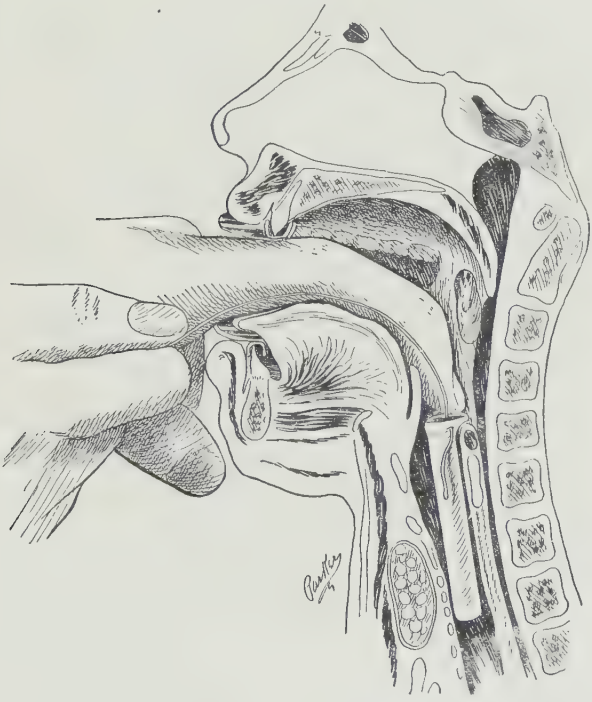


The tube in position in the larynx. The loop of thread is still attached, as the tube may have to be removed by the nurse to relieve impending suffocative symptoms.

introduce the mouth gag, turn it over to the assistant, who holds it between his hand and the patient's left cheek while the operator introduces the index finger of his left hand and hooks it over the epiglottis (Figs. 307 and 308). Then, after crowding his finger as far to the left as possible, the intubation tube, on the introducer, is carried into the mouth, immediately over the centre of the posterior portion of the tongue, the handle of the introducer being on the chest of the child. As the tip of the tube passes back of the epiglottis under the finger of the operator, the handle should be gradually elevated, until the tip of the tube is directly over the chink of the glottis, when it should be suddenly lowered, thus passing

the tube into the box of the larynx, and on downward into the glottis and the trachea. The tip of the finger then engages the rim at the head of the tube (Fig. 310), the introducer is loosened and removed, and with a gentle pressure the tube is firmly pushed deep into the larynx and the trachea. If after waiting twenty to thirty minutes the child tolerates the tube, the loop of string should be cut (Figs. 309, 310, and 311), the index finger reintroduced against the head of the tube, and the string removed. For obvious reasons the child should be kept wrapped until the string is removed. Fig. 312 shows a false entry of the tube into the esophagus, because the handle of the introducer was not sufficiently elevated before the tube was dropped into the laryngeal box.

Fig. 310



The removal of the loop of thread, the index finger of the left hand being placed against the head of the tube to prevent its displacement.

Intubation may also be performed in the dorsal position, the same relative positions and steps being observed as in the upright position.

Extubation or the Removal of the Tube.—The removal of the tube may be done by observing the same precautions as are used in intubation, the index finger of the left hand guiding the extractor to the opening in the tube (Fig. 313). Another method now occasionally used is to leave the silk string attached, looping it over the left ear and securing it to the cheek with adhesive plaster. The removal of the tube is thereby

rendered quite easy. It is also easy for the child to remove it, hence this is a serious objection to the method. One grain of Dover's powder, or $\frac{1}{16}$ to $\frac{1}{12}$ gr. of morphine, may be given a few minutes before extubation, to prevent spasm and reintubation for its relief.

When to Remove the Tube.—Under antitoxin treatment the tube may ordinarily, in a child over two years of age, be removed in from three to five days. Should the tube become obstructed, it should be immediately removed.

FIG. 311

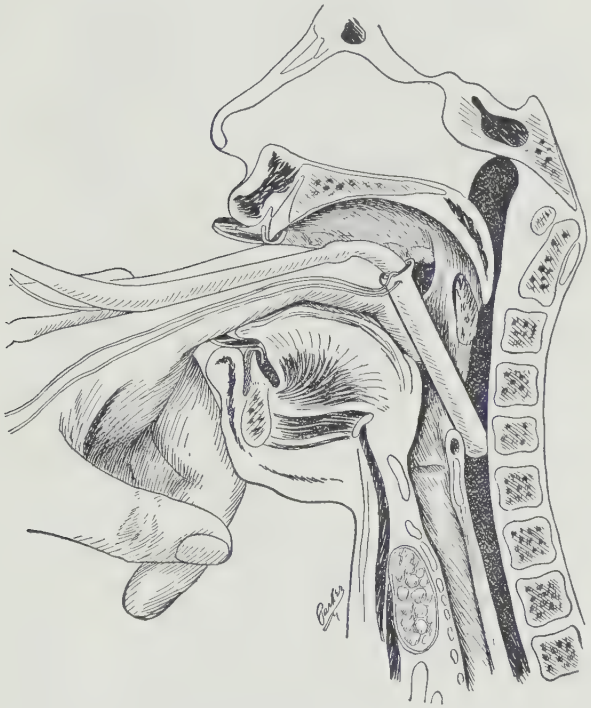
The tube in position after the withdrawal of the thread.

Complications and Difficulties.—(*a*) If the finger of the operator is short and stubby, it may be difficult to introduce the tube beside and beneath it. (*b*) The tube may make a false passage through the ventricles of the larynx. (*c*) The prolonged efforts of an awkward or inexperienced operator may cause suffocative symptoms. (*d*) Transient spasm of the glottis may cause temporary delay in introducing the tube. (*e*) The narrowest point through which the tube must pass is the cricoid ring, and edema or swelling at this point may give rise to some difficulty in introducing it. A smaller one may be passed with slight force. The action of the tube in being expelled in this condition has been aptly said to "creep back like an oiled cork in a bottle." (*f*) Prolonged retention of the tube may be necessary on account of the persistence of the pseudomembrane, ulcerations about the cricoid cartilages, traumatisms,

cicatricial contractions, edema, abductor paralysis, or exuberant granulations. (*g*) More rarely, the tube may be swallowed (no danger from it). (*h*) The tube may become obstructed by the thread or catgut being aspirated into it and swollen by the secretions; even food may obstruct it.

The Feeding of Intubated Children.—Most cases take liquid food very well when in the upright position, although some take it with pain and cough. If the upright position is not practical, Casselberry's position may be resorted to. It consists in placing the patient on his back with

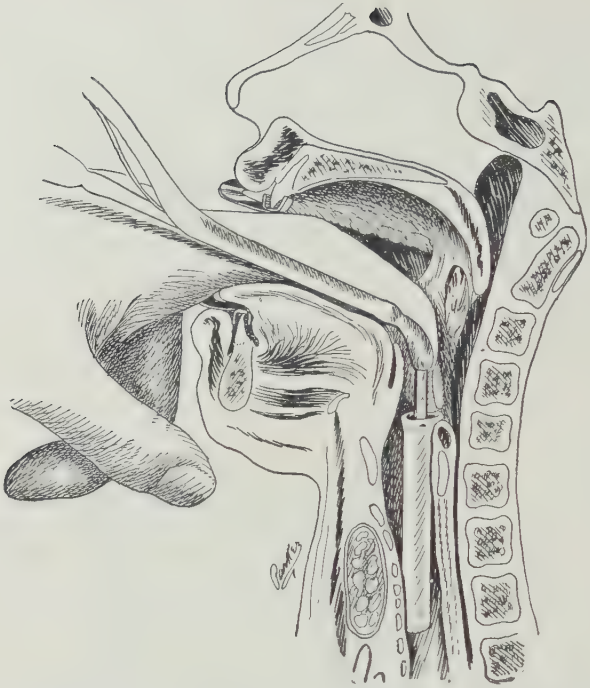
Fig. 312



Making a false passage into the esophagus on account of lowering the handle of the obturator. The tip of the tube should be introduced by the side of the finger tip, and the handle of the obturator elevated until the tube stands perpendicularly, and then passed directly downward through the chink of the glottis

a pillow beneath the shoulders, his head bent downward and backward at an angle of 45 degrees, the legs being elevated (Fig. 314). Liquid or semisolid food may be given in this position. The child should be allowed to swallow several times before assuming the upright position, to remove the food from the epipharynx. Hillis places the patient upon his stomach, as shown in Fig. 315. Gavage may be resorted to if the pharynx and the larynx are not too swollen and painful. The tube should be introduced through the nose and rapidly passed into the esophagus. Food being poured into the funnel passes into the esophagus and the stomach.

Fig. 313



Introduction of the obturator for the removal of the tube. The finger is first introduced to lift the epiglottis and to guide the tip of the obturator into the intubation tube.

Fig. 314

Feeding an intubated child with a nursing bottle. Casselberry's position. The shoulders are raised to allow the head to assume a lower position than the shoulders.

When removing the tube, pinch it to prevent the liquid passing into the larynx as it comes out.

Fig. 315

Feeding an intubated child through a rubber tube by suction.

Rectal alimentation may be resorted to if feeding by either of the foregoing methods is not practicable.

CHAPTER XXVI

PACHYDERMIA LARYNGIS. MALFORMATIONS AND DEFORMITIES PROLAPSE OF THE VENTRICLES. STENOSIS SUBGLOTTIC STENOSIS

According to Chiari, the verrucous form of pachydermia is identical with the papilloma of the laryngologist, and has no relation to the diffuse form. Diffuse pachydermia may be primary, or it may be secondary to some other affection of the larynx, such as tubercle or syphilis. In Chiari's experience typical pachydermia is a very rare disease. He describes the following forms:

1. The most frequent and mildest form is a thickening and loosening of the epithelium of the interarytenoid fold and the vocal cords, such as frequently occurs in chronic catarrh. The treatment is the same as for chronic catarrhal laryngitis, and consists of inhalations, insufflations, applications by means of a brush, and cauterization. The best applications are lactic acid and iodine. The nitrate of silver is apt to cause increased thickening. Small singer's nodules may disappear under the influence of rest and the application of the nitrate of silver in solution or in the solid stick. If they are of considerable size, forceps should be used to remove them.

2. The typical form of pachydermia laryngis (chorditis nodosa), as it affects chiefly the vocal processes, calls for a plan of treatment varying according to the circumstances of the case, authors differing greatly in their opinions. Some recommend purely expectant treatment and avoidance of tobacco, strong drinks, and the abuse of the voice; others recommend the internal administration of the iodide of potassium, which, though occasionally of some benefit, may also at times produce general impairment of health. Chiari recommends the use of electrolysis, as employed by Moll, of Arheim, a current of from 10 to 12 milliampères being used for from three to five minutes at a time. He considers it the best means of preventing recurrence, though good results have also followed operative procedures.

3. Large genuine pachydermia growths in the interarytenoid space interfere very materially with the voice. Unfortunately, treatment by means of cutting forceps, hot or cold snares, etc., do not guarantee freedom from recurrence.

4. The last group includes those circumscribed thickenings, outgrowths, or nodules which accompany tuberculosis, syphilis, chronic perichondritis, and perhaps also lupus, which have been referred to as secondary or "accessory" pachydermia. The prognosis depends on their etiology, as also does the treatment, the latter varying according

to the nature of the most distressing symptoms. Naturally the syphilitic form is much more favorable than the tuberculous, though not infrequently it resists specific remedies. Operative treatment of the same kind as for the typical primary form is called for in suitable cases; that is, if the general health is good and the respiration or voice is not seriously interfered with by the local disease. The method of treatment which is most highly recommended is the use of electrolysis by means of a bipolar instrument with a current of from 10 to 15 ma. This causes no reaction, and seems to protect against recurrence better than any other treatment.

There is no doubt that pachydermia laryngis, whether in the simplest form in the interarytenoid space or in the typical form on the processus vocalis, is only a symptom of chronic catarrh, and is not to be looked upon as a disease itself.

MALFORMATIONS AND DEFORMITIES OF THE LARYNX

Malformations of the larynx may be either congenital or acquired. But little is known concerning the true cause of congenital malformations, only that some paternal disease or taint acts as a predisposing factor. Acquired deformities are the result of postnatal disease.

Malformations of congenital origin are often associated with arrested development of the genitalia. The lungs, the bronchi, and the trachea have the same embryological origin (the foregut) as the larynx, hence in malformations of the larynx there is also a similar defect in these organs. In monstrosities having no larynx the lungs are also absent. If the larynx is diminutive, the lungs are likewise affected. Of the other congenital deformities, webs or bands across the glottis are a common form. The webs usually connect the vocal cords at the anterior commissure, though they are sometimes between the ventricular bands. They are of a pale color, but may be differentiated from the vocal cords by their position. They may be either fragile or resilient. The perforated diaphragm variety is rare, and is associated with poorly developed lungs. Another form of congenital malformation consists of clefts in the interarytenoid space extending to the palate and the cricoid cartilage. The epiglottis is often deformed by arrested development, the small V-shaped epiglottis of childhood being a common variety. A very small larynx and total absence of this organ have been reported.

Hypertrophy or *hyperplasia* at the anterior commissure has been mentioned as being of congenital origin.

Laryngocele (dilatation of pouches) is due to congenital malformation and failure of union in portions of the thyroid cartilage. It is rare in man, though common in the lower animals.

In *acquired malformations*, erosions from syphilis, tuberculosis, etc., may result in the partial destruction of the framework of the larynx, and the epiglottis is also often thus partially destroyed.

Acquired stenosis (see also Stenosis of the Larynx) may follow traumatism or constitutional causes such as syphilis. These cases are serious

on account of the edema and the dyspnea. Tracheotomy or intubation may become necessary. Redundant granulations following the prolonged use of the tracheotomy tube caused laryngeal stenosis in one of my cases. The child had been tracheotomized four years before he came under my care, and upon examination I found him unable to breathe through his larynx. The larynx was opened by bougies passed upward through the tracheal wound and through the glottis. This procedure was performed under general anesthesia.

Hypertrophies or growths, usually of a *papillomatous nature*, form at the anterior commissure in either the single or the multiple variety. Microscopically they appear as local hypertrophies of the mucous membrane, having a stratified epithelial covering, enclosing a core of connective tissue with some bloodvessels and a glandular substance near the base. Indeed, they are but elevations of the normal tissue. This seems to distinguish them from true papilloma. Though these papillomatous elevations of the mucous membrane are congenital, mouth-breathing, according to Lennox Browne, tends to perpetuate them.

PROLAPSE OF THE VENTRICLE OF MORGAGNI

Watson Williams claims that there can be no prolapse of the ventricles, but that which appears to be a prolapse is, in fact, an infiltration of the tissues. This is apparently supported by the fact that nearly all reported cases have been either syphilitic or tuberculous. On the other hand, the tumor-like mass is quite soft to probe pressure, and a number of observers have reported successful, though fugitive, replacement of the pouching membrane.

The presence of this condition should arouse suspicion of either syphilis or tuberculosis. The treatment by local applications is useless. Replacement, followed by cauterizations to excite inflammatory reaction, offers some hope of permanent cure. Extirpation of the mass with cutting forceps, or by thyrotomy, may be resorted to if simpler measures fail. Antisyphilitic remedies should first be tried, however, before surgical interference is attempted, unless it becomes necessary to perform tracheotomy to relieve suffocative symptoms.

STENOSIS OF THE LARYNX (MALFORMATION OF THE LARYNX)

Stenosis of the larynx properly comes under malformations, but its importance merits separate treatment; hence, the various types of stenosis are included in this section, regardless of their relationship to malformations. Stenosis arising from constitutional disorders, as syphilis, tuberculosis, and leprosy, each have their peculiarities.

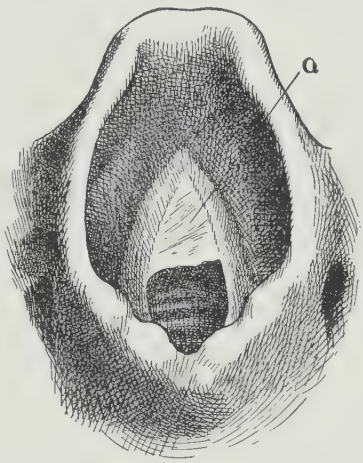
Syphilitic Stenosis.—There are three prominent conditions arising in the course of syphilitic laryngitis which may cause laryngeal stenosis, namely:

(*a*) Chronic edema.
(*b*) Cicatricial contraction or webs.
(*c*) Hyperplastic or papillary growths.

(*a*) **Chronic Edema.**—Chronic edema is commonly present in syphilitic laryngitis, though it does not always seriously occlude the glottis. Nevertheless, it presents favorable conditions for the supervention of an acute process, which may produce serious stenosis. This is especially true in children who inherit a syphilitic taint. Such children are predisposed to acute edema, which gives rise to symptoms quite like those found in croup. Fortunately the infantile cases respond quickly to antisyphilitic remedies. In adults, as well as in children, the treatment consists in the administration of the iodide of potash or iodonucleoid, which often reduces the local edema in a short time.

It should be stated that it is the tertiary stage of syphilis that results in stenosis, hence the treatment should be conducted accordingly.

Fig. 316



a, cicatricial web across the anterior commissure of the vocal cords.

(*b*) **Webs and Cicatricial Contraction.**—Webs and cicatricial contraction are the most common manifestations of syphilitic laryngitis. The webs vary in color and thickness. They are usually pale, and may be indistinguishable from the cords over which they extend. The vocal cords and the ventricular bands are usually bound together, and the web often extends across the chink of the glottis, especially at the anterior portion (Fig. 316). Lennox Browne cites a case in which the epiglottis was bound down by cicatricial adhesions.

The voice is hoarse or restricted in its register, while the breathing is dyspneic. The degree of the dyspnea depends upon the amount of edema and fixation of the cartilages, as well as upon the overlying web or cicatricial tissue. When a patient gives a history of recurrent attacks of dyspnea extending over several years, it is presumptive evidence that he is suffering from syphilis of the larynx. A spasmodic cough, not unlike that in pertussis, is usually present. Pain is not uncommon. There may be an admixture of syphilis and tuberculosis, which may somewhat obscure the diagnosis.

(*c*) **Hyperplastic or Papillary Growths.**—These usually form near the anterior commissure of the glottis, and they may be either single or multiple. The treatment should be antisyphilitic and expectant. If the growths produce stenosis, they should be removed with laryngeal forceps, the snare, or by laryngofissure.

Tuberculous Stenosis.—Tuberculosis of the larynx does not often close the glottis by cicatricial contraction, as in syphilis. This is explained by the slight reparative effort following tuberculous ulceration. It may produce stenosis by the excessive infiltration of the arytenoid cartilages, which may overhang the glottis and occlude the respiratory passage. Tuberculous perichondritis and chondritis may result in fixation of the arytenoids, and thus prevent abduction of the vocal cords. The lumen of the glottis is thereby rendered very narrow, and distressing dyspnea results.

Lupous Stenosis of the Larynx.—Lupus of the larynx is characterized by a cicatricial contraction and matting together of the parts. Lupus runs a much more chronic course than active tuberculosis of the larynx, hence the greater changes. Virchow says that the arytenoids are occasionally surrounded by hard papillary growths in the active stage of lupus. The scar tissue in lupus is very unyielding and not readily absorbed, even under the pressure of laryngeal tubes.

Leprous Stenosis.—The stenosis rarely occurs until the patient is in the last stages of the disease. In this stage it often becomes so great as to necessitate tracheotomy to relieve the distressing dyspnea.

Ventricular Eversion and Stenosis.—The eversion of the sacculus laryngis is scarcely possible as a primary condition. (See Prolapse of the Ventricle of Morgagni.) Anatomically it appears to be too firmly adherent to the adjacent tissues to permit of its prolapse. There may be a disease of the underlying perichondrium of the laryngeal cartilages which predisposes to the eversion and the consequent stenosis. Tumors and glandular enlargement may also push the sacculus toward the median line and cause stenosis.

Traumatic Stenosis.—Stenosis of the larynx may be due to the inhalation of hot vapors or to ingestion of corrosive fluids, as carbolic acid. It may also be due to a penetrating wound. In a case recently under my care the stenosis was due to the use of a 60 per cent. solution of carbolic acid as a gargle. The acute edema rendered it necessary to perform tracheotomy. The tube had been worn for seventeen months when I first saw him, and had excited a hyperplastic nodule just below the posterior portion of the cords. The stenosis seemed to be due more to hyperplastic nodule than to cicatricial contraction caused by the carbolic acid. We may, therefore, include the prolonged use of the tracheotomy tube as a cause of laryngeal stenosis.

Treatment.—The treatment of laryngeal stenosis is both medical and surgical.

Medical Treatment.—(*a*) In syphilitic edema and infiltration without cicatricial contraction, the iodides are indicated. Saline laxatives may be given with good results.

(*b*) Acute edema supervening upon a preëxisting fibrous stenosis should be treated by the local application of adrenalin and by free saline catharsis.

(*c*) The edema of tuberculous laryngitis may be relieved by tonic remedies and the cautious administration of mild cathartics.

Surgical Treatment.—(*a*) Webs of syphilitic origin should be broken down by systemic dilatation by means of Schroetter's laryngeal tubes (Fig. 317). The larynx should be cocainized, the index finger of the left hand introduced through the narrowed chink of the glottis. The web will thus be stretched and torn. A larger tube should be introduced after leaving the first one in place a few minutes. This process should be continued three times a week until the stenosis is completely overcome. Even then the tubes should be introduced at intervals of a few weeks to prevent the reformation of the webs.

(*b*) Cicatricial contraction due to syphilis should be overcome in the same manner as described in the preceding paragraph, though the dilatations will have to be performed more persistently.

(*c*) Hyperplastic or papillary growths of syphilitic origin do not always yield to the iodides, and should, therefore, be removed with laryngeal forceps under general or cocaine anesthesia, by either direct or indirect method. Occasionally the papillary growths become wedged in the chink of the glottis and cause sudden and alarming dyspnea, and necessitate an emergency tracheotomy. (See Tracheotomy.)

(*d*) Tuberculous chondritis and abscess of the larynx, when causing stenosis, should be relieved by the removal of the diseased and dislocated cartilage with a laryngeal curette or biting forceps.

Fig. 317

Schroetter's laryngeal dilator.

Tuberculous ankylosis of the arytenoid cartilages, attended by fixation of the cords in adduction with severe dyspnea, necessitates tracheotomy for the immediate relief of the symptoms, or laryngofissure may be necessary at a later time to overcome the ankylosis, or to remove the arytenoid cartilages. The abduction of the cords during respiration is thus made possible and the distressing dyspnea relieved.

(*e*) Cicatricial stenosis of lupus should be treated by dilatation with Schroetter's tubes, as described in a preceding paragraph, excepting that it may require greater persistence.

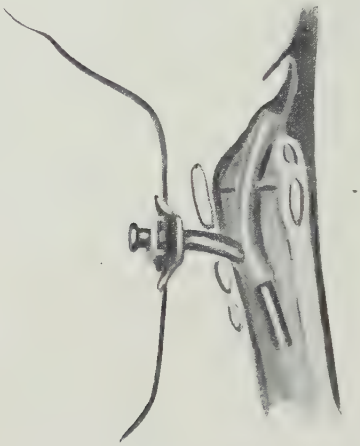
(*f*) Leprous stenosis should be relieved by tracheotomy if the gravity of the suffocative attacks warrant it.

(*g*) Ventricular eversion with stenosis, while secondary to some diseased process of the underlying perichondrium, should be overcome by removing the prolapsed sacculus membrane with a snare under cocaine anesthesia. Failing in this, tracheotomy may be performed, and the everted mass removed subsequently by laryngofissure. (See Laryngofissure.)

Traumatic stenosis, whether of chemical or mechanical origin, may

often be successfully treated by first performing laryngofissure (see Laryngofissure), and then introducing a tracheotomy tube with a rubber

Fig. 318



Tracheotomy tube with rubber tube extension for stenosis of the larynx.

Fig. 319

Tracheotomy tube with rubber tube extension for stenosis of the larynx.

tube extending upward from it through the chink of the glottis (Figs 318 and 319. The rubber tube exerts constant pressure and gradually

removes the hyperplastic tissue causing the stenosis, by pressure atrophy. Chevalier Jackson recently reported seven cases successfully treated by this method. My own case is progressing favorably and promises to be entirely successful. The tube should be worn for from four to sixteen weeks, and should be removed every two or three days.

Subglottic Stenosis.—Sajous pointed out that the subglottic space has not received the attention which its importance as an inherent portion of the larynx warrants. He urges systemic examination of this space in all laryngeal cases. The forms of stenosis peculiar to the lower subglottic region present features of unusual danger and symptoms likely to be ascribed to syphilitic disease. Inasmuch as the iodide of potassium greatly increases the danger in subglottic stenosis, it should not be administered in a case presenting dyspnea as a symptom, unless the non-existence of this condition is determined by infralaryngoscopic examination, or the causative disease is clearly recognized as being independent of the respiratory tract. He advised that preliminary tracheotomy be performed when the iodide of potassium is to be administered during the existence of advanced subglottic stenosis.

Massei states that the subglottic space is the most frequent seat of syphilis, tuberculosis, tumors, rhinoscleroma, and foreign bodies. Slight syphilitic stenosis is frequently curable without local treatment by the administration of sublimate injections with or without the iodides. In simple inflammatory and neoplastic stenosis, intubation offers the best results. He agrees with Sajous that too great dependence is placed in general antisyphilitic treatment in severe stenosis, and that such a course may be fatal.

CHAPTER XXVII

NEUROSES OF THE LARYNX

NEUROSES OF MOTION

The classification of J. Solis-Cohen is as follows:

Neurosis of the Motor Nerves of the Larynx.—The motor neuroses are divided into two groups:

1. Spasms of the larynx, or hyperkinesis, *i. e.*, excessive motion.
2. Paralysis of the larynx, or akinesis, *i. e.*, absence of motion.

Spasms of the Larynx.—Spasms of the larynx may be due to irritation of the central brain cells, whereby all the intrinsic muscles are thrown into violent action, or to irregular nervous impulses sent out from the motor centres of the brain, causing incoördination of the laryngeal muscles.

Paralysis of the intrinsic laryngeal muscles may be limited to one muscle or to a group of muscles, or it may affect all of them.

The spasms may be either tonic or clonic.

Tonic spasms are (*a*) of central origin; (*b*) from irritation of the trunk of the recurrent laryngeal; and (*c*) from reflex irritation.

(*a*) **Tonic Spasms of Central Origin.**—In tabes dorsalis, spasm of the adductors of the larynx occurs. The clinical picture shows sudden dyspnea with loud inspirations, the cords remaining in adduction for a variable time. It also occurs in tetanus and hydrophobia.

(*b*) **Tonic Spasm from Irritation to the Trunk of the Recurrent Laryngeal Nerve.**—When the injury is transient and slight, the laryngeal spasm is a forerunner of paralysis. Aneurysm of the arch of the aorta, cancer of the esophagus, pleuritic adhesion of the apex of the right lung, and tumors of the mediastinal glands may cause the irritation. A slight lesion may also occur in tabes.

(*c*) **Tonic Spasms from Reflex Irritation.**—These may occur from irritation of the larynx, the fauces, and the neighboring parts. In highly sensitive children irritation in a remote part of the body may cause adduction spasms. The latter condition has been described as laryngospasm infantum, and is usually due to intestinal irritation, tapeworm, a tight prepuce, or constipation.

Clonic spasms of the laryngeal muscles are always of central origin, and they consist of rhythmical inward movements of the cords. The condition may last but a few minutes, or it may persist for many months. The pillars of the fauces are also often affected in a like manner.

Both tonic and clonic spasms may be present in the same case, especially in the depressors of the epiglottis. The disease most often causing

clonic spasms of the larynx are syphilis, meningitis, and intracranial tumors.

Clinically, spasm of the larynx may be classified as follows:

(*a*) Spasm of the adductor muscles (laryngismus stridulus).

(*b*) Spasm of the tensor muscles.

(*c*) Spasmodic laryngeal cough or laryngeal chorea.

(*a*) **Laryngismus Stridulus (Adductor Spasm).**—*Synonyms.*—Spasm of the larynx, laryngeal spasm; spasm of the adductors of the vocal cords; spasm of the glottis; spasmus glottidis; false croup; child-crowing; thymic asthma; asthma rachiticum; Miller's asthma.

Laryngismus stridulus is a spasmodic act of the intrinsic muscles of the larynx accompanied by stridor. It is a neurosis, and is not necessarily associated with laryngeal disease. It is not a disease, but a symptom. While it is not a disease, it is a symptom causing great alarm. It is often associated with laryngeal or tracheal diseases, though it may be a reflex phenomenon from irritation in either contiguous or remote organs. It is sometimes a symptom of acute laryngitis, pseudomembranous croup, and diphtheritic croup, especially in children. It may also occur in non-inflammatory diseases of the larynx. It is common in children, but rather rare in adults. It is sometimes associated with intestinal disorders, as indigestion, worms, and constipation. Uterine disorders and sexual excesses have been known to produce it. Disorders of the contiguous organs, as the lingual tonsils, the teeth (dentition), elongated uvula, and inflamed tonsils, sometimes excite the spasm. Irritation of the fauces with a brush, or a foreign body in the pharynx, sometimes causes the symptom. Cases have been reported in which the pressure from an enlarged thymus gland caused laryngismus stridulus. Cerebral irritation, caries of the vertebræ, and rickets are known causes. Laryngismus stridulus appears in the laryngeal crises of tabes.

Treatment.—The treatment consists in relieving the source of the irritation rather than in applications to the larynx. For the immediate relief from the suffocative spasm, the application of cold water to the chest or hot water to the nape of the neck should be made. If suffocation seems imminent and the lower jaw is relaxed, seize the tongue between the thumb and the forefinger and exert traction about every three seconds, to excite the respiratory centre through the reflex action of the phrenic nerve. If the jaw is set, the same result can be accomplished by exerting pressure with the fingers under the angles of the jaw. Should these measures fail, resort to intubation or tracheotomy.

(*b*) **Spasm of the Tensor Muscles of the Vocal Cords; Aphonia Spastica; Phonatory Spasms.**—Spasm of the tensor muscles is essentially a neurosis from overuse of the voice. The muscles are fatigued and fail to respond to the nervous stimulus sent out from the motor centres of the brain; they are tired and irritated by a local accumulation of the toxins from faulty metabolism. Writer's and telegrapher's cramp are similar affections.

Symptoms.—Spasm of the tensor muscles is characterized by sudden onset at any moment during speech. It may come on at the beginning

or in the midst of a sentence. I have seen cases in which the speech was suddenly almost or entirely lost for some minutes, after which it would quickly clear up and remain so for an indefinite period. The patient complains of a rough, harsh feeling in the larynx, accompanied by the spontaneous flow of a few tears and slight congestion of the conjunctivæ. A drink of water hastens the cessation of the spasms. The cords are tense and approximated in the median line.

Treatment.—The cases seen by the author have been mild, and occurred only at long intervals. They required no special treatment other than a few minutes' rest of the voice and a drink of cold water.

In severe and oft-recurring spastic aphonia, prolonged rest of the voice is necessary. Such cases are usually overtaxed, or are affected by a slight general debility, and they should, in addition to prolonged rest away from the persons with whom they are daily associated, be given tonic or specific remedies to correct the debility or the specific diseases with which each is affected. To this end iron, strychnine, arsenic, cathartics, iodide of potash, eggs, milk, etc., should be given.

(*c*) **Spasmodic Laryngeal Cough or Laryngeal Chorea.**—This condition is quite similar to chorea in other parts of the body, though it is not usually associated with it. There are, however, synchronous contractions of other respiratory muscles which furnish the blast of air back of the cough. The choreic cough occurs at frequent intervals, and is a dry, noisy, respiratory explosion resembling the yelp or bark of a dog. It occurs most often in females at about the age of puberty, or at the age of greatest instability of the nervous system. It rarely occurs during sleep. Between the intervals the voice is clear. The vocal cords appear normal, and are closely approximated during the attacks.

Treatment.—The cough is due to an hysterical temperament or to a lack of balance of the nervous system at or about the age of puberty, and little can be done to improve it. A sea voyage or an outdoor life will add tone to the system, and thus tend to check the recurrence of the attacks. Tonics and sedatives may also be administered. The child should be taken from school and sent to the country, or in some way kept outdoors. Fresh air and sunshine will do more for these cases than any other mode of treatment.

NEURALGIA OF THE LARYNX

True neuralgia is rare, and is characterized by pain without a visible cause. Similar pain may be caused by malaria, gout, rheumatism, pressure from some tumor or swelling, epipharyngitis, and angina of the pharynx. It is obvious, therefore, that the foregoing diseases should be excluded before making a diagnosis of neuralgia.

Treatment.—The treatment of a true neuralgia is successfully accomplished with phenacetin, gr. v to x, every three hours, also with cannabis indica, aconite, and morphine, which should be administered until they produce their physiological effects. Though cocaine, if sprayed into the throat, affords immediate relief, it is not to be recommended because

neuralgic patients easily acquire the cocaine habit. Menthol affords relief. Cold or hot applications to the neck also prove grateful to these patients.

If the pain is due to gout, rheumatism, malaria, or pressure of a tumor or a gland, treatment appropriate to these conditions should be instituted.

MOGIPHONIA

Mogiphonia is characterized by a difficulty in maintaining the tension of the vocal cords while singing, or during forced accentuated speaking. In ordinary conversation no difficulty is experienced.

Treatment.—The treatment is rest. Overtaxation being the cause, other forms of treatment are not indicated, unless the condition has recurred often and at frequent intervals. When this is the case, tonics, massage, cathartics, and eliminative treatment should also be used.

NERVOUS COUGH

This is a spasmodic, croupy, or even musical laryngeal cough, for which no physical cause can usually be assigned. It is peculiar to neurotic individuals who present other stigmata of a neurosis. It is a "daytime" cough, which subsides entirely during sleep, but returns the following morning, often with increased severity. It may be a reflex disturbance from a hypersensitive area in the nose, the epipharynx, or the chest, hence a careful examination of these parts should be made. The sensitive areas in the nose and the epipharynx may be located by gentle probe pressure without the use of cocaine. In the nose Jacobson's tubercle near the anterior end of the middle turbinated body may be the seat of the sensitive area. When this is touched with the probe it will give rise to the peculiar nervous cough, provided, of course, that it is the source of the reflex. Impacted cerumen in the external auditory meatus may cause it. The reflex may also have its origin in the gastro-intestinal tract.

Treatment.—As most cases are due to a true neurosis rather than to some physical lesion, the treatment must be of a tonic and sedative character. Sprays of iced lime water, or menthol in combination with camphor, gr. ij to an ounce of liquid petrolatum, etc., may be used to relieve the laryngeal irritations. Antispasmodics and sedatives, as aconite, cannabis indica, and the bromides, may be given internally to allay the spasms and the local irritation.

LARYNGEAL APOPLEXY

Synonyms.—Laryngeal vertigo; laryngeal syncope; bronchial syncope; complete glottic spasm in the adult.

Laryngeal apoplexy is characterized by a transient irritation and burning sensation in the lower part of the throat, followed by a fit of coughing, dimness of vision, dizziness, and unconsciousness, the patient falling to the floor. The face may be either congested or pale.

The disease is a neurosis affecting the coördination of the respiratory centres and the nerves of the larynx. It is rare. The attacks may last but a few seconds, when the spasms cease and the mind becomes clear again. They may recur at intervals of a few weeks.

Etiology.—The disease is chiefly found among the well-to-do and those leading sedentary lives, though one case is reported as occurring in a sailor (Whalan). Getchell reported 77 cases ranging in age from seventeen to seventy-seven years. All but four were males. Rheumatism and gout are occasionally associated with it. Neurasthenia is a rather constant factor. Local inflammatory disease of the bronchi, the pharynx, and the larynx is commonly present, and may be an important causative agent. Lennox Browne reported 3 cases in which there was varix at the base of the tongue.

Among the exciting causes may be named worry from strenuous business or social conditions, and either physical or mental overwork. A pinch of snuff or other irritating substance inhaled into the larynx and the bronchi may bring on an attack.

Symptoms.—The face is usually flushed, though it may be pale. A deep breath is taken, followed by laryngeal spasm. There may be epileptiform convulsions, and the sequence ends in a few moments by a return to consciousness. After the attack all signs of the disease disappear. The disease is clinically like apoplexy with a laryngeal aura and laryngeal spasm, the latter being continued long enough to produce unconsciousness. Such spasms are likely to occur in neurasthenia and in tabes. Other signs of neurasthenia, epilepsy, and tabes should be sought for before pronouncing the case one of laryngeal apoplexy.

Treatment.—The treatment should be addressed to the correction of alimentary and hepatic disorders and to the regulation of the excretory organs of the body. Tonics and antispasmodics may be given to tone and tranquillize the nervous system. Local lesions, if present, should receive appropriate treatment. For instance, bronchitis is the most common concomitant disease, and possibly has something to do with its causation. It should, therefore, be treated by the administration of 4 grains of iodide of potassium in a glass of water after each meal for several weeks or months. By relieving the associated diseases of the upper respiratory tract, the laryngeal spasms and the syncope are sometimes entirely relieved.

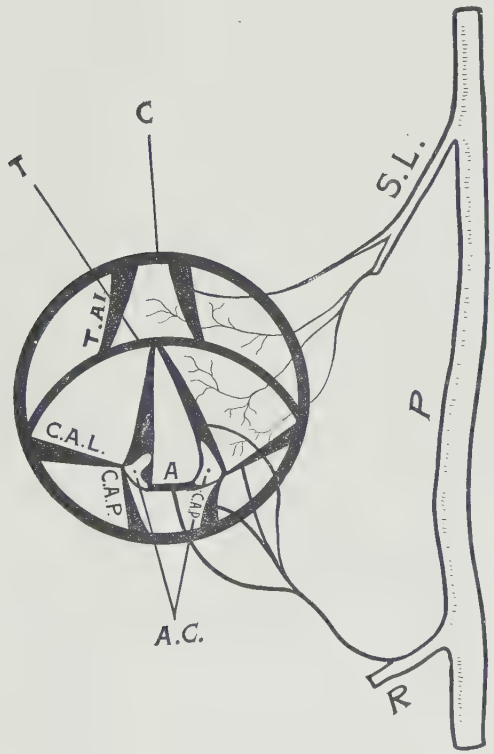
PARALYSES OF THE INTRINSIC MUSCLES OF THE LARYNX

It is difficult to make a classification of the paralyses of the laryngeal muscles in such a way as to have it coincide with clinical observation. The intrinsic muscles are supplied by branches of the right and the left

pneumogastric or vagus nerves. It will be remembered that these nerves have their origin near the median furrow beneath the floor of the fourth ventricle. Two motor branches, the superior laryngeal and the recurrent or inferior laryngeal, are given off from each vagus to the larynx. The superior laryngeal also supplies sensation to the whole laryngeal mucous membrane.

By reference to Fig. 320, it will be seen that the superior laryngeal supples only one pair of the intrinsic muscles of the larynx, the crico-thyroidei. These muscles are tensors of the vocal cords, hence the wavy outline of the cords (Fig. 321) in superior laryngeal paralysis.

FIG. 320



Schema of the nerve supply of the intrinsic muscles of the larynx: *P*, the pneumogastric nerve; *R*, recurrent laryngeal nerve; *S.L.*, superior laryngeal nerve; *A.C.*, arytenoid cartilages; *T*, thyroid cartilage; *C*, cricoid cartilage; *A*, interarytenoideus muscle; *C.A.P.*, crico-arytenoideus posticus muscle; *C.A.L.*, crico-arytenoideus lateralis muscle; *T.A.I.*, cricothyroidei interni muscles.

The recurrent or inferior laryngeal nerves supply all the other intrinsic muscles of the larynx, namely, the arytenoideus, the crico-arytenoidei postici, the crico-arytenoidei laterales, and the internal tensors of the vocal cords.

If the lesion involves all the fibers of the left recurrent laryngeal nerve, there is total paralysis of all the muscles of the left side of the larynx

except the cricothyroideus (external tensor). The same is true of the right side (Fig. 321). If the lesion involves only a small branch of the left recurrent, one muscle alone may be involved, say the crico-arytenoideus lateralis. This muscle is an adductor, hence there would be incomplete adduction of the anterior two-thirds of the vocal cord on the left side, while the opposite cord would slightly encroach beyond the median line. The adduction of the posterior third is controlled by the arytenoideus, hence, this muscle being unaffected, closure in that region is complete. Single muscles are rarely affected except in diphtheria and other local inflammations of the larynx, and in hysteria. It is always a question when a single muscle is affected, excepting one of the cricothyroidei, as to whether the lesion is in a nerve twig or in the muscle itself. Inflammatory infiltration may inhibit the nerve twig supplying a certain muscle, or the infiltration may cause a mechanical barrier to the proper motion of the muscle. Hysterical paralysis is, of course, not a true paralysis.

FIG. 321

Paralysis of the cricothyroidei. The only muscles of the larynx supplied by the superior laryngeal. All the other intrinsic muscles of the larynx are supplied by the recurrent laryngeal nerves.

Paralysis of involuntary muscles usually has its origin in a lesion of the medulla oblongata or the spinal cord. Lesions of the cerebral cortex, on the other hand, cause central paralysis of voluntary motion. In making a diagnosis in this class of cases, aphasia must be distinctly separated from aphonia; the same is true in considering the etiology. Kraus, in 1884, demonstrated that stimulation of the gyrus prefrontalis in the lower animals produced a contraction, or muscular movements, of the larynx, the pharynx, and the palate. Semon and Horsley fully substantiated the findings of Kraus by a long series of experiments on the lower animals.

Irritation of one of the external borders of the restiform bodies produces unilateral adduction of the vocal cords. Bulbar lesions usually produce unilateral paralysis, but many cases of unilateral paralysis are also caused by lesions in the medulla.

Laryngeal paralyses are seldom brought about by tumors of the medulla or the pons. Gottstein thoroughly reviewed this aspect of the question, and refers to several cases of glioma and one of aneurysm of the basilar artery. A bulbar lesion causing laryngeal paralysis usually involves the dorsal motor nucleus of the pneumogastric, which lies near the median furrow, and is beneath the floor of the fourth ventricle.[1] In

[1] Edinger, Anatomy of Central Nervous System of Man, English translation from fifth German edition, p. 375, says:

"We have learned, then, two nuclei for the vagus, *a ventral one*, which from its position (in the prolongation of the ventral horn) and from the appearance of its cells (multipolar with axis cylinders passing directly into the nerve) *is motor;* and *a dorsal one*, which, lying in the prolongation of the gray matter of the base of the posterior horn, is also by its structure characterized as *sensory*."

laryngeal paralysis the abductors are usually the first, perhaps the only, muscles affected as a result of a central or a peripheral lesion, while in hysterical aphonia the adductors are affected.

Tumors, traumatisms, and other lesions at the base of the skull give rise to laryngeal paralysis by implicating the trunks of the pneumogastrics. It is often difficult to differentiate these conditions from bulbar lesions, as they frequently involve the facial, the glossopharyngeal, the acusticus, the spinal accessory, also other branches of the pneumogastrics besides the laryngeals, depending upon the extent of the lesion. The portion of the pneumogastric which lies in the neck (usually the trunk and the recurrent laryngeal after it winds around the large vessels in the thorax, travelling back along the esophagus to the larynx) is very often the seat of the lesion causing the laryngeal paralysis. Among the lesions in this locality causing paralysis of the nerves just mentioned are enlarged glands, traumatisms due to wounds in operating, goitres, aneurysms, mediastinal tumors, tumors of the esophagus and the pharynx, pleurisy, scoliosis of the cervical vertebræ, tuberculosis of the apices of the lungs, and even pericarditis.

Laryngeal paralysis may be the very first and for a long time the only significant indication of an aneurysm of the arch of the aorta. Often no palpable reason for the paralysis can be ascertained, and then recourse must be had to a tentative diagnosis of a simple neuritis. The rare cases of paralysis of individual muscles must be ascribed to lesions of their respective nerve twigs, or to an involvement of the muscular structure itself. Paralysis of the abductors is now and then due to traumatism by the passage of a bolus of food or cold drinks through the lower pharynx into the esophagus, as the location of the muscles is very superficial. In paralysis of the pneumogastric nerve due to a bulbar lesion the involvement of the other nerves readily establishes the diagnosis. However, an injury to the base of the skull may simulate a bulbar lesion by implicating several nerve trunks in addition to the pneumogastric. Jackson, Proust, Senator, and Eisenlohr have reported cases of bilateral paralysis as being due to bulbar lesions, though they are comparatively rare. There is no authenticated case of paralysis of the adductors alone from a central lesion. Occasionally a bulbar lesion produces bilateral paralysis, in which instance the abductors alone are usually involved; more often the paralysis is unilateral, though not so often as when due to other lesions.

PARALYSIS FROM DISEASE OR INJURY OF THE SUPERIOR LARYNGEAL NERVE; PARALYSIS OF THE EXTERNAL TENSORS OF THE VOCAL CORDS

So far the only lesions which have been noted as causing paralysis of the cricothyroid muscles are diphtheria, enlarged glands, and inflammation of the areolar tissue beneath the angle of the jaw. Typhoid fever may cause it. Paralysis of these muscles is extremely rare.

Symptoms.—Anesthesia of the larynx, the phenomenon which was described under neurosis of the larynx, is a prominent and significant symptom. The anesthesia is explained by the fact that it is the superior laryngeal nerve, a branch of the pneumogastric, which is affected. This branch supplies the cricothyroid muscles with motor stimulus, and the whole of the mucosa with sensation. Whenever, therefore, there is anesthesia of the whole mucosa of the larynx, the lesion involves the superior laryngeal nerve fibers, either after they leave the pneumogastric or higher up in the pneumogastric itself. A low-pitched voice and inability to sing high tones is characteristic of this affliction. When the thyro-epiglottic and the aryteno-epiglottic muscles are paralyzed, the epiglottis stands upright, hence the larynx cannot be closed. Because of this and the attending anesthesia, food often finds its way into the larynx and upper respiratory tract. No warning is given the patient until the food reaches an area below the vocal cords. Hence, pneumonia is frequently a serious sequence. Complete bilateral paralysis of the cricothyroid muscles is manifested by the peculiar wavy outlines of the vocal cords (Fig. 321). According to E. MacKenzie, when this paralysis is unilateral the laryngoscope shows one vocal cord on a higher plane than the other.

Diagnosis.—The peculiar wavy outline of the vocal cords and the local anesthesia clear up the diagnosis as to the hoarseness and aphonia, and distinguish it as a true motor paralysis rather than a neurosis or an inflammatory disease.

Prognosis.—It is very bad if there is complete bilateral paralysis, but not so very grave when only one cord is implicated. The patient may succumb to inanition or pneumonia. Lobar pneumonia is the usual type, and cases have been recorded in which death from this disease could only be ascribed to the passage of food or other foreign substance into the trachea because of the anesthesia. The prognosis is very bad if the recurrent laryngeal nerve is involved at the same time.

Treatment.—Nourishment by the esophageal tube, galvanism, strychnine, and general tonics are indicated.

PARALYSES OF THE RECURRENT OR INFERIOR LARYNGEAL BRANCH OF THE PNEUMOGASTRIC NERVE

All the intrinsic muscles of the larynx except the cricothyroidei are supplied with motor stimulus by the recurrent laryngeal nerves. The crico-arytenoidei postici are abductors of the vocal cords and therefore muscles of respiration, in a sense, also, of phonation, as their action is necessary to maintain the required equilibrium of the other muscles in this act and in modulating the voice.

The recurrent laryngeal nerve supplies motor stimulus to the following muscles:

Recurrent laryngeal (inferior laryngeal nerve).	Crico-arytenoidei laterales (adductor). Arytenoideus (adductor). Crico-arytenoidei postici (abductor). Thyro-arytenoidei interni (relaxors).

The superior laryngeal nerve supplies the cricothyroidei (external tensors).

It is clear, from the above analysis, that the recurrent laryngeal nerve is the chief motor supply to the larynx, and that it presides over both adduction and abduction of the vocal cords.

It is obvious, therefore, that when all the fibers of the main trunks of the recurrents are affected there is total paralysis of both the adductor and the abductor muscles of the larynx. The only intrinsic muscles of the larynx not affected are the external tensors, the cricothyroidei, which are supplied by the superior laryngeal nerves. These play so small a part in the general movements of the cords that their action under these circumstances is practically nil. The cords, therefore, assume the so-called cadaveric position (Fig. 322). In studying the various paralyses of the recurrent laryngeals I shall first speak of total paralysis, and follow with the partial paralyses. I mean by the term partial paralysis, the paralysis of certain groups of muscles rather than an incomplete paralysis of part or all of the muscles of the larynx.

FIG. 322

Larynx in quiet breathing and the cadaveric position.

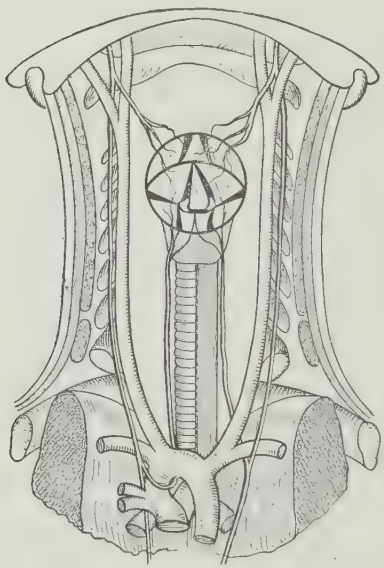
COMPLETE PARALYSIS OF BOTH RECURRENT LARYNGEAL NERVES

Etiology.—By reference to Fig. 323, the course and distribution of the right and the left recurrent laryngeal branches from the pneumogastrics is illustrated in diagrammatic form. The left recurrent is given off at the level of the transverse portion of the arch of the aorta, and passes under it, thence upward in the groove between the trachea and the esophagus to the muscles of the larynx. As it reaches the larynx it breaks into several twigs, thus supplying motor stimulus to all the intrinsic muscles of the left half of the larynx except the cricothyroid, which is supplied by the superior laryngeal. The left recurrent nerve is the one most often affected, on account of its relationship to the arch of the aorta and the left subclavian artery. Aneurysm of the transverse portion of the arch of the aorta causes compression and neuritis of the left recurrent laryngeal, and thus inhibits the motor impulses reaching the left half of the larynx. Unilateral paralysis results. Occasionally the aneurysm is so large as to encroach upon the structures on the right side of the chest, and may thus also cause compression and neuritis of the right recurrent, in which event the paralysis would be bilateral.

While the right recurrent laryngeal is not so often involved, it is,

nevertheless, so situated with reference to the subclavian artery and the apex of the right lung as to be somewhat frequently the source of laryngeal paralysis. The right recurrent nerve is given off on the level with the subclavian artery, and curves around the latter as it starts upward to the larynx. Aneurysm of the subclavian may therefore compress it and cause neuritis and consequent laryngeal paralysis of the intrinsic muscles of the right half of the larynx. The right recurrent nerve is in close proximity to the apex of the right lung, and may become involved in pleuritic exudates and adhesions in this region, and thus cause paralysis of the right half of the larynx.

FIG. 323



Schema showing the relations of the pneumogastric nerve to the trachea, esophagus, and vessels of the thorax. Also the recurrent laryngeal and superior laryngeal branches and their distribution to the intrinsic muscles of the larynx. (See Fig. 320.)

The mediastinum is frequently the seat of malignant or other growths which press upon one or both of the recurrent nerves. Enlarged glands of the neck, malignant tumors of the esophagus, and other growths in the neck may cause pressure and degeneration of one or both pneumogastric nerves, and produce unilateral or bilateral paralysis of the larynx. Scoliosis, goitre, and pericarditis may also injure the recurrent nerves. Gummata are frequently the source of the nerve lesion.

The central lesions which cause laryngeal paralysis are in the medulla oblongata or the spinal cord. The exact location of the pneumogastric nuclei seems to be, according to Kraus, Semon, and Horsley, in the gyrus prefrontalis. Tumors of the medulla and the pons rarely cause laryngeal paralysis. Aneurysm of the basilar artery is a known cause. Bulbar lesions causing laryngeal paralysis usually involve the dorsal motor nucleus of the pneumogastric nerve which lies near the median furrow beneath the floor of the fourth ventricle.

Tumors, traumatisms, and other lesions at the base of the skull give rise to laryngeal paralysis by implicating the trunks of the pneumogastric nerves. It is often difficult to differentiate these from bulbar paralysis, as these conditions often involve the facial, the glossopharyngeal, the acusticus, the spinal accessory, or other branches of the pneumogastric nerve.

The nerves and their filaments may be completely atrophied. The remains of the neurilemma have been found, but fatty degeneration is the most frequent degenerative change.

Symptoms.—The symptoms, whether due to lesion of the pneumogastric trunk or to the recurrent laryngeal nerve, are very much alike. The voice is usually weak and husky. The sensibility of the mucous membrane is usually unimpaired, unless the lesion of the pneumogastric trunk is above the point where the superior laryngeal nerve is given off. If both pneumogastric trunks or both recurrent nerves are injured, the voice is aphonic, as the cords stand in the cadaveric position. If the recurrent nerve on one side only is affected, the vocal cord on that side rests in the cadaveric position, while the opposite cord has its normal movements. Indeed, it encroaches beyond the median line upon attempted phonation, while during deep inspiration it is widely separated from the opposite cord. In one-sided paralysis the position of the arytenoid cartilages is characteristic; the arytenoid cartilage on the unaffected side overlaps the opposite arytenoid, and is either anterior or posterior to it. Cough is usually absent, and when present is usually due to an irritation of the trachea by the pressure of a tumor in the neck or upper mediastinum. The cough is like that in aneurysm of the arch of the aorta. I have seen a few cases of aneurysmal cough, and they were dry and slightly harsh or brassy. One case in particular was free from cough except in public gatherings or other places likely to excite the heart's action. Coughing and expectorating are performed with great difficulty in bilateral paralysis.

Dyspnea is absent in unilateral paralysis, but may be present in bilateral paralysis in spite of the fact that the cords are separated in the "cadaveric" position. In the "cadaveric" position the cords stand midway between adduction and complete abduction. They are not as widely separated as is usual in inspiration, hence the dyspnea.

In some cases the paralysis is partial, and the symptoms are, therefore, correspondingly modified.

Sir Felix Semon and Rosenback have shown that the abductor nerve fibers degenerate earlier than the adductor nerve fibers, hence the abductor muscle (crico-arytenoideus posticus) is paralyzed earlier than the adductor (crico-arytenoideus lateralis). This phenomenon is usually referred to as "Semon's law." If, therefore, the case is seen early the abductors may be paralyzed. If, however, the case is examined at a later period, the degeneration will have extended to both the abductor and the adductor nerve fibers, and the paralysis will affect both the abductor and the adductor muscles. This causes the so-called "cadaveric" position of the vocal cords.

Diagnosis.—Bilateral paralysis of the abductor nerves during quiet respiration bears a slight resemblance to complete paralysis. The act of phonation, however, is attended by the adduction or approximation of the cords, which readily distinguishes it from the passivity of the cadaveric position.

Prognosis.—In view of the serious nature of the causes which produce complete paralysis of one or both recurrent laryngeal nerves, the prognosis is grave. In case it is due to syphilitic gummata or to the pressure of enlarged glands, the prognosis under appropriate treatment is good.

If due to the toxemia of diphtheria or to an acute inflammation, complete recovery may occur in a few weeks.

Treatment.—The treatment depends upon the cause of the paralysis and the duration of the symptoms. If enlargement of the thyroid gland is the cause, the administration of thyroid extract may diminish the size of the tumor and thus relieve the pressure upon the nerve. An operable tumor causing pressure upon the trunk of the pneumogastric or the recurrent laryngeal nerve should be removed in order to relieve the pressure. If the nerve has undergone degenerative changes, improvement may be slight or may not result; if, however, the nerve is still healthy, the paralysis may disappear after the operation. In aneurysm of the arch of the aorta or of the right subclavian, dependence should be placed in the use of iodonucleoid in from 5 to 15 grain doses three times a day. Syphilitic gummata may be treated with mercurial inunctions and the internal administration of iodonucleoid in doses ranging from 10 to 25 grains three times a day; or the iodide of potash 10 to 60 grains three times a day. The iodonucleoid is as reliable a drug as the iodide of potash, and has the advantage of being tolerated by the most sensitive stomach. It is free from potash, having a nucleoid base. It is absorbed more readily by the blood and rapidly saturates the system with iodine, which is the active agent in both the iodide of potash and the iodonucleoid.

Galvanism and faradism combined with external massage over the laryngeal region may increase the circulation and nutrition of the atrophied muscles. Strychnine is also a valuable remedy, because it increases the nerve energy and tone of the muscles.

If the paralysis is due to diphtheria or one of the exanthemata, constitutional remedies, as strychnine, iron, and the bitter tonics, should be given to build up the waning and depleted cell energy. Eliminative remedies, to stimulate the excretory powers of the intestines, the kidneys, the liver, and the skin, should be given to clear the toxins from the blood and the lymph.

Tracheotomy may become necessary in a case of severe dyspnea.

UNILATERAL PARALYSIS OF THE RECURRENT LARYNGEAL NERVE

Etiology. -Unilateral paralysis of one-half of the intrinsic muscles of the larynx is quite common, as each nerve traverses a long and uninterrupted course before it gives off the terminal twigs to the muscles of the larynx. The left recurrent is given off from the pneumogastric nerve on a level with the transverse portion of the arch of the aorta around which it curves (Fig. 323) and passes upward in the groove between the trachea and the esophagus to the larynx. Aneurysm of the transverse portion of the arch of the aorta compresses it and causes degenerative changes and consequent laryngeal paralysis. Tumors of the mediastinum and of the neck or enlarged glands of the neck may compress and injure it. The right recurrent nerve is given off from the right

pneumogastric on a level with the right subclavian artery, around which it curves in close contact with the apex of the right lung. Aneurysm of the right subclavian causes compression and degeneration of the right recurrent laryngeal nerve, and paralysis results. Pleuritic inflammation and adhesions at the apex of the lung may involve the right recurrent and cause laryngeal paralysis upon that side. Malignancy of the esophagus or other growth, or inflammatory swelling, may involve either the right or left recurrent laryngeal nerve and produce unilateral paralysis.

Symptoms.—The symptoms include hoarseness or even aphonia at the beginning of the paralysis. Later, the unaffected cord compensates for the loss of motion on the affected side, and the aphonia or hoarseness is improved. Dyspnea is absent. The laryngeal image shows the vocal cord on the affected side in the "cadaveric" position, *i. e.*, half-way between adduction and abduction, while the unaffected cord performs both adduction and abduction without restraint. The epiglottis may deviate from the median line.

Prognosis.—The prognosis depends upon the cause. If due to a transient inflammation or exudate, it is good under appropriate treatment. If due to syphilis, the prognosis is good if the case is properly treated. If due to some incurable disease, the prognosis is correspondingly grave. If dyspnea is present, the prognosis is more grave.

Treatment.—When practicable, treat the disease causing the paralysis as in postdiphtheritic or postexanthematic and syphilitic affections. If an incurable disease, as carcinoma or sarcoma of the mediastinum, the esophagus, or the larynx, is the cause of the paralysis, treat the distressing symptoms as they arise. If the thyroid gland is enlarged, give thyroid extract, or perform thyroidectomy if the extract fails.

LARYNGEAL PARALYSIS FROM LESIONS OF THE MEDULLA AND THE NUCLEI OF THE SPINAL ACCESSORY NERVE

Laryngeal paralysis from disease or injury of the medulla oblongata and the nuclei of the accessory portion of the spinal accessory is characterized by paralysis of all the intrinsic muscles of the larynx on the side involved, or if only a few filaments are involved there will be paralysis of only one or at most two muscles of the larynx. It is still further characterized by the paralysis of certain muscles, extrinsic to the larynx, which are supplied by nerves having their origin in the immediate vicinity of the motor nucleus of the pneumogastric. Thus there may be paralysis of the facial, the acusticus, or of the nerves leading to the extremities.

Pathology.—Laryngeal paralysis due to a central lesion is dependent upon the involvement of the spinal accessory roots, from which some of the fibers of the pneumogastric nerves arise in the floor of the fourth ventricle. There must be a lesion in the medullary or nerve roots supplying the larynx. Syphilis, locomotor ataxia, progressive bulbar paralysis, multiple sclerosis, and tumors of the neck and the brain comprise the chief morbid anatomy of central paralysis of the larynx.

Diagnosis.—The diagnosis depends on the symptom complex of all the nerves involved. There is usually an associated paralysis of the nerves supplying the tongue, the palate, and the facial muscles, or of the nerves of audition, or of the extremities. Other regions supplied by the accessory root may be paralyzed. All the intrinsic muscles of the larynx may be paralyzed, or only a part of them, depending on whether all or only a few of the fibers from the pneumogastric motor nucleus are diseased.

Prognosis.—The prognosis is nearly always very grave, and even when the disease is due to syphilis it should be guarded, though under antisyphilitic treatment improvement may be expected.

Treatment.—The treatment should be varied to meet the symptomatic indications. If syphilis is present, the iodonucleoid or the iodide of potash should be given in large doses. If a malignant growth is the cause, treat the unfavorable symptoms as they arise. If marked dyspnea is present from paralysis of the abductors on both sides, either intubation or tracheotomy should be performed.

BILATERAL ABDUCTOR PARALYSIS

Etiology.—The causes of bilateral abductor paralysis of the vocal muscles are syphilis, mediastinal tumors, aneurysm, and enlarged mediastinal lymphatic glands. Neurasthenia is also a cause of the paralysis.

FIG. 324

Bilateral paralysis of the thyro-arytenoidei interni and of the arytenoideus.

FIG. 325

Position of the cords when emitting a high-pitched tone and in abductor paralysis.

Symptoms.—The symptoms have been so admirably given by N. L. Wilson in an article read before the American Laryngological, Rhinological, and Otological Society, in 1900, that I will quote him:

"The patient gave a remote history of syphilis, and was somewhat addicted to alcohol; has had a few attacks of dyspnea, especially at night, for the past eight months. Voice only slightly husky, inspiration a little noisy, and expiration soundless. Occasionally had headaches. Ophthalmoscope showed nothing abnormal. Heart and lungs normal;

urine, acid and clear, specific gravity 1020. There was no albumin or sugar. The laryngoscopic examination showed the epiglottis to be normal, mucous membrane of the larynx normal, the vocal cords white, with a small slit between them during inspiration. The left vocal band was immovable in the median line; the right moved slightly." (Fig. 325.)

The patient was warned of the danger of sudden death from dyspnea, but refused to be tracheotomized. Three months later he died suddenly from dyspnea.

Fig. 326

Unilateral paralysis of the thyro-arytenoidei interni and of the arytenoideus.

Fig. 327

Paralysis of the thyro-arytenoidei interni.

Fig. 328

Bilateral paralysis of the arytenoidei.

Fig. 329

Unilateral paralysis of the right arytenoideus.

Fig. 330

Paralysis of the adductor muscles of the larynx. It also shows the position of the cords in deep inspiration.

Fig. 331

Paralysis of the adductors and arytenoideus.

Pathology.—When due to syphilis, the disease may affect the abductor muscles, the peripheral nerve filaments of the recurrent nerves, the nerve trunk, or the medulla. When due to mediastinal tumors, aneurysm, or enlarged glands, the recurrent trunk is pressed upon, causing atrophy or other degenerative changes in its nerve fibers. When due to neurasthenia, the flow of the nervous impulses through the recurrent nerve are inhibited.

Prognosis.—The cases of paralysis due to neurasthenia generally recover, though death may occur. When the paralysis is due to other

causes, more than half of the patients die. When operated upon, more than two-thirds recover. In the syphilitic cases the administration of the iodides and mercury sometimes effects a cure. When due to mediastinal tumors, aneurysm, and enlarged glands, it may be necessary to remove a portion of the vocal cords pending the consideration of the operation or other treatment of the mediastinal disease.

Treatment.—The faradic and galvanic currents have been used, and in but few cases with success. Antisyphilitic treatment has proved of value in a number of cases. Surgical treatment should be early recommended, as procrastination may lead to a fatal issue.

Surgical Treatment.—Three methods of procedure are available, namely: (*a*) Tracheotomy, (*b*) intubation, and (*c*) laryngofissure and the removal of a part or all of the vocal bands.

Tracheotomy is usually preferable, as it affords the least inconvenience to the patient and is ordinarily easily performed. The cyanosis, congestion, and edema of the tissues which sometimes complicate the case (A. G. Root) may, however, render this procedure difficult to perform. (See Tracheotomy.)

Intubation may be performed for the temporary relief of the dyspnea. It is not suitable for permanent relief, as the tube may be coughed up, and its use is uncomfortable to the patient.

Laryngofissure and the removal of a portion or all of the vocal cords may be practised if the tracheotomy tube is objected to. After this operation the vocal functions are sometimes gradually resumed. (See Laryngofissure.)

CHAPTER XXVIII

THE SINGING VOICE

The range of the average voice is from two to two and one-half octaves, although many singers embrace three to four octaves.

The singing voice begins from the third to the sixth year, and changes but little until puberty. At this time there is a great change, especially in boys, in whom it becomes deeper or lower in pitch, assuming more the quality of the voice of an adult male. There is some change in girls' voices, although it is not so noticeable as in boys. The larynx becomes larger, the cartilages consolidated, and the cords longer and thicker.

The vocal organs should not have special stress put upon them during this transition period, as coördination is distributed by the rapid changes in the shape, the size, and the position of the parts of the larynx.

Voice production is dependent upon three functions of the vocal apparatus. By "vocal apparatus" is meant the larynx (primary source of tone), the chest (source of motive power), and the resonant chambers of the chest and the head.

Without the motive power of the outgoing current of air through the larynx there could be no vibration of the cords, and without the vibration of the vocal cords and the outgoing current of air through the upper respiratory tract there could be no vibration or secondary tones or harmonics to enrich the laryngeal or primary tone. In other words, a voice, to be pleasing or "sympathetic," must have all the qualities which can be imparted to it by a proper respiratory act, a normal placement of the larynx, and unimpeded vibration of the vocal cords; also the richness or quality imparted to it by the resonance chambers of the chest and the head.

Defects of the singing voice are, therefore, largely due to the following causes:

(*a*) Improper methods of breathing.

(*b*) Improper action of the extrinsic and the intrinsic muscles of the larynx.

(*c*) Local disease of the larynx.

(*d*) Faulty or imperfect use of the resonance chambers of the head and the chest.

The nose is one of the most important resonant chambers, hence diseases or abnormalities in this region are especially productive of harm to the singing voice. The epipharynx, the soft palate, the uvula, and the tongue are also largely concerned in voice production. Growths or diseased conditions of the epipharynx, the soft palate, and the tongue are therefore potent factors in defects of the singing voice. Enlarged

tonsils, especially if cicatrices interfere with the movements of the pillars of the fauces, mar the purity of the tone and interfere with its placement. The same is true of postnasal adenoids. In both instances the mobility and the normal action of the uvula form a curtain or valve which regulate the volume and the direction of the vibrating air current from the larynx in its passage through the epipharynx and the nasal chambers. It is important that their action should be free and untrammelled. Postnasal adenoids push the soft palate forward and downward, while enlarged and adherent tonsils interfere with its free movement in an upward and backward direction toward the posterior wall of the pharynx. A voice thus modified loses its charm. Not only is the quality or timbre impaired, but the range is also curtailed. I could cite instances in which the quality has been improved and the range increased one to three intervals by the removal of the tonsils. As adenoids are more obstructive in children, they do not greatly affect the adult voice. On account of an associated postnasal catarrh with adenoids, the singing voice is often thereby indirectly affected. Postnasal catarrh involves the postsuperior surface of the soft palate and produces a laxity of the tissues composing it, including the palatine muscles. There is an increase in the fibrous tissue, together with an edema (slight) and boggy condition of the muscle fibers. The uvula is relaxed and often hangs down until it touches the base of the tongue or the posterior wall of the pharynx. This gives rise to a tickling sensation, and is often a source of annoyance to singers and speakers.

The presence of enlarged and diseased tonsils not only interferes with the muscular activity of the soft palate, but causes a chronic enlargement of the mucous membrane of the epipharynx and the mesopharynx, thus augmenting the catarrhal condition already mentioned. A very common symptom of tonsillar disease is a sensation of a splinter of wood lodged in the throat. This is a symptom which, so far as I know, has not heretofore been attributed to this condition. I have often noted it, and regard it as significant of cryptic infection.

Defects of the singing voice due to nasal diseases are chiefly due to an interference with the production of the harmonics or overtones which give quality and character to the voice. The bones of the face are so constructed that there are numerous cavities communicating with the nasal chambers. The lightness of the bones makes them admirable sounding boards for the primary tones of the vocal cords. It becomes apparent at once that any condition of the nose which interferes with the proper entrance of the column of air into the nasal and the accessory cavities will prevent the voice taking on the rich qualities of tone which make it pleasing to the human ear.

Deflection of the septum, thickening of the nasal mucosa from chronic catarrhal inflammation, polypi, and other morbid processes interfere with the resonant chambers of the head. The mucosa of the nose is reflected through the normal openings into the accessory sinuses, and is here affected by catarrhal or other thickening simultaneously with the invasion of the nasal membrane. The openings into the sinuses are more

or less closed by the thickening, and the resonant quality of the cavities is thereby diminished. More often the middle turbinal or a high deviation of the septum blocks the nose and affects the resonance of the voice.

Jean de Reszke has well said that the more he studies the voice the more he is convinced it is a question of the nose. I have for many years been impressed that the chief charm in a public speaker's voice is imparted to it by the nasal resonance. If this were lacking it failed to hold the attention of his auditors. I only speak of this to emphasize the fact that there is something very attractive to the average person in the resonance of nasal origin. There seems to be no other quality that can take its place. What is true in this regard of the speaking voice is doubly true of the singing voice.

The mouth influences the singing voice to a marked degree, not only in modifying the resonance, but, more particularly, in enunciation and articulation. The placement of the tongue, its concave-convex shape, with the tip elevated against the roof of the mouth, etc., modify the musical quality of the voice. Hence, all abnormal conditions of the tongue which interfere with its movements affect the voice. If it is "tongue-tied," adherent to the anterior faucial pillars, or the geniohyoglossus muscle is too short, the musical value of the voice is impaired. Hypertrophy of the tongue is occasionally an impediment to the acquirement of vocal excellence.

The larynx being the primary source of tone, it is natural to presume that most defects of the singing voice are due to some lesion or faulty method of using it. This is probably true, although it should be remembered that many of the laryngeal inflammations are indirectly the result of nasal disease. Chronic laryngitis and, in many instances, acute laryngitis are secondary effects of chronic nasal obstruction and catarrhal sinuitis. Recurrent or persistent hoarseness should, therefore, lead to a thorough inspection of the nasal chambers for obstruction or diseases of the sinuses. Hoarseness is not necessarily a sign of an antecedent nasal disease, as it is also a prominent symptom of laryngeal tuberculosis, cancer, etc.

Papillomata or other *laryngeal neoplasms* interfere with the motility and the adjustment of the vocal cords, and thus produce hoarseness, aphonia, or spasm of the muscles of the larynx. Morbid growths in this region should be removed with great care and with due regard to the functional integrity of the vocal apparatus. Awkward or aggressive surgery might forever banish the possibility of a musical career, or even a voice for ordinary social purposes.

Any of the various forms of laryngeal paralysis described in the previous chapter will, of course, impair or entirely destroy the singing voice.

Methods of Breathing.—**Defects of the Singing Voice Due to Improper Methods of Breathing.**—To obtain the purest and richest singing voice, the method of breathing should be carefully cultivated. The natural method of breathing is not suitable for the singing voice (H. Curtis). It is adapted to the ordinary function of oxygenating the blood, but is poorly suited for singing. For this purpose the respiratory acts should

be done in such a way as to give the most perfect control over the expiratory current, and at the same time maintain the same quality or tone of the voice during the varying stages of the act.

In order to obtain the most *perfect control* of the expiratory current of air for artistic purposes, the respiratory method should be such as will give the greatest chest capacity, as well as full control over the emission of the air for phonatory purposes.

The *quality* or *timbre* is best maintained throughout all the registers by such a method as will keep the upper portion of the thorax in a fixed position.

The *control of the expiratory current* for artistic purposes is a complex coördination of the muscles of the chest walls (scaleni and intercostals), the diaphragm, the abdominal walls, and the larynx. The singer should not, however, be made conscious of the part the larynx plays in this capacity, as this would lead to an undue tension of the laryngeal muscles. Nothing could be more damaging to the quality of the voice than this. In fact, the larynx has but an infinitesimal muscular function in voice production. The singer should be made to understand clearly that only when the laryngeal muscles are at "ease" can the voice charm the listener. The auditory nerve should only be conscious of quality, richness, sweetness, fulness, splendor, unlimited reserve, and all the emotions that make the inner self a free spirit, travelling through the world of ennobled thought and imagination. The most beautiful song, when coming from an overtense larynx, calls attention to the material, the singer, as opposed to the ethereal, the song, thus defeating the purposes of artistic singing.

I have thus digressed at this point in order to emphasize the importance, indeed, the absolute necessity, of maintaining a proper poise of the laryngeal muscles during the artistic activity of the expiratory current of air with which the singing voice is produced.

The Inferior Costal Type.—The *chest cavity* is conical in shape, with the apex at the top. It may be increased in all its diameters during the inspiratory act by the action of the scaleni, the intercostals, and the diaphragmatic muscles. All these muscles should, therefore, be used to fill the lungs to their greatest capacity. The inferior intercostals and the diaphragm are especially important for this purpose, hence it is usually spoken of as the inferior costal type. The upward and outward movement is chiefly confined to the ribs and the sternum below the sixth rib. The downward movement of the diaphragm pushes the abdominal viscera with it, and thus tends to increase the abdominal convexity. The experience of the great artists has shown that the lower portion of the abdominal walls should not be allowed to participate in this distention, as the perfect control of the expiratory current is thereby hindered. The lower portion of the abdominal wall should, therefore, be retracted, while the upper portion is allowed to distend.

The upper chest wall should be maintained in the position it assumes during deep inspiration. That is, during expiration it should remain fixed in the position assumed during deep inspiration. In this way the

resonance imparted to the voice by the thoracic cavity is increased and maintained of the same quality throughout all the registers of the voice. Failure thus to fix the upper chest wall will result in the voice taking varying tonal qualities as it passes from one register to another. I have heard singers whose voices were rich in quality in the middle register, but in passing into the upper or the lower register, assumed an entirely different quality. This change is not always due to a failure to fix the upper chest wall as described, as it may also arise from improper placement of the soft palate. Nevertheless, it is important that the upper wall of the thorax should be maintained in the position assumed during deep inspiration.

The inferior costal or artistic type of breathing may be analyzed as follows:

(*a*) It is chiefly performed by the inferior portion of the chest walls and the diaphragm.

(*b*) The upper abdominal walls also participate in the outward expansion.

(*c*) The inferior abdominal walls are maintained in a retracted position during inspiration and expiration.

(*d*) The upper chest walls are maintained throughout inspiration and expiration in the position assumed during deep inspiration.

The effects sought for are:

(*e*) The greatest chest capacity.

(*f*) Perfect control of the expiratory air current.

(*g*) A maintenance of the same resonant quality throughout all the registers.

Factors Which Influence the Voice.—Deviation from the foregoing type of breathing during the act of singing are detrimental to the artistic qualities of the voice. It is true that some of the greatest artists do not use this method of respiration. What their voices would have been had they used this method can only be conjectured. There are so many elements entering into the composition of a great artist, that a fault in one direction may be obscured or compensated for in other ways. For instance, an artist may use superior costal breathing and overcome in a large measure any defect of the voice resulting therefrom by the brilliancy of vocal execution or by the transcendent spiritual or mental conception which dominates the mind and the body during the singing. There is no shadow of doubt as to the transforming power of an exalted or overmastering conception of the part being rendered. This alone does not make one a great artist. The physical mechanism whereby this conception is expressed, should be so coördinated and adjusted as to not detract from its full expression.

The Vocal Resonators.—The voice, like musical instruments, has its sounding board. The sounding board of the piano and the violin are familiar to all. If the string of a violin were stretched upon a heavy slab of marble the tone given off would be weak and disagreeable. It would lack the overtones or harmonics which make it rich and grateful to the ear. The same string when adjusted on a violin gives forth a tone of

great sweetness and power, as the sounding board adds numerous overtones to the fundamental tone of the string. The fundamental tone predominates while the harmonics coördinate in such a way as to give it "color" or timbre.

What is true of the violin string is also true of the vocal cords. The fundamental tone is weak and thin, but it is enriched by the harmonics of the resonance chambers of the chest and the head.

The resonance chambers (sounding board) of the head are: (*a*) The ventricular pouches; (*b*) the pharynx; (*c*) the epipharynx; (*d*) the nares; (*e*) the accessory nasal cavities; and (*f*) the mouth.

The resonance from the chest has been referred to under Methods of Respiration.

The ventricular pouches do not, perhaps, play an important role in the production of overtones. The pharynx (including the epipharynx) communicates with the mouth and the posterior nares. The soft palate acts as a valve or curtain which regulates the amount of the vibrating current of air going to the nose and mouth. In this way the quality of the resonance is regulated to suit the musical expression of the singer. The soft palate is, therefore, an important part of the vocal apparatus. If it is elevated against the posterior wall of the pharynx, the voice assumes a peculiar and objectionable quality known as *throatiness*, a condition also assisted by the elevation of the posterior portion of the tongue (H. Curtis).

The soft palate is prolonged downward in two pairs of folds known as the pillars (palatine arches) of the fauces.

The anterior pillar contains the palatoglossus (glossopalatine) muscle, while the posterior pillar embraces the palatopharyngeus (pharyngopalatine). They assist in the modulation of the voice by coördinating with the movements of the soft palate. The function of the uvula is not well understood.

The *faucial tonsils* lie between the pillars, and when enlarged or diseased, affect their motility and impair the voice. They often become adherent to the sinus tonsillaris and thus very materially interfere with their action. I have no hesitancy in indorsing the opinion of Sir Morrell Mackenzie, H. Curtis and others who advocate their removal in adults when they give rise to the slightest trouble. Curtis says their existence in the adult is unnecessary, as they serve no good purpose. When we remember that in childhood they are composed of lymphatic tissue, to meet the exigencies of the infectious fevers to which childhood is so susceptible, and that in adulthood they are usually fibrous from repeated and long-continued inflammation or irritation, it is easy to understand why they no longer serve any useful purpose.

If the pillars are adherent to the tonsils, they should be freed, and in most instances this should be followed by complete ablation of the tonsils. (See Operations of the Tonsils.) The immediate effect of their removal is sometimes detrimental to the voice. After a few weeks this passes away and the voice begins to show the value of the procedure. At first the loosened pillars may relax and fail to perform their muscular

function. After a few weeks they become attached to the fibrous tissue formed in the sinus tonsillaris, and perform their functions in a much better manner than before the tonsillectomy. Sir Morrell Mackenzie says he has never seen any other than beneficial effects to the voice follow their removal.

The *pharynx* is supplied with numerous lymphatic masses, especially near its vault and along the lateral walls. The enlargement of the lymphatic tissue in the vault is commonly known as *postnasal adenoids*, while that along the lateral walls of the pharynx is called *pharyngeus hypertrophica lateralis*. When the scattered masses over the posterior wall of the pharynx are diseased and enlarged, the condition is known under various names as follicular pharyngitis, granular pharyngitis, or "clergymen's sore throat."

Adenoids are not commonly present in adults, although they may be. Many children, however, have marked defects of the voice from their presence. The resonance is interfered with by the obstruction in the epipharyngeal space and the entrance to the nares. The soft palate is crowded forward and downward by them. The voice has a dead or so-called "nasal" quality, which in reality is an absence of nasal resonance. In other words, the nasal chambers are the chief resonators of the voice. It is obvious, then, that adenoids are an absolute hindrance to the singing voice. The treatment is their complete removal (see Adenoids).

Hypertrophica lateralis impairs the voice by perpetuating a chronic irritation and congestion of the parts, including the larynx. The voice becomes husky and the muscles of the larynx tire upon slight or moderate singing. The hypertrophic glandular masses should be removed.

"*Clergymen's sore throat*," or chronic pharyngitis, is, according to Sir Morrell Mackenzie, the most common cause of trouble to singers, the voice becoming husky and tiring upon slight use. Just behind the soft palate the muscles of the posterior pharyngeal wall contract in coördination with those of the soft palate, and aid in closing or constricting the pharynx at this point. Resonance is, therefore, modified by the existence of inflammatory disease of the pharynx, as the muscles of the pharynx and the soft palate are edematous and somewhat restricted in their movements.

Chronic pharyngitis is accompanied by a similar affection of the posterior wall of the soft palate and the uvula. A relaxed or *elongated uvula* is nearly always a sign of chronic epipharyngitis. The practice of amputating the uvula under such circumstances should not be done without first attempting to cure the preëxisting pharyngitis.

The *tongue* performs an important function in regulating the resonance chamber of the mouth. If there is a shortening of the geniohyoglossus muscle, or an hypertrophy of the entire tongue, this function is impaired. I have frequently seen the tongue adherent quite high on the anterior pillars of the fauces. This not only interferes with the correct movements of the tongue, but with those of the anterior pillars also. In one case of this kind, where the tonsils had been completely removed by cautery dissection, hoarseness became a troublesome factor.

Lingual tonsils and *varicosities* sometimes give rise to hoarseness and a web-like feeling in the larynx.

"Tongue-tie" interferes with the proper performance of the glossal function, especially in articulation.

The *absence of some of the front teeth*, or even marked *irregularity* of the same, might also interfere with resonance and articulation in singing.

Cleft palate (either hard or soft) would, for obvious reasons, interfere with both resonance and articulation.

The Nasal Chambers.—As these are the chief resonators or sounding boards of the voice, special attention should be directed to their condition in searching for defects of the singing voice. This is of special importance in view of the fact that many pharyngeal and laryngeal affections are caused by preëxisting disorders of the nose.

The nose is divided into two cavities by the nasal septum, and these cavities are still further partially divided by the turbinated bodies. The lateral walls of the nares are in communication with numerous air cells or sinuses which communicate with the nasal chambers. Above the nose they open into the frontal sinuses, while posteriorly they open into the sphenoidal sinuses. Thus the bones of the face form numerous bony chambers which make up the chief sounding board of the vocal apparatus. At least it is this portion of the resonance apparatus that gives the voice its sympathetic and attractive quality. I would not minimize the importance of the chest and other resonance chambers, but I would emphasize the importance of the resonance chambers of the nose.

Defects of the Singing Voice from Improper Methods of Respiration.—While there can be no well-defined analysis of the defects due to improper methods of breathing, there can, nevertheless, be a classification which will emphasize the underlying principles. The following is given for this purpose rather than to catalogue a series of defects:

(*a*) Superior costal breathing does not use the entire thoracic capacity, hence the voice does not possess the reserve force and the evenly sustained quality afforded by the *inferior costal type of breathing*.

(*b*) The same may be said of the abdominal type of breathing with even greater emphasis. The resonance is less pronounced than in either the superior or the inferior costal type, while the control of the expiratory breath is jerky. The voice is thereby rendered uneven and less sympathetic in quality.

(*c*) On account of the greater difficulty in controlling the expiratory breath, the extrinsic and the intrinsic muscles of the larynx are put upon a tension in an involuntary attempt to compensate for the lessened control of the thoracic and the abdominal muscles. This at once impairs the artistic qualities of the voice and in some cases almost destroys its singing qualities. The voice becomes rough, metallic, unsympathetic, and forced. The laryngeal muscles tire easily, and prolonged singing is an impossibility. There is a feeling as of a web across the cords. Frequent ineffectual attempts are made to clear the throat.

The foregoing symptoms may be present in so slight a degree as to escape notice, or they may be so severe as to ruin the voice.

The inferior costal or artistic type of breathing, if intelligently and faithfully practised, will avoid these difficulties and add materially to the power and attractive qualities of the singing voice.

Defects of the Singing Voice Due to Tone Blindness.—J. Mount-Bleyer has called attention to a condition of the hearing centres of the brain which is neither a disease nor a defect, but is the result of inattention or lack of training. For instance, some hear an orchestra as a whole, while others distinguish the tone of each instrument; still others distinguish the exact musical quality of each instrument. The difference is not so much in the mechanism of hearing as it is in the training which the brain centres have received. One, through a love of music, seeks for the finer qualities and variations, while another casually receives only the most general impressions from music. In the first place, there is eager, expectant attention, while in the latter there is an indifferent, passive attention. It cannot be said that one has a good ear and the other a poor ear. Each may have equally good ears, or the one hearing the less may have the better. One, however, has a cultivated brain centre, which enables him to distinguish tones and qualities unnoticed by the other. Suitable training of mechanically perfect "ears which hear not," and "ears that hear and hear not," would rapidly convert them into highly discriminating organs of hearing.

We often hear the remark, "I do not sing because I have no ear for music." In other words, he sings poorly because he has not educated the so-called ear to a full appreciation of musical intervals, rhythm, and the other qualities which make music so attractive. His belief is that his ears are defective as to musical matters, while the opposite may be true. The whole matter may be summed up in the statement that his "ears" have not been educated.

J. Mount-Bleyer refers to Mr. Evans' work as superintendent of singing in the London schools, where he has 300,000 pupils under his direction. In no instance of obstinate inability to distinguish one sound from another has he failed to educate them to appreciate such distinctions. This fact is significant, and should encourage those interested in the cultivation of the voice to give more attention to the exact education of the "ear."

Treatment.—I will here briefly outline the method of procedure used by M. Duchemin, director of music in the asylums of Paris:

"M. Duchemin, setting aside all ideas of notations, commences by demonstrating to the pupil, by means of any musical instrument whatever, the interval of a note and that of a half-note. When the pupil has been sufficiently instructed in the distinction of these intervals, he makes him listen to the interval of a note and to that of a major third. He next makes him compare the major third with the fourth, and thus successively all the major intervals of the same octave. He then returns to the point from which he started, and makes him compare the major with the minor intervals. When the pupil is acquainted with all the ascending intervals, he then repeats all the intervals, but in the descending scales. Finally, when the pupil has compared all the intervals by twos and twos,

M. Duchemin makes him listen to isolated intervals, either ascending or descending, at first to those comprised within a single octave, afterward to those within two octaves, and so on." (J. Mount-Bleyer.)

I have recently tried this method in a few cases where the claim was made that they "had no ear for music," with gratifying results. The quickness with which they learned to differentiate between the various intervals was surprising to me. Both vocal and instrumental music, including the orchestra, assumed a new and delightful place in their lives. I would, therefore, urge that further attention be given to this part of the subject.

It is not within the province of this work to speak of methods of teaching, except in so far as they may apply to the defects of the singing voice. I cannot refrain, however, from the remark that, in my judgment, M. Duchemin's method of procedure might be used with greater advantage in both vocal and instrumental instruction as a preliminary training in musical education. Public schools, conservatories of music, and private teachers might, with great advantage to their students, follow this method. As music is made up of these intervals arranged in varying rhythm, periods, and sequence, it is of primary importance that the ear be trained to recognize them readily. This is all the more apparent when we remember that only when sensory impressions become intimate parts of one's experience can they be reëxpressed with power and beauty. An "ear" trained in this way will not only hear the music of others more accurately, but its possessor will be able to render music more accurately himself.

I wish here to consider a few of the more common conditions which impair the singing voice.

Laryngitis of a subacute or chronic type is one of the most frequent derangements of the vocal apparatus to be found among singers. It renders the voice slightly rough or hoarse, and in extreme cases aphonic. The impairment is not constant, but comes and goes with the changes of the weather or with fatigue and use of the voice. Its tendency is to become more and more fixed with each recurrence. The etiology may be embraced in an antecedent nasal disease, an improper use of the laryngeal apparatus, or in some general condition which lowers the vital energy. If it is due to the first, the nose and the epipharynx should receive appropriate attention, with a view to restoring their respiratory functions. Nasal obstruction, chronic sinuitis, etc., should be treated according to the descriptions given elsewhere in this work. The hoarseness may be due to an improper use of the vocal apparatus; the faulty method should be detected and corrected if possible. Six years ago a lady consulted me concerning her throat, stating that she was a student of vocal music, and that after moderate use of the voice she became slightly husky, there being a sensation of a web over the cords. Upon examination of the nose and throat, I could detect no apparent cause for the condition. I found her, however, to be quite "high-strung," and asked her to go through some of her exercises in my presence. It was quite apparent that the whole muscular system, including the larynx,

was of a "high tension." As she was a woman of culture and intelligence, I explained to her the necessity of overcoming this overtension, and offered her some suggestions as to how to do it. She was told to assume a natural and comfortable position in the chair, and to allow her arms, including the hands, to drop at her sides in extreme relaxation. She was then to allow the whole body, including the tongue and the lower jaw, to participate in the relaxation. Next she was to hum very softly the note that came naturally to her throat. After she had gone through with this exercise for a few minutes, the vocal exercise was varied by singing the tones within a range of one-half octave, cautioning her all the time to maintain extreme relaxation of the whole body. The exercises were gradually broadened to those she was in the habit of singing, the difference being in her physical condition during their production. In a surprisingly short time she thus trained the extrinsic and the intrinsic muscles of the larynx to a normal tension, which not only caused the hoarseness to disappear, but resulted in a placement of the larynx which gave added richness to her voice. There were poise and dignity in it, which were hitherto undeveloped.

I do not mean to imply that all persons suffering from "high tension" can be made to sing beautifully, but I do want to say that many singers who become hoarse from overtension of the laryngeal muscles may be speedily and effectually relieved of the hoarseness and other tension anomalies of the voice by suitable advice and vocal exercises. The *manner* of going through with the exercises should be emphasized.

If the hoarseness is due to some general *systemic disturbance* which results in laxity of the cords or the laryngea mucosa, remedies suited to the case should be given.

CHAPTER XXIX

DEFECTS OF SPEECH

Defects of speech are due to a great variety of causes, most of which are extralaryngeal. The larynx is the primary source of spoken tones, but it is not the complete vocal apparatus. It has been customary, in times past, to speak of it as the vocal organ, but this can no longer be done in strict conformity to well-known facts concerning voice production. While the vibrations of the vocal cords produce the primary tone, it is much modified by the chest, pharynx, epipharynx, nasal and accessory chambers, tongue, and the mouth. The character of the tone is also somewhat dependent upon the respiratory movements of the chest, abdominal muscles, and diaphragm. The voice changes when there is a marked increase in the physiological activity of other parts of the body, as at puberty. This is especially noticeable in boys. Mental states exert a marked influence on the quality of the voice, as may be noted in anger, joy, hatred, and love.

It is, therefore, apparent that defects of speech may have their origin in parts remote from the laryngeal apparatus. The demands of domestic and social life often make it important that one possess a voice that is pleasing in timbre, range, pitch, and modulation, as well as in articulation. Hence, attention should be directed to some of the more important lesions which impair the quality and integrity of speech.

Speech and Brain Development.—That there is an intimate connection between the development of the organs of speech and the cerebral centres of intelligence is, I think, scarcely open to question. This is especially true in children. I have seen them four years of age, apparently as bright and intelligent, with the exception of speech, as other children of the same age. They had reached the age at which spoken language should be used to communicate their wants and express their ideas. If it is not acquired within a reasonable length of time, they are in danger of becoming mentally inferior to other children of the same age. That this inferiority is not altogether due to their inability to acquire knowledge through the senses, and through the natural inquisitiveness of childhood, has been shown by various writers who have reported remarkable increase in the mental development in children who were only trained to use the muscles of articulation, not yet having been led into the realm of thought in which information concerning things and affairs is inculcated. Makuen, of Philadelphia, reports cases in which the simple training of the muscles of the mouth, tongue, and fauces aroused the dormant faculties of the brain. The use of the motor tracts, of the muscles of speech, stimulated the centres of speech and thought, and

the patient passed rapidly from a "backward child" to one of ordinary intelligence.

I will not at this time consider fully the interdependence of the organs of speech and mental development, but will only thus briefly refer to it in order to emphasize the importance of slight impediments of speech in children who are of the age at which language is most naturally acquired. It is obvious that an impediment at this time is a much more serious hindrance than it is after speech has been acquired. It is very much easier for him to cover up or compensate for a defect in the organs of speech, if the faculty of speech has been already acquired, than it is if that faculty is not developed. Hence, abnormalities of the organs of speech, which develop after speech has been acquired, result in but slight defects of speech; whereas abnormalities of a similar nature, in a child who has not yet acquired the faculty of speech, will in some cases prevent the acquisition of spoken language, while in others it will only interfere with it to such an extent as to make it defective. If this were the extent of the damage done, it might be passed over with comparative indifference; but, as I have already suggested, mental development is also hindered. I have no doubt that a considerable number of the so-called "backward children" coming under this category are so chiefly on account of a slight physical imperfection of some part of the organs of speech. I do not mean to say that all "backward children" come under this classification, as no doubt many of them are defective in cerebral development from quite different causes. I only wish to call attention to the fact that each case should be carefully studied, the physical impediments to spoken language corrected, and suitable training of the organs of speech instituted, in order to give the child the best possible chance of taking the position in society to which he was born.

An analysis of the peripheral causes of the defects of speech is interesting as well as instructive, especially to those who meet them in practice, or at least to those who attempt to treat them. Defects of speech are subdivided into six varieties by R. Cohen, of Vienna, as follows:

1. Stammering.
2. Stuttering.
3. Nasal twang.
4. Defects due to malformations of the hard and soft palates.
5. Deaf-mutism.
6. Defects of speech due to diseases of the central nervous system.

Instead of following the classification given by Cohen, the author will treat the subject under the following heads:

1. Defects of speech of nasal origin.
2. Defects of speech of epipharyngeal and faucial origin.
3. Defects of speech of lingual origin.
4. Defects of speech of laryngeal origin.
5. Defects of speech of thoracic and abdominal origin.
6. Defects of speech due to deaf-mutism.
7. Defects of speech due to malformations of the palate.
8. Defects of speech of central origin.

1. **Defects of Speech of Nasal Origin.**—The etiology may be: (*a*) Deflection of the septum. (*b*) Spurs or ridges on the septum. (*c*) Split or double convexity of the septum from an old traumatic lesion or abscess. (*d*) Nasal polypi or other neoplasms. (*e*) Chronic turgescence of the inferior nasal conchæ. (*f*) Hypertrophy of the inferior nasal conchæ. (*g*) Hypertrophy (mulberry) of the posterior ends of the inferior and middle conchæ. (*h*) Congenital occlusion of the posterior nares. (*i*) Displacement of the columnar cartilage. (*j*) Enlargement of the middle conchæ from hyperplasia or cystic degeneration. (*k*) Obstruction to the olfactory fissure.

The foregoing conditions do not cause great defects of speech, as they only interfere with the resonant quality of the voice. Nor do they materially interfere with the muscular mechanism of speech production.

In a general way, they may be said to produce those changes in the voice which make it "dead," "muffled," "thick," "flat," or lacking in resonance. The speech is still further modified by diffidence, which so often accompanies nasal obstruction. The diffidence, backwardness, or timidity is due to a self-consciousness, to which the defect gives rise, and to a direct effect upon the brain and general system, through the lymphatic and venous stasis attending nasal and postnasal obstruction. Guye, of Amsterdam, has called attention to a condition which he calls "aprosexia," or difficult attention.

Inability to fix the attention is often attended with diffidence and timidity, and not only is articulation impaired thereby, but fluency and coherency is also somewhat affected.

The elementary sounds of spoken language which depend largely on the resonance of the nasal chambers are not so markedly impaired as those but slightly depending upon it. For instance, the letters *m*, *n*, *b*, and *d* derive their peculiarity from the initial sound, while the final vowel and nasal tones are secondary. Notwithstanding the fact that they are secondary, their absence or suppression makes a noticeable change in the speech, and amounts to a defect. If the final vowel-nasal sound in the above examples were more prominent, the nasal obstruction would not interfere with speech nearly so much, as the speaker could "force" them, and thereby somewhat overcome the apparent effects of the stenosis. The letters *m* and *n* end in a kind of "hum" which is very difficult to produce when nasal obstruction is present, especially when the hum is somewhat suppressed.

The letters *b* and *d* seem to begin with the sound thrown forward against the lips (*b*) and against the tip of the tongue and roof of the mouth (*d*) respectively. The initial sound is, however, made in the larynx and rendered resonant in the chest and nasal chambers. Nasal obstruction modifies the resonance, thus causing a "dead" or "flat" tone to explode at the lips or the tip of the tongue. Thus the speech is rendered defective. We might continue the analysis of the various sounds in speech, showing how nasal obstruction, from one or more of the foregoing conditions, affects the beauty, music, rhythm, and coherency

of speech. We might go still farther and show that coherency of thought is impaired also.

2. **Defects of Speech of Epipharyngeal and Faucial Origin.**—These may be caused by the following: (*a*) Postnasal adenoids. (*b*) Fibroma or other neoplasms of the nasopharynx (epipharynx). (*c*) Chronic catarrhal thickening of the mucosa of the epipharynx. (*d*) Hypertrophied or hyperplastic faucial tonsils. (*e*) Adhesions of the anterior and posterior pillars of the fauces to the tonsils. (*f*) Depression of the soft palate against the root of the tongue by the postnasal adenoids. (*g*) Paralysis of the palatine muscles, especially those of the membranous curtain which control the current of air passing to the nares. (*h*) Paralysis of the soft palate and uvula. (*i*) Adhesion of the anterior faucial pillars to the base of the tongue. (*j*) Cleft soft palate and uvula. (*k*) A shortened soft palate, as is sometimes found after operation for cleft palate.

In the above table the muscular mechanism of speech is affected, and the defects of speech are correspondingly more pronounced. The explanation of the more marked defects which seem to have their origin in this classification is not as easy as may appear on first thought. We cannot say that the speech is defective because the muscular action of the parts is interfered with, because many cases come under our observation in which there is great muscular impairment but little impediment of speech, while others can scarcely be said to have articulate speech at all; and in still others they cannot be said to have coherent thought. The explanation in some cases is that the muscular impairment existed quite early—before articulate speech was acquired. The impediment thus interfered with the acquirements of articulate speech. The presence of postnasal growths produced mental hebetude (aprosexia), heretofore referred to, and the mental ability to acquire articulate speech and consecutive thought was thus impaired. In a few years the growing child becomes more vigorous in mind and body, and makes renewed and voluntary efforts at articulate speech. His failures humiliate and irritate him. He avoids the necessity of speech as much as possible. The speech centres and motor vocal tracts are little used and lie dormant. His mental growth is thereby retarded. The sensitive, reticent child loses the mental growth to be gained by spoken language. He becomes and is regarded as a "backward child."

It becomes the duty and privilege of the rhinologist and laryngologist to loosen the bonds which fetter his imprisoned mind, thus enabling him to enjoy the common pleasures of life, even though he may never become a brilliant member of society.

3. **Defects of Speech of Lingual Origin.**—The causes may be: (*a*) Inflammatory adhesions binding the tongue to the anterior faucial pillars and epiglottis. (*b*) A congenital shortness of the geniohyoglossus muscle. (*c*) Tongue-tie. (*d*) Enlargement of the tongue. (*e*) Excessive enlargement of the lingual tonsils.

Of the foregoing, the most important are adhesions of the tongue to the anterior faucial pillars, tongue-tie, and shortening of the genio-

hyoglossus muscle. Either condition materially interferes with the articulatory function of the tongue, thus impairing speech. Lisping is a common sign in these conditions. If these lesions exist prior to the acquirement of speech, they may give rise to the clinical picture heretofore referred to under "backward children." The early correction of these physical imperfections may place the child on an equal footing with his fellows, and save society the disagreeable presence of a crippled mind in its midst.

4. **Defects of Speech of Laryngeal Origin.**—The etiology may be: (*a*) Too great strength in the uplifting muscles of the larynx. (*b*) A weakness of the down-pulling muscles of the larynx. (*c*) Laryngitis. (*d*) Chorditis nodosum. (*e*) Tuberculous inflammation and infiltration. (*f*) Perichondritis. (*g*) Laryngeal rheumatism. (*h*) Catarrhal accumulations. (*i*) Neoplasms. (*j*) Paralysis of the intrinsic laryngeal muscles.

If the acute affections of the larynx, as laryngitis, and the chronic conditions, such as chronic laryngitis, laryngeal tuberculosis, perichondritis, paralysis, rheumatism, and neoplasms which cause hoarseness or aphonia, are omitted, there is little to catalogue as causes of defects of speech. This is the more surprising when we recall the fact that the larynx is the primary source of the voice.

Makuen has referred to a condition of the extrinsic muscles of the larynx which rendered the voice sibilant and *falsetto*. It is given in the table above in *a* and *b*, and is interesting because it illustrates one of the fundamental problems in voice culture, namely, voice placement. If the larynx is allowed to rise too high, the voice becomes *falsetto* and unnatural in quality. If, on the other hand, the laryngeal box is held down in its proper position, the voice assumes its natural register, the tone being pure and pleasing to the ear—that is, it is natural.

The natural and simple things of life appeal most strongly to normal minds. The simple rural scenery, the grandeur of the mountains, the simple melodies of the negroes, the rugged vitality of the Wagnerian opera, and the eloquence of the orator stir the imagination, quicken and fascinate the mind, as the unnatural, the complex, and the artificial cannot do.

Hence, the aim should be to give those having defective speech a speech that is simple and natural. It should be natural in quality, tone, pitch, *timbre*, and rhythm, as well as in modulation and articulation.

5. **Defects of Speech of Thoracic and Abdominal Origin.**—The causes may be: (*a*) Pulmonary tuberculosis in its relation to stammering. (*b*) Irregularity of the respiratory rhythm.

Irregularity of the respiratory movements is an almost constant factor in stammerers. Whether this is due to some fault of the respiratory centre, or to some peripheral lesion, has not yet been determined. Makuen has called attention to the fact that all, or nearly all, stammerers are either tuberculous, or come from families with this disease well marked in its history. He thinks the peripheral tuberculous lesion

accounts for the irregularity of the respiratory rhythm, which in turn causes the stammering.

His conclusion is not necessarily correct, as the lack of rhythm may be due to developmental causes within the medulla, or along the motor nerve tracts leading to the diaphragm, lungs, and intercostal muscles. It is a well-recognized fact that those having a tuberculous tendency, especially those inheriting it, have a lowered cellular vitality, and that nutrition, or the processes of metabolism, are imperfectly performed. It is therefore possible to explain the lack of respiratory rhythm as being the result of the malnutrition and faulty development of the respiratory centre and the motor respiratory tracts.

Whatever the explanation may be, the clinical fact remains, that nearly all persons who stammer are of tuberculous parentage and complain of ill health. Another fact, however, which makes it seem probable that the lesion is peripheral (in the lungs and diaphragm) is that under suitable treatment and training they may be freed from the defect.

La Fayette Page calls attention to intoxications arising from diseased conditions of the upper respiratory tract. He cites the work of Schwalbe and Retzius, who demonstrated the connection of the lymphatic vessels of the nasal mucous membrane and those of the cranial cavity. Through the lymphatic and venous stasis of the nasal mucous membrane, the effects extend to the cranial cavity, thus giving rise to mental dulness.

He also cites the intimate nervous connections between the nasal mucous membrane and the cortical centres of the brain as a possible source of mental dulness and irritability.

Makuen in his writings seems to lay greatest stress on impairment of the organs of speech, as the larynx, fauces, nose, or tongue, as the chief hindrance of mental growth and development.

In the opinion of the author, defects of speech and mental acumen are due to complex conditions which it would be difficult to define. It appears, nevertheless, that children who are defective in speech are improved by correcting, either surgically or by training, the physical impediments to speech. We also know, from clinical observation, that upon the removal of postnasal adenoids or section of the geniohyoglossus muscle, etc., the mechanism of speech and the mental activity of the child are often much improved. Those who hold, as Guye and Page, that the mental quickening is due to the removal of the cause of the venous and lymphatic stasis, overlook the fact that the mechanism of speech is at the same time improved. The soft palate which was crowded down against the base of the tongue is freed, or the tongue is loosened, and resumes its normal function in articulate speech. Again, those who hold the views of Makuen to the exclusion of all others overlook the fact that the venolymphatic stasis, with its attendant toxemia and brain hebetude and irritability, is overcome and allows the brain to resume its normal activity.

It should not be forgotten that the toxemia referred to by Page affects the system much deeper than the brain. The whole system is poisoned, as has been shown by the author in various articles on mouth breathing.

There may be great imperfection of speech without impairment of the mental faculties. Nevertheless, it must be said that in nearly all cases "the speech belieth the man."

Elegance of speech is an index of a finished mind. Training the organs of speech improves not only the expression of thought, but the thought itself is more elevated, more finished. The quality of mind is improved by a better mode of expression.

6. **Defects of Speech Due to Deaf-mutism.**—This subject is quite fully considered under deaf-mutism, and will only be briefly analyzed here. It may be caused by:

(*a*) Congenital defect of the auditory apparatus.
(*b*) Acquired defect of the auditory apparatus.
(*c*) Nasal and epipharyngeal diseases.
(*d*) Improper and untimely training.
(*e*) Lack of training.

Congenital defects of the auditory apparatus are probably present in about one-half of the cases of deaf-mutism, whereas in the balance the defect is due to the ravages of some disease, usually one of the exanthematous fevers. In either instance the child is partially or totally deaf, and cannot, therefore, readily acquire the faculty of speech. He is not mute because the organs of speech are defective, nor because the centres of speech are impaired. Both the peripheral organs of speech and the central mechanism of the brain may be in perfect condition. The child is mute because he cannot hear others speak, and is thereby deprived of the most useful aid in learning—namely, imitation. If he learns to speak, he must be taught by other and more difficult methods. He must be given timely and proper special training. If he has acquired deaf-mutism after having some ability to speak, he may not be a mute in the full sense of the word, but may need some special training to prevent his losing the little speech he already possesses. If the deafness comes before the seventh year of age, there is a strong tendency to lose the faculty of speech; hence, special training is necessary to maintain that already acquired, as well as to broaden it. If the deafness comes on after the seventh year, the patient rarely loses the faculty of speech; hence, his training can be more simple than that of a child losing his hearing before that age.

Reference has been made under Deaf-mutism to the interdependence of the brain development and the use of the organs of speech. Brain development and intellectual growth depend largely upon the voluntary use of the organs of speech. It is a common observation with most of us that an idea or train of thought is much clearer after having been expressed in words. The growth of the brain seems to depend upon the coöperation of the various senses and the peripheral organs. The intelligence of the child will, therefore, largely depend upon the use of its vocal apparatus, as well as all the other peripheral organs of the body.

At certain ages the various faculties of the brain develop most naturally, and these periods should be taken advantage of by his instructors. At one time the imagination, which later in life finds expression in so many

practical ways, has the ascendancy. The training at this period should be of such a character as to lead the imagination along wholesome lines. It should be bridled, but not suppressed. When adulthood is reached, and the practical affairs of life must be faced, the faculty once known as imagination is utilized in foreseeing the outcome of a given series of events. Cause and effect, and the sequence of events, will be correctly interpreted, somewhat in proportion to the character of the training received during the imaginative period in childhood.

The other faculties of the mind should also receive due consideration in the training of the child. The child that is deaf needs this training tenfold more than the one with normal hearing. It becomes obvious, therefore, that the deaf mute needs a teacher well schooled in the knowledge of the child mind, that he may facilitate its unfolding in the most wholesome and natural manner. *Not one mother in ten thousand* is fitted for this task, and even if she were, her love for the child would probably make her its worst enemy, in so far as its proper training and restraint are concerned. The proper thing to do, therefore, is to place the child who is a deaf-mute under the care of the most competent teacher available for the purpose, at the earliest possible time, certainly before the sixth year of age.

The child that has no training will remain a deaf-mute. He may go through the manual sign language, learn to communicate with his fellows, but he will always be much handicapped in the race of life, as his communication with his fellows must be limited to the few who have likewise learned the sign language. Then, too, he is forever debarred from the pleasure and developmental power derived from the mechanical action of the vocal apparatus, and the pleasurable sensation experienced in ventilating the blood and stimulating articulation, which accompany voice production (Makuen).

CHAPTER XXX

NEOPLASMS OF THE LARYNX

Benign tumors of the larynx and the trachea are characterized by the absence of pain and by non-recurrence. Malignant neoplasms, on the contrary, are characterized by pain, recurrence, and destructive processes.

Varieties.—Almost all types of benign tumors which occur in other parts of the body are found also in the larynx. The following are more or less frequently reported in the literature: papilloma, fibroma, myxofibroma, polypus, cystoma, lipoma, telangiectases, chorditis nodosa, and pachydermia laryngis.

Location. In looking over the literature for a period of ten years, I found lipoma and cystoma on the epiglottis; cystoma on the ventricular pouches; lipoma, cystoma, and papilloma in the arytenoid region; polypus telangiectasis, fibromyxoma, papilloma, fibroma, singer's nodules (chorditis nodosa), and myxocystoma on the upper surface of the vocal cords and in the subglottic region. These and doubtless other benign neoplasms occur in the locations indicated.

Etiology.—Much has been written, while but little is known, concerning the exciting causes of these growths in the larynx.

Jonathan Wright says: "There is a strong likelihood that if these tumors are not the result of chronic inflammatory changes, the chronic inflammations play an important role in their etiology, and that this should be borne in mind in the treatment." They occur at all ages, but most frequently in middle adult life. Papilloma, however, occurs more frequently in children, and measles is apparently a prolific exciting cause. Both men and women are affected, but the tumors are found more frequently in men. Sir Felix Semon has called attention to the fact that they are thought to occur more frequently in Germany and France than in the United States or England.

Benign neoplasms are relatively common among street vendors, singers, and speakers. Congenital tumors are rare. Papilloma is the most common variety. The anterior commissure is the most frequent site for laryngeal tumors. Lipoma rarely occurs within the cavity of the larynx, but is located extrinsically on the anterior surface of the epiglottis. Syphilis and tuberculosis, though they produce growths of their own kind, have little influence in causing innocent neoplasms. Papilloma, fibroma, and singer's nodules are more frequent than lipoma, myxoma, and cysts. Gerhardt says he has never seen an adenoma, a chondroma, angioma, or a neuroma. Others, however, have reported adenoma in the larynx. Moritz Schmidt, in his work on *Newgrowths of the Upper*

Air Passages, gives the following table of laryngeal tumors seen in his clinic of 32,997 cases in ten years:

	Men.	Women.	Cases.
Fibroma	178	78	256
Papilloma	31	15	46
Singer's nodules	56	53	109
Lipoma	1	0	1
Myxoma	3	0	3
Fibromyxoma	1	0	1
Tuberculous tumors	14	22	36
Cysts	2	6	8
Sarcoma	3	0	3
Carcinoma	61	15	76
Tracheal carcinoma	1	1	2

This table is significant, and is contrary in some respects to the accepted opinion. For instance, in the above table, fibroma occurs more frequently than papilloma. He found 256 fibromata and only 46 papillomata. Singer's nodules occurred in 109 cases, hence both fibromata and singer's nodules (chorditis nodosa) were found more frequently than papillomata. The apparent discrepancy is, no doubt, in the differential diagnosis, which is often carelessly made. It is too often made without a microscopic examination, and is, therefore, often incorrect.

The discussion concerning the exciting causes of benign neoplasms may be summarized as follows:

The causes are (*a*) local and (*b*) constitutional.

(*a*) Prominent among local causes is irritation. This produces hyperemia and cell activity, hence the persistence and the exaggeration of these two conditions may endanger life by allowing the tumor to grow so large as to interfere with respiration, or they may assume malignant tendencies. Mouth breathing is an important factor in producing irritation of the larynx. The required amount of moisture and warmth is not carried to the larynx, and the mucous membrane is overtaxed by the burden thrown upon it. The imperfectly prepared air causes a dryness as well as a hyperemia incident to the increased physiological activity of the mucosa, and the resultant irritation leads to an increased cellular activity. Under these conditions, the cellular arrangement is disturbed and neoplastic growths result.

(*b*) Constitutional influences play an insignificant part in the etiology of innocent neoplasms. This does not take into consideration the specific constitutional dyscrasias, as syphilis and tuberculosis, which produce peculiar local laryngeal redundancies.

In an adult, laryngeal papilloma is often associated with a warty skin, so much so that we can almost speak of a "warty diathesis." This theory was advanced by Fauvel, but it may be said, on the contrary, that the skin and the larynx have a totally different developmental origin. Sir Morrell Mackenzie maintained that syphilis and tuberculosis exercised a decidedly antagonistic influence to the development of new formations. Lennox Browne did not share this view, his experience rather proving the reverse. Moritz Schmidt thinks that they favor new forma-

tions, because they always induce a low state of resistance or a local vulnerability.

The Tendency to Malignancy.—It has been held that operative interference has a tendency to convert benign growths into malignant. This belief grew out of the fact that tumors which were operated upon and thought to be benign, were shown to be malignant in the recurrent state. Sir Felix Semon has shown that unoperated cases show even a greater percentage of converted malignancy than the ones which were operated upon. The following are his figures:

In a total of 10,747 benign cases reported in the literature, 45 afterward became malignant. They were divided as follows:

In 8216 operated cases, 33, or 1 in 249, became malignant.

In 2531 non-operated cases, 12, or 1 in 211, became malignant.

It is thus shown that a greater percentage of the non-operated cases become malignant. These figures should disprove the old theory that operative interference is an active factor in converting non-malignant neoplasms into the malignant variety. At the same time we must reckon the immense benefits which result from operations upon cases which do not become malignant, but continue to be troubled by the benign neoplasms.

Neoplasms of the Subglottic Space.—Ferreri states, with reason, that subglottic polypi often cause greater obstruction to respiration than polypi of the supraglottic space. They do not, however, cause a change in the voice until they come in contact with the vocal cords, whereas, tumors of the supraglottic region cause it from the beginning.

The development of subglottic polypi is insidious, hence they are not usually diagnosticated until well advanced, a fact which explains why they are usually larger than supraglottic polypi.

The most common form of benign subglottic tumor is the fibroma. Myxoma does not occur quite so frequently, but it is not uncommon to find it associated with fibroma in the form of a myxofibroma. Ferreri also says that, exceptionally, cysts, chondromata, and circumscribed keratosis have been observed in the subglottic space. Papilloma is rarely found in the subglottic region. When present they are difficult to remove by the intralaryngeal route, except by direct laryngoscopy. Thyrotomy (laryngofissure) may therefore become necessary, or infrathyroid laryngotomy may be the chosen method of operation.

The endolaryngeal methods of operating are with forceps, the snare, or the galvanocautery, either by direct or indirect laryngoscopy. Attacks of suffocation may render tracheotomy imperative, in which case the growth may be removed through the tracheal wound.

Papilloma.—**Etiology.**—According to Jonathan Wright, this type of neoplasm occurs more frequently in the larynx than any other variety According to the table of Moritz Schmidt, fibroma occurs more frequently. They are closely related to various inflammatory growths which accompany syphilis, tuberculosis, and pachydermia. In view of this fact, many laryngologists regard chronic inflammation as an

etiological factor. As already stated under General Etiology, this is still a mooted question. According to Jonathan Wright, they are usually classified as papillary fibromata. This may account in part for the discrepancy between Schmidt and other writers. Schmidt may have classified as fibromata what others call papillary fibromata. Schmidt observed papilloma in about 9 per cent. of his cases, Schrötter in about 18 per cent., and Moure in about 50 per cent. Schnitzler and Killian say they occur more frequently in children, and that fibromata occur more frequently in adults. Harmon Smith, J. Payson, C. Clark, Faurd, and Sir Morrell Mackenzie found them much less frequently in children.

Symptoms.—Papillomata are usually attached to the anterior third of vocal cords, or at the anterior commissure, though they may spring from any portion of the larynx. Tuberculous papillomata often grow at the posterior commissure.

Microscopically they have a stratified epithelial covering over a core of more or less vascular connective tissue. The outward growth of the epithelium is in contrast to the involuted growth of carcinoma. True nests or pearls of epithelial tissue have been found.

Papilloma may appear upon inspection to be either pedunculated or sessile, though upon microscopic examination all varieties have the same structure. It is probable that those having a sessile or diffused base are in reality only numerous sessile pedunculated growths closely crowded together and fused in the process of development. When single, the growths may present a distinct pedicle with a warty growth at its extremity. When multiple, it may appear to be sessile, or it may have the appearance of a cauliflower-like growth.

Papillomata may be pale or congested; when congested they are more active in their growth. These appearances may be used for prognostic purposes. For example, when pale their activity is diminished and their removal is not so likely to be followed by recurrence, and *vice versa*. In one of the cases reported by Harmon Smith, there was a fibrosis at the anterior commissure of the cords, which Jonathan Wright thinks might disappear when the papillomata cease to recur.

Like warts on the skin, papillomata of the larynx come and go without any apparent reason. J. Payson Clark emphasizes the importance of a physiological change which marks the limit of their growth. When this period occurs their removal may be accomplished with a reasonable hope of non-recurrence. He also emphasizes the futility of operating when they regrow immediately after operation; tracheotomy is then the rational mode of treatment.

Hoarseness or aphonia are characteristic symptoms, though Logan Turner exhibited the larynx of a child crowded with papillomata, which died, without previous symptoms, during a choking fit at dinner. The hoarseness and aphonia may be transitory or constant. Dyspnea and cyanosis are sometimes severe, and when present, necessitate immediate tracheotomy. If the dyspnea is great, the supraclavicular region will be depressed.

The general health is often impaired and the weight diminished by several pounds.

Pathology.—Papillomata may be either hypertrophied normal papillæ or they may be newgrowths.

Prognosis.—According to J. Payson Clark, the prognosis during the retrogression stage, or stage of physiological limit, is quite favorable. This stage is also favorable for the removal of the growths. Conversely, during the stage of active growth, or before the stage of physiological limit, the prognosis is much less favorable either as to life or hope of cure by operation. Some cases get well without operative interference. According to Clark, the prognosis is influenced by the technique with which tracheotomy is performed. A preliminary tracheotomy performed at leisure and with exactness is more favorable than an emergency tracheotomy done in haste with poor technique. He therefore urges that a preliminary tracheotomy be performed when dyspnea first develops, and that the removal of the growths be delayed for several weeks, or until the growths begin to diminish in size.

The prognosis is bad when the patient develops a cold or contracts measles or other infectious sequelæ, especially if a tracheotomy tube is being worn.

According to Harmon Smith, B. V. Burns collected statistics of 127 children with laryngeal papillomata, of which 48 were not operated upon, and of these, 32 died, 28 by suffocation. Three were cured spontaneously; 25 were tracheotomized, 7 died after operation. Twenty-one were subjected to laryngofissure, 8 being permanently cured. Forty were operated upon by the intralaryngeal route, and 13 were permanently cured. In Rosenberg's statistics of 109 children with papillomata subjected to laryngofissure, 20 died from suffocation due to recurrence of the growths. In 43 there was recurrence after repeated operations, though 40 were finally cured.

The prognosis is much more favorable in adults.

Treatment.—*Local.*—Delevan reports good results from the local application of alcohol in adults; Shurly from the use of thuja occidentalis. Zinc chloride, nitrate of silver, adrenalin, and lactic acid have been tried with slight success.

Internal.—Of the internal remedies, arsenic has produced the best results. Bostoc favors the use of potassium iodide. The value of these remedies seems to depend upon their regenerating effect upon the general system.

Surgical.—The trend of opinion is away from laryngofissure (thyrotomy) and the indirect laryngeal method, and toward tracheotomy and the direct laryngeal method.

Laryngofissure is not favored on account of the frequent recurrence of the growths. The operation is attended with shock, possibly by death, and is somewhat disfiguring. It is often attended with stenosis of the larynx and an impairment of the voice. The chief argument against this operation for laryngeal papilloma is that other methods afford a better means of relief.

Operation by direct laryngoscopy (Chapter XXXI) with Jackson's self-illuminated tube spatula is much superior to indirect laryngoscopy. The growths are brought into clearer vision and greater accessibility. Removal by direct laryngoscopy may be attempted when dyspnea and cyanosis are not present. If these symptoms are present, the instruments for tracheotomy should be in readiness if suffocation occur. The growths may be removed through Jackon's self-illuminated tube spatula with straight forceps.

Operation by indirect laryngoscopy may be practised when symptoms of suffocation are absent and Jackson's or Killian's tube spatula is not at hand. The surgeon should, however, be prepared to perform tracheotomy if suffocation threatens during the operation.

Tracheotomy should be performed in all cases in which dyspnea and cyanosis are present. The procedure should not be postponed until it becomes an imperative measure, but should be done while the patient is still in a condition to permit the operator to do it with deliberation and good technique, as suggested by J. Payson Clark. According to G. Hunter Mackenzie, tracheotomy is sometimes followed by a cure of the papillomata. While this is true of some cases, it is not true of all, nor of the majority of cases. Tracheotomy should be done to avoid the dangers of suffocation, aside from its curative influence. It should rarely be followed by the immediate removal of the growths. Weeks or months should usually intervene. Indeed, it is useless to remove the growths while they are in the active stage, as they will recur, often in greater abundance, than before their removal. Indeed, if the healthy tissue is injured during the operation, the growth will often promptly appear at this point (H. L. Swain).

When the growths show a state of quiescence or of retrogression, they may be removed by indirect or direct laryngoscopy or through the tracheal wound.

OPERATION BY INDIRECT LARYNGOSCOPY

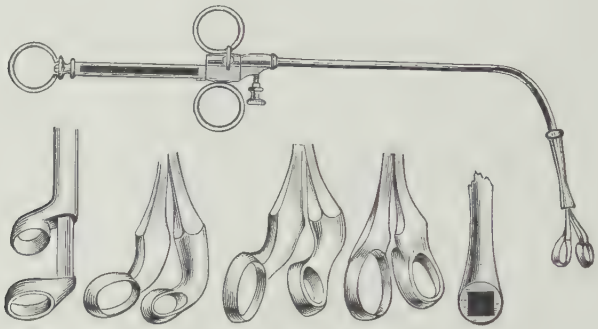
In describing this operation for the removal of papilloma, it must be taken as a type of surgical procedure used in the removal of nearly all varieties of benign laryngeal neoplasms. Each case will, of course, require some modification of the various steps in the operation.

Technique.—Preparation of the Patient.—(*a*) The throat should be gently sprayed with Seiler's or Dobel's solution. The fauces and the larynx should then be sprayed with a 2 per cent. solution of cocaine to reduce the reflex irritability.

(*b*) The larynx is then swabbed with a 10 per cent. solution of cocaine. This should be repeated at intervals of five minutes until anesthesia is induced. If this does not produce anesthesia after several applications, one or two applications of a 20 per cent. solution should be made. This strength of solution should be used sparingly and with caution, although in my experience the larynx has been quite tolerant of cocaine.

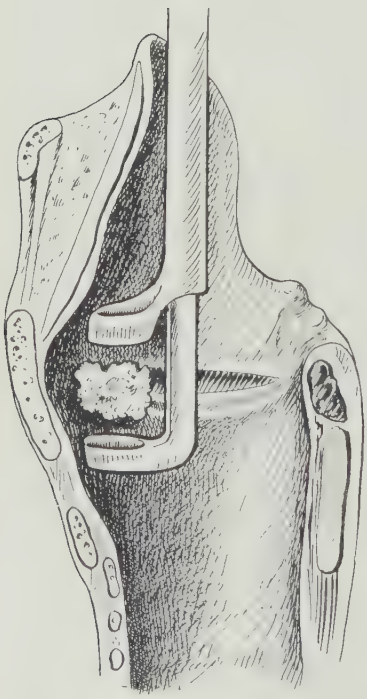
(*c*) The laryngoscopic mirror is introduced into the oropharynx with its reflecting surface directed downward and forward so as to reflect the rays of light from the head mirror to the growth, the tongue being gently

FIG. 332



Krause-Heryng laryngeal forceps.

FIG. 333



Detailed drawing showing the laryngeal forceps placed to remove the neoplasm.

rolled forward on the forefinger of the left hand. The epiglottis is thereby lifted, exposing the larynx to view.

(*d*) Next introduce the curved laryngeal pincette, or double cutting forceps (Fig. 332), into the upper space of the larynx until its cutting extremity touches the growth (Fig. 333). It must be borne in mind that the image in the mirror is reversed, hence the movements of the instrument should be directed in an exactly opposite direction from what appears to be necessary according to the image in the mirror. For example, if the tip of the instrument seems to need a more forward position, so manipulate the handle as to move the tip backward, *i. e.*, lower the handle. If the tip of the instrument seems to be too near the posterior portion of the image, it is in reality too near the anterior portion. A little practice upon a model or upon a patient will familiarize the student with this procedure. The surgeon soon learns intuitively to move the instrument in the proper direction.

It is of great aid first to fix firmly in the mind the anatomical relations of the various parts of the larynx. For example, it must be remembered

that the epiglottis stands at the anterior commissure of the larynx, and the arytenoid prominences at the posterior commissure. These simple anatomical guides, if impressed upon the memory of the operator, will lead him unconsciously to guide the laryngeal instrument in the proper direction.

(*e*) Having located the growth with the laryngeal forceps or pincette, so manipulate the handle of the instrument as to separate the tips, and then with a slight downward movement of the instrument close the forceps upon the neoplasm and remove it *en masse* or in part. If the growth is large or multiple, several repetitions of the foregoing procedure may be required. The growth should be removed with as little trauma to the surrounding tissues as possible.

OPERATION BY DIRECT LARYNGOSCOPY

(See Direct Laryngoscopy, Chapter XXXI)

MALIGNANT NEOPLASMS OF THE LARYNX

The Lymphatic Drainage of the Larynx.—The lymphatics of the larynx are of clinical importance in malignant neoplasms and infectious diseases of the larynx. According to Most, Cunes, Boubland, and Green, the following summary gives the essential facts:

The lymphatic trunks which take their source from the larynx are derived from a network of radicles which extend throughout the larynx beneath the mucous membrane. This network is divided by a horizontal plane at the level of the vocal cords into a supraglottic and an infraglottic portion. The supraglottic portion includes the lymphatics of the epiglottis, arytenoids, ventricular bands, ventricles, and vocal cords. The network of vessels is continuous throughout these areas. Over the upper portion and posterior surface of the epiglottis the network is fine and the meshes are far apart. In front and lower down, especially at the sides, the meshwork is denser and the strands thicker. Over the arytenoids, ventricular bands, and throughout the ventricular pouches the lymph channels are thick and closely woven. In the vocal cords, however, the network is very fine and more sparse than in any other part of the larynx. The infraglottic network is finer than that above the vocal cords, but by no means as fine as that of the cords themselves. The lymph from these radicles is collected into trunks which leave the laryngeal cavity at certain definite places.

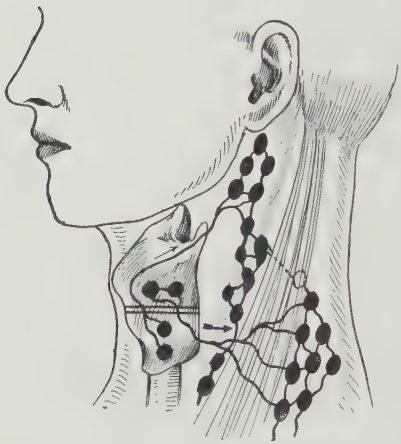
In the upper part of the larynx the only place of egress is through the thyrohyoid membrane. The lymph vessels of the upper network assemble in the vicinity of the aryepiglottic folds into several trunks, three to six in number, which leave the larynx through the above-mentioned membrane near the superior thyroid artery, a corresponding group being on either side of the larynx.

These trunks course outward and backward, more or less in relation to the superior thyroid artery, to the carotid region, and terminate in

nodes which lie along the surface of the internal jugular vein at the level of the bifurcation of the carotid. The upper trunk of this group often runs backward, after emerging from the thyrohyoid membrane, along the hyoid bone to the tip of the lesser cornu, and thence outward to a node lying on the inferior aspect of the posterior belly of the digastric muscle. The lower trunks of this group may run by a lower course, outward and downward, into glands in the chain lying on the surface of the internal jugular vein, below the lower border of the lateral lobe of the thyroid gland (Fig. 334).

The collecting trunks of the infraglottic network are divided into an anterior and a posterior division. The anterior division consists of three or four small trunks, which pierce the cricothyroid membrane in the median line and terminate in small glands, which lie in the median line at uncertain locations. The uppermost of these is fairly constant and lies in the V-shaped space of the cricothyroid membrane formed by the inner borders of two thyroid isthmuses, and a third on the anterior surface of the trachea. These two are denominated respectively the prethyroid and the pretracheal glands. They may receive trunks from the anterior infraglottic group. Efferent trunks from these glands run to the beforementioned chain of glands lying on the anterior external surface of the internal jugular vein.

Fig. 334



Schema of the lymphatic flow from the supraglottic and the infraglottic regions of the larynx. The glands of the supraglottic region flow into the posterior chain, while the infraglottic glands flow into the anterior cervical chain of glands. This is of diagnostic significance in determining if a cancer is supraglottic or infraglottic.

In the posterior division are three to five infraglottic collecting trunks, which penetrate the cricotracheal membrane at or near the line of junction of the cartilaginous and membranous portions of the trachea and run into a chain of glands, two or five in number, which lie along the course of the recurrent laryngeal nerve known as the recurrent chain. From these glands run vessels communicating with the lowermost glands of the internal jugular chain and a few to the supraclavicular group of glands.

The lymphatic drainage from all parts of the larynx thus eventually leads into the chain of glands lying under the sternomastoid muscle, along the surface of the internal jugular vein, or into the supraclavicular glands. The prelaryngeal, prethyroid, and pretracheal glands are merely intercepters of the current on its way to the deeper glands.

The spread of infection or of malignant neoplasms from either the supracordal (glottic) or infracordal region is to the deep lymphatic

nodes along the internal jugular vein beneath the sternomastoid muscle, or, in other words, to the same lymphatic system into which the tonsils drain. In infectious and advanced malignant processes of the larynx the deep cervical glands along the internal jugular vein and beneath the sternomastoid muscles are enlarged. In malignant tumors of the larynx such enlargement of the glands constitute a contra-indication to operative interference.

Varieties.—Epithelioma, adenocarcinoma, and sarcoma. Of these the epithelioma occurs the most frequently. Ziemssen reported 57 epitheliomata in 68 malignant cases, while 9 were sarcomata. Bosworth collected 344 cases, of which 204 were carcinomata and 130 sarcomata. Sir Felix Semon, in 1899, gathered the statistics of all laryngeal growths, amounting, all told, to 10,747 non-malignant cases and 1550 malignant cases, 1 in 7 being malignant.

General Facts.—It may be stated, with some confidence, that malignant neoplasms may be cured if operated upon sufficiently early. This is not done as often as it should be, hence the mortality rate is still extremely high. The crying need of the hour is "an early diagnosis." How sad the comment upon medical attainments is the "fact" that but few practitioners are able to diagnosticate laryngeal cancer until the patient is *in extremis*. Yet how easy it is to learn one or two simple indications that should at least put them on their guard, and save their self-respect, their reputation, and the lives of their patients.

What, then, are the early indications of laryngeal cancer? The early signs of cancer of the larynx are:

(*a*) Continued hoarseness without cough, and without other known cause.

(*b*) Sharp, sudden pains in the larynx, the ear, or the pharynx.

(*c*) Age, the fortieth year and upward; though cancer, especially sarcoma, may occur at a much younger age.

(*d*) A laryngoscopic examination may show loss of movement of one of the vocal cords.

The above symptoms are not conclusive, but they should arouse suspicion of malignancy. The practitioner may, upon the foregoing data, make a tentative diagnosis of a malignant growth in the larynx; and he will be correct in nearly every instance.

To sum up: If a patient, forty or more years old, complains of continued hoarseness without cough, and of sharp, sudden pains through the larynx, pharynx, or ear, he should be suspected of having a malignant growth in the larynx.

What other diseases cause this symptom-complex? Perhaps laryngeal tuberculosis, syphilis, perichondritis, or rheumatic laryngitis may approximately duplicate it. There are other peculiar symptoms of these diseases, however, which readily distinguish them from malignant neoplasms In rheumatism there may be sharp pains and hoarseness, but the symptoms are fugitive; they do not persist as in malignant neoplasms. In tuberculosis and syphilis a casual examination should readily enable the practitioner to make the differentiation.

The extreme simplicity of the symptom complex of the early stage of malignant growth of the larynx encourages me to emphasize the symptoms, as I have in the preceding paragraphs. I wish to urge every practitioner of medicine and surgery to impress indelibly upon his mind the few facts just given. Cancer of the larynx is not a rare disease, but, on the contrary, is quite common; more than 1500 cases were on record in 1889, and since then as many more have been diagnosticated and treated, though many have not been published. Inasmuch, therefore, as the disease is comparatively common, I desire to make plain the tentative diagnosis, and divest it all of complex considerations. It may be reduced to (*a*) age, forty years or more; (*b*) continued hoarseness without cough; and (*c*) sudden, sharp pains in the larynx, pharynx, or ears.

Etiology.—The exciting cause of malignant neoplasms of the larynx is not clearly understood. Chronic inflammation of the larynx seems to be a factor, as the statistics show that families having a history of malignant growths are more often attacked in the larynx when subject to chronic inflammations. The use of tobacco also seems to be an exciting cause.

Virchow tersely says that healthy tissues, if continually subjected to irritations, may be the seat of heteroplastic growths, and that the larynx, more than any other organ, where no trace of heredity or predisposition exists, is likely to be the site of malignant growths.

Age.—The age at which malignant growths of the larynx appear varies somewhat with the variety of the cancer. Sarcoma often occurs in the very young. The author saw a case of melanosarcoma in a child eighteen months old, which pursued a very rapid course with a fatal termination. It is, however, more frequent in young adult life. Epithelioma occurs in middle adult life and in old age; carcinoma, chiefly between the ages of forty and sixty.

Malignant growths of the larynx, without reference to their variety, according to the following table from Gerhardt, occur with greatest frequency between the fiftieth and sixtieth years:

	Cases.
20 to 30	4
30 to 40	18
40 to 50	49
50 to 60	76
60 to 70	30
70 to 80	10
Total	187

Schrötter observed carcinoma in a child aged three and one-half years, and in a girl aged ten and one-half years.

Sex.—Sex influences the formation of malignant growths to a marked degree. Gerhardt found carcinoma three times as prevalent in males as in females, while Semon found them in males four times as frequently.

Social Standing.—The conditions in life seem to influence the occurrence of malignant growths of the larynx, the well-to-do being more often afflicted than the poor.

Pathology.—The pathological anatomy of laryngeal cancers is quite similar to that of carcinoma and sarcoma elsewhere in the body, and will not be described in detail. Under Symptoms will be found a brief characterization of malignant epithelial neoplasms, to which the reader is referred.

Symptoms.—The chief clinical symptoms are: (*a*) Continued hoarseness without other known cause. (*b*) Sharp lancinating pains in the ear and pharynx. (*c*) Loss of movement of the vocal cord on the affected side. (*d*) The patient is forty years of age, or more, except in sarcoma, which may occur at any age.

Continued hoarseness may be the only symptom for several months, and the pain and the loss of movement of the cord may commence at a later period; hence, continued hoarseness, without other known cause, should, in a patient forty or more years of age, be sufficient to arouse suspicions as to the presence of a malignant growth in the larynx. While it may be said that a positive early diagnosis is difficult to make, it is, on the other hand, easy to make a provisional diagnosis and place the patient under observation so as to give him the advantage of the earliest possible diagnosis. I make a plea, therefore, with Sir Felix Semon, von Bergmann, Chevalier Jackson, Otto Stein, and others for an early diagnosis. This alone offers a reasonable hope for the successful treatment of this disease.

The hoarseness grows progressively worse, and the voice may finally become aphonic.

As the edema develops, and the growth encroaches upon the lumen of the glottis, dyspnea, of greater or less intensity, may embarrass the patient.

Cough, increasing with the progress of the disease, is usually present. The expectoration is at first similar to that in chronic laryngitis, and later is admixed with purulent secretion, and with blood in the ulcerative stage.

Dysphagia, or difficult deglutition, is a late symptom in the intrinsic variety of the disease. If, however, the primary cancer is in the pharynx or the esophagus, it may appear at a much earlier period.

The enlargement of the lymphatic glands of the neck is a late symptom, only occurring after ulceration of the tumor has taken place. Epithelioma is often attended with a very tardy enlargement of the glands. In intrinsic tumors of the larynx two sets of glands are secondarily affected, namely, the group at the angle of the jaw and those behind the sternocleidomastoid muscle. The subglottic glands of the larynx empty into those at the angle of the jaw, while the supraglottic glands empty into those posterior to the sternocleidomastoid muscle. If, therefore, the glands at the angle of the jaw are enlarged, it should arouse suspicion, at least, of a subglottic cancer.

The late involvement of the lymphatic glands in intrinsic laryngeal cancer is another argument in favor of an early diagnosis, as the tumor can then be easily removed *in toto*. Should the diagnosis be made only after glandular enlargement has taken place, the operation is a much

more formidable one, as it necessitates the removal of the glands. Furthermore, the probability of total resection of either tumor or glands is generally lessened in the advanced stage of the disease, for recurrence generally takes place.

Laryngoscopy.—The laryngoscopic examination often presents a picture so characteristic as to confirm at once the suspicion aroused by the other symptoms present. When only one side is affected, the abductors, and later the adductors, are paralyzed on the affected side. Both sides are paralyzed when the entire larynx is involved.

According to Semon's law, the abductor muscles atrophy before the adductor fibers, hence abductor paralysis appears first and is followed by adductor paralysis.

By reference to Figs. 335 and 336, illustrating two of the author's cases, the laryngeal image in unilateral cancer of the larynx is shown.

FIG. 335

Carcinoma of the right ventricular band of the larynx. It was removed by the intralaryngeal route by the author, returned in one year, was reoperated upon by the same route without relief, the patient dying two months later. (Author's case.)

FIG. 336

Paralysis of the thyro-arytenoidei interni and the arytenoideus in attempted phonation, more severe on the left side. Drawn from the author's case of subglottic carcinoma of the larynx.

The microscopic diagnosis is not always reliable, especially if the tissue is removed by the endolaryngeal route (W. J. Terry), as the cancerous growth may be deeply seated beneath the mucous membrane. If, however, the specimen for examination is removed by laryngofissure, it can be obtained from the deeper structures, and should, therefore, afford an accurate means of diagnosis. B. Fraenkel maintains that the microscopic diagnosis is of fundamental importance. Negative results should not, however, be taken as final, especially if the specimen is obtained by the endolaryngeal route. A positive finding, however, is dependable if made by a competent pathologist. A globular collection of epithelial cells is suspicious only. Epithelial cells must be found where they do not belong. The irregular structure of the epithelium, such as is found in typical epithelial nests, is characteristic of cancer.

When the microscopic findings include the foregoing points, a positive diagnosis of cancer of the epithelial variety may be made.

Diagnosis.—Cancer of the larynx should be differentiated from (*a*) chronic laryngitis, (*b*) syphilitic laryngitis, (*c*) tuberculous laryngitis, (*d*) perichondritis, and (*e*) benign neoplasms of the larynx.

(*a*) Chronic laryngitis: hoarseness, while present in both chronic laryngitis and carcinoma, is more persistent in carcinoma. In chronic laryngitis the voice is husky upon arising, but becomes clear during the day, and in the hypertrophic variety there are discrete enlargements of the mucosa, but they do not have the distinct nodular surface which is present in carcinoma. In chronic laryngitis the vocal cords are freely movable in both abduction and adduction, whereas in carcinoma one or both cords are immovable.

(*b*) In syphilitic laryngitis, the hoarseness is low-pitched, and brassy or raucous in character. In carcinoma of the larynx it is higher pitched, and softer in character; indeed, it may become aphonic in the later stages. The cords are freely movable in syphilitic laryngitis, and the history of the case usually clears the diagnosis.

(*c*) Tuberculous laryngitis is characterized by hoarseness and pain, and when perichondritis is present, by fixation of one or both vocal cords. The history and the examination of the sputum render the diagnosis so clear that malignancy is practically excluded.

(*d*) Benign neoplasms of the vocal cords (the most frequent site of intrinsic malignant neoplasm) are characterized by hoarseness, though pain and paralysis of the laryngeal muscles are absent.

Prognosis.—The general prognosis of malignant growths of the larynx is bad. This would not be so if an earlier diagnosis were made. In other words, the prognosis depends in a large measure upon the early recognition and surgical removal of the diseased tissue. Sir Felix Semon claims that 90 per cent. of his cases have been cured by thyrotomy. All, or nearly all, of his surgical cases were diagnosticated and operated upon in the early stage, hence the high percentage of cures. Jackson, in a total of 9 complete laryngectomies, including the epiglottis, had but 1 death immediately following the operation. The other cases lived eight or more months after the operations.

Gluck in his first 10 cases reported 2 as cured (three years without recurrence). In his last series of 22 cases 1 died, making a percentage of recoveries higher than Semon's. Of a total of 23 complete laryngectomies, he claims 3 good results. In 1903, out of 125 cases he claimed he could show 38 living cases, the oldest still alive and in good condition thirteen years after the operation.

Of those dead, some lived eleven, eight, six and one-half, five and one-half, four and one-half, and three and one-half years. Some died of illness other than recurrence.

Kocher in 12 cases had 6 recurrences. White and Powers, after reviewing a large number of cases, conclude that in complete laryngectomies the death rate is 35 per cent., while in partial laryngectomies it is about 27 per cent.

Werckmeister collected 297 cases of complete laryngectomy, of which 36 were fatal, by which he probably means that 36 died during or soon

after the operations. How many died later from recurrence is probably not shown in these figures.

In a collection of 105 cases operated on by laryngofissure, 4 died of pneumonia within eight days. The low death rate from this cause stamps the procedure as safe from a surgical standpoint. The voice after laryngofissure varied with the extent of the operation. In benign tumors it usually remains fair or good. In malignant neoplasms, as they generally affect the integrity of one or both cords, it is not so good. If only one cord is involved, a useful voice is retained in simple laryngofissure and in hemilaryngectomy.

In summing up the prognosis under operative treatment, it may be said: (*a*) That in those cases diagnosticated and operated on in the early stage, before ulceration and extension to the neighboring parts, the prognosis is good. (*b*) In those cases operated on in the late stages the prognosis is bad. (*c*) The personality of the operator and the fortunate opportunity of seeing the cases in an early stage favor a better prognosis. (*d*) Laryngofissure gives a better chance of recovery when the disease has not extended to the extrinsic parts of the larynx. (*e*) Total laryngectomy is attended with greater shock and a higher mortality than the more limited operations. It should be remembered, however, that this method of operating is usually adopted in the more advanced and hopeless cases. (*f*) Keishaber has divided cancer of the larynx into two clinical groups, which, from the standpoint of prognosis and treatment, is important, namely: (1) Intrinsic cancer of the larynx, and (2) extrinsic cancer of the larynx Intrinsic cancer has its origin in the vocal cords, the ventricular bands, and the ventricular pouches. Extrinsic cancer of the larynx arises from the arytenoid cartilages, the epiglottis, and other parts contiguous to the larynx. In intrinsic cancer the growth develops slowly and extends with extreme reluctance by metastasis to the lymph glands behind the sternocleidomastoid and to the neighboring tissues surrounding the larynx.

In the extrinsic variety, the reverse of the above facts is true. In other words, the prognosis in intrinsic cancer of the larynx is naturally much more favorable than it is in the extrinsic variety. To make accurate deductions from the statistics of cancer of the larynx, it is necessary to know whether it is intrinsic or extrinsic, sarcomatous (for it is much more favorable in this variety) or carcinomatous; whether operated in the early, middle, or late stage; whether by laryngofissure, partial laryngectomy, hemilaryngectomy, complete laryngectomy, or by ligation and resection of the external carotid arteries and their branches, as advocated by Dawbarn.

The foregoing data fairly represents the prognosis under existing methods and conditions, though I fear that it presents it in a too favorable light.

Frank Hartly, in 1902, reviewed the literature from 1833, when Brauers performed the first thyrotomy, and the first laryngectomy by Watson in 1878, down to the more improved methods of operating in 1900. The death rate within the first days after the operation, up to 1889, for laryn-

gectomies was 44 per cent., and of those remaining cured for three years prior to 1889 it was 7 per cent. Since 1889 the death rate within the first ten days has been 8.5 per cent.; in those remaining cured, 15 per cent. The following tabulation shows the improvement in the immediate and the remote death rate and the net gain in the mortality:

Death Rate in Laryngectomies for Every One Hundred Operations.

	Immediate deaths. Per cent.	Remote deaths. Per cent.	Total deaths. Per cent.	Living. Per cent.
Prior to 1889	44.0	52.0	96.0	4.0
1889 to 1900	8.5	47.5	56.5	44.0

The present total death rate before the end of the third year is 56 per cent., as against 96 per cent. prior to 1889. The tremendous improvement in the mortality rate is encouraging, and stands as the strongest argument in favor of still further improving the surgical technique for the cure of this dread disease. It should be remembered, however, that the improved mortality rate following the surgical treatment is largely due to the more intelligent selection of cases, as well as to the improved technique and asepsis now in vogue. In the period prior to 1889 the failure to elect the proper method of operating probably largely contributed to the high death rate. There is still room for improvement in this regard, and it is to be hoped that in the near future a still lessened mortuary report will be given.

Pean reports a case of extirpation of the larynx and part of the esophagus for a cancerous tumor diagnosticated by laryngoscopic examination. Although apparently limited to the left side, it was found to extend to the right side, and to the upper portion of the esophagus, the hyoid bone, and the base of the tongue. The whole mass was removed, and, to compensate for the extensive loss of substance, the esophagus was drawn up and stitched to the skin in the upper angle of the wound. The trachea with a cannula inserted in it was also secured by suture to the skin. An artificial larynx was supplied, which not only enabled the patient to swallow, but also allowed him to inhale air physiologically prepared in passing through the nose.

Pean draws the following conclusions from the case:

1. That it is impossible, prior to operation, to be certain of the extent of the disease when no subjective symptoms are present.

2. That the surgeon must never promise beforehand to limit the operation to the removal of only a part of the larynx.

3. That an extensive operation, including the removal of the hyoid bone and the base of the tongue, may be undertaken with safety and success.

4. That after such operation, important modifications of the anatomy of the parts operated on always follow, the abnormal openings of the trachea and the esophagus being raised, the epiglottis and the root of the tongue being lowered.

5. That, thanks to suitable mechanical appliances, the functions of the parts can be, to a large extent, restored, even after the most extensive operations.

Treatment.—The various methods of treating laryngeal cancer may be appropriately studied under the following heads:

1. The endolaryngeal operation.
2. Laryngofissure or thyrotomy.
3. Subhyoid pharyngotomy.
4. Partial laryngectomy or hemilaryngectomy.
5. Complete laryngectomy.
6. Ligation or injection of the external carotids and their branches.
7. Tracheotomy.

Each of the foregoing methods of treatment has its advocates, and, in selected cases, its advantages. I shall endeavor to point out the most prominent indications for each in such a way as to enable the surgeon to elect the one most suitable for the case in hand.

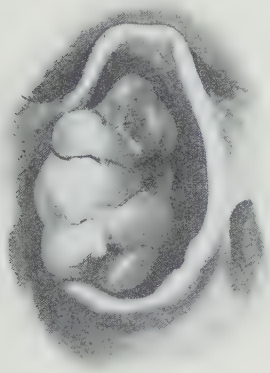
1. **The Endolaryngeal Operation.**—The endolaryngeal operation for cancer of the larynx is not unlike that described for papilloma of the larynx. The responsibility attending it is, of course, much greater on account of the gravity of the disease. The most distinguished advocate of this method of operating is B. Fraenkel, who cured three cases by operating on them by the endolaryngeal route at intervals covering a period of five years. At the time of his published report there had been no recurrence after two years of quiescence. I have operated on a few cases by this method, in one of which there was recurrence in ten months, with pronounced hoarseness, dyspnea, pain, and cachexia. The second operation did not relieve the patient as did the first. He gradually grew worse, and died two months after the second operation, which was performed twelve months after the first. The case (Fig. 337) should have been subjected to hemilaryngectomy or complete laryngectomy at the time of the first operation, notwithstanding the fact that the tumor was apparently accessible to the double cutting forceps *via* the mouth. It is quite probable that I did not succeed in removing all the cancerous tissue, which I could have done had I resorted to an operation by the external route. Notwithstanding the brilliant results reported by B. Fraenkel, I think the endolaryngeal operation should rarely be the operation of choice. It may be chosen when other methods are refused. Direct laryngoscopy promises better results than are obtained by the indirect method. Laryngofissure may be performed, if a pathologist be present in order to make an examination of the specimen by the freezing method, which only requires a few minutes. In Figs. 332 and 333 are shown the author's cases of pedunculated carcinoma of the larynx. This is a rare condition, and I know of only two similar cases on record (B. Fraenkel). The glands of the neck were large and firm. A gland was first removed and submitted to microscopic examination and carcinoma was found. The laryngeal neoplasm was then removed with a snare. As the patient swallowed the growth, warm salted water was given and the tumor ejected. The patient, aged forty-five years, died eighteen months later, metastatic carcinomata being found post-mortem in the liver, spleen, and stomach.

The operation may be completed by the method which appears to be

best in view of the macroscopic and microscopic findings. The precise location and extent of the growth, whether intrinsic or extrinsic, should also be determined after the larynx is opened by laryngofissure.

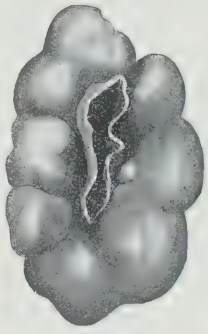
In order to render the thorough examination of the parts through the laryngofissure possible, the interior of the larynx should be brushed or sprayed with a 10 per cent. solution of cocaine to abolish the reflexes. Adrenalin, 1 to 1000, may be used to shrink the mucous membrane, and thus bring the limitations of the growth into greater prominence.

FIG. 337



The author's case of pedunculated carcinoma of the larynx growing from the left ventricular band. The tumor was distinctly movable. It was removed with a cold-wire snare through the mouth. The patient swallowed it, was given warm salt solution, after which he ejected it, and the rare specimen was thus preserved. A gland was previously removed from the corresponding side of the neck, and upon microscopic examination by the Columbus laboratories, it was pronounced carcinoma. The laryngeal tumor was likewise submitted and pronounced carcinoma. Peculiar interest attends the case on account of the distinct segregation of the tumor from the surrounding tissues and its pedicled attachment.

FIG. 338



View of the inferior surface of the author's case of pedunculated carcinoma of the larynx in a man aged forty-five years. The peduncle was tubular and composed of mucous membrane, and was attached to the ventricular band of the left side. The tumor was freely movable in the larynx, occasionally obstructing the breathing. The tumor presented the appearances of a gland dislocated beneath the mucous membrane.

2. **Laryngofissure or Thyrotomy.**—This operation is one that should be chosen for the purpose of obtaining a specimen for examination and for the removal of cancerous and benign growths.

The indications: (*a*) For the removal of foreign bodies lodged in the ventricular pouch which cannot be removed by either the direct or indirect endolaryngeal route.

(*b*) For the removal of benign neoplasms which cannot be reached successfully by the endolaryngeal route.

(*c*) To obtain a specimen from a suspected malignant neoplasm of the larynx, for microscopic examination, especially when the one removed by the endolaryngeal route gives a negative result.

(*d*) To expose the interior of the larynx to view in order to determine the gross appearance, site, and extent of a laryngeal neoplasm, preliminary to the election of the method of removal.

(*e*) As a method of election for the removal of an intrinsic malignant growth of the larynx.

When should laryngofissure be the method of choice or election in malignant neoplasms?

(*f*) When, upon laryngoscopic examination, the growth is found to be limited to the soft parts or to a small area, and can be removed through the laryngofissure, with the sacrifice of but little or none of the cartilaginous framework of the larynx.

(*g*) When, upon laryngoscopic examination, the growth, while somewhat extensive, does not appear to involve the deeper tissues, and can in all probability be entirely removed by laryngofissure.

(*h*) When the growth is somewhat more extensive than in (*f*) and (*g*), but is still circumscribed within a fractional part or one-half of the larynx, having its origin from one cord, or the ventricular pouch or band is not ulcerated, and there is no enlargement of the glands posterior to the sernocleidomastoid muscle.

(*i*) When the growth is intrinsic, the vocal cord, the ventricular pouch, or the ventricular band, even though it is quite large, and the lymphatic glands posterior to the sternocleidomastoid muscle are not enlarged, it is barely possible that operation by laryngofissure may be successfully done. If the growth has involved the cartilaginous framework of the larynx to such an extent as to necessitate the removal of a considerable portion of it on one side, laryngofissure should not be the method of choice. Hemilaryngectomy or incomplete laryngectomy should be chosen after a preliminary laryngofissure.

Axiom: Laryngofissure should be the operation of choice when the malignant neoplasm is intrinsic, and when diagnosticated in the early stage.

Laryngofissure or thyrotomy has been frequently referred to as a method of removing growths, foreign bodies, and stenosis of the larynx. It will be described as such, and cross-reference will be made to it wherever the author thinks it the proper procedure for other affections.

Technique.—This operation consists in splitting the larynx in the anterior median line and removing the growth through the fissure thus made. It is not a formidable procedure, and should be done much oftener than it is.

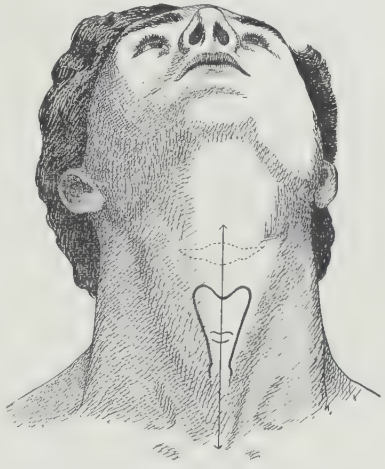
(*a*) Preparation of the patient: In this, as in all cases where a general anesthetic is to be administered, the patient should be placed in a hospital twenty-four to forty-eight hours before the time of operation. Broken doses of calomel, followed by a saline cathartic the following morning, should be administered in time to produce a free evacuation of the bowels a few hours before the operation. The patient should be given no food within nine hours of the operation.

(*b*) The preparation of the field of operation: The neck should be cleansed and shaved twelve hours prior to the operation, and a moist

carbolic dressing placed over the laryngeal region and held in position with a bandage. The cleansing should be repeated after the patient is under the influence of the anesthetic.

(*c*) Anesthesia: Rectal anesthesia, as practised by Cunningham, of Boston, and Stucky, of Lexington, is performed by the administration of the vapor of ether with Cunningham's apparatus. It combines simplicity and safety; a small amount of ether is used; and its administration is not followed by nausea or vomiting, though prolonged diarrhea may be produced. The method is especially useful in operations about the head, as the anesthetist is removed from the field of operation. In throat operations it is especially recommended, as the anesthesia may be administered throughout the operation and the secretions are not stimulated thereby.

FIG. 339



The line of incision for the complete or partial removal of the larynx.

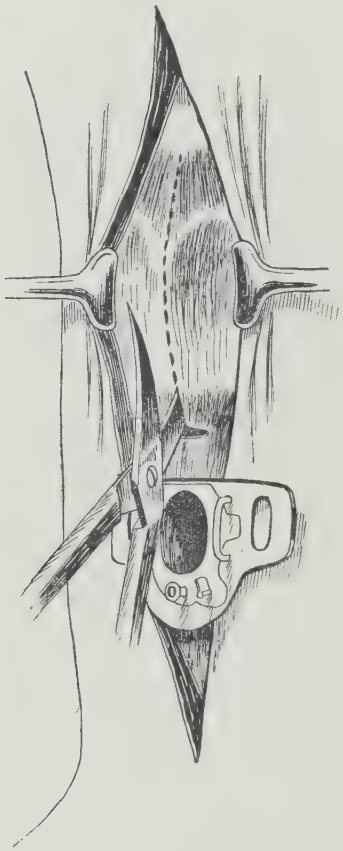
(*d*) Cutaneous incision: The incision should be made in the anterior median line, and should extend from the os hyoides above to the sternoclavicular notch below (Fig. 339). There are but few structures of importance which are encountered in this region, excepting a small amount of areolar tissue and the anastomosis of the inferior laryngeal arteries in the median line. These arteries are encountered at either the inferior border of the thyroid cartilage or the superior border of the cricoid cartilage, hence it may not be necessary to cut them, as they can be pushed aside. There are no serious objections to severing them, but if this is done it is better to locate them and tie them off with absorbable catgut on either side of the median line before dividing them. The venous oozing may be controlled by pressure, or, if too profuse, the venous trunks may be ligated.

(*d*) Incision of the thyroid cartilage: This should be done in the median line with knife or scissors (Fig. 340). The knife is preferable unless the cartilage has become ossified, as the dissection can be carried to the mucous membrane without cutting it. This is important, as the incision through the membrane at the anterior commissure of the glottis should be exactly in the median line, as otherwise one of the cords will be injured.

(*e*) Incision through the mucous membrane: First locate the median line at the anterior commissure. If in doubt, begin the incision at the upper limit of the wound, and cut downward to the anterior commissure. The knife should then be inserted through the incision and

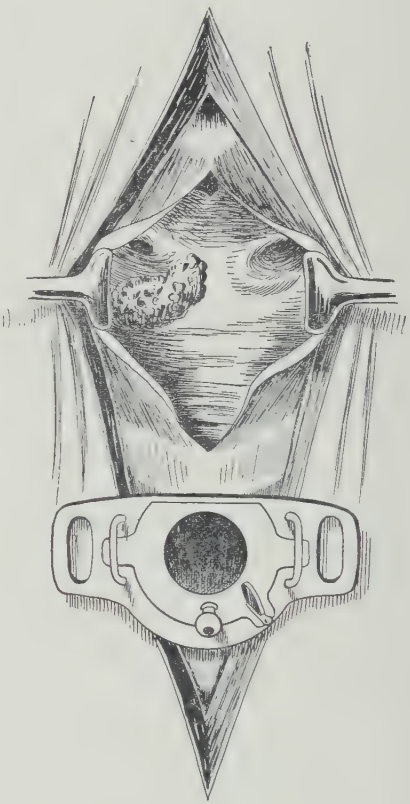
between the cords, and the incision at the commissure made from within outward. In this way the cords will not be injured. The incision is then extended to the lower limit of the thyroid cartilage.

Fig. 340



Laryngofissure. Tracheotomy has been performed, a cross-puncture at the lower border of the thyroid made, and the scissors blade introduced through it preparatory to making the incision through the anterior commissure of the thyroid cartilages. (After Moure.)

Fig. 341



Laryngofissure (thyrotomy) completed, the tumor exposed ready for removal. (After Moure.)

(*f*) The larynx should then be opened by retracting the two thyroid cartilages from the median line (Fig. 341). This is done by the assistants with retractors.

(*g*) Removal of the growth: Having completed the laryngofissure, and having separated the incised thyroid cartilages, the location and character of the growth should be studied. The growth may be removed through the laryngofissure with a snare, scissors, or knife. The better surgical procedure is with the knife or scissors, as with either of

these instruments the scope of the operation is entirely under the control of the operator, whereas with the snare the depth of the cut cannot be accurately estimated.

(*h*) Hemorrhage: The hemorrhage in the preliminary part of the operation, *i. e.*, the laryngofissure, is comparatively slight, as it is controlled by pressure and ligatures as the bleeding points appear. In the removal of the growth, however, there may be considerable hemorrhage both during and after the operation. This is easily controlled with artery forceps or with the actual cautery applied to the bleeding areas. The hemorrhage which occurs after the patient becomes conscious is expectorated, and causes little or no trouble. During the operation the patient's head should hang over one end of the table, which should be lowered to prevent aspiration of blood into the lungs.

(*i*) Closure of the laryngofissure: Having removed the neoplasm (or foreign body), the thyroid cartilages are reunited with an absorbable ligature. The coaptation of the cut edges of the cartilages should be carefully done. If, for instance, one side is higher than the other the vocal cords at the anterior commissure will not approximate on the same level, and vocalization will be somewhat modified.

(*j*) Closure of the cutaneous wound: This should be done with simple sutures about one-fourth of an inch apart, and the whole covered with plain sterile gauze. The tracheotomy tube may be removed in twenty-four hours or at the end of the operation, and the wound entirely closed. At the end of from three to six days the stitches should be removed. At this time the wound should be thoroughly healed, little additional attention being required.

Laryngofissure is also the preliminary step in the treatment of stenosis of the larynx, in which the tracheotomy tube with the upward extending rubber tube in the chink of the glottis is used.

3. **Subhyoid Pharyngotomy.**—Subhyoid pharyngotomy for the removal of malignant neoplasms of the larynx is rarely used. There are cases, however, when it should be elected for this purpose in preference to any other method.

Indications: The indications for subhyoid pharyngotomy are few, and it is used chiefly in cases of extrinsic malignant neoplasms of the larynx, and in cases complicated by extension to or by origin in the pharynx. They are as follows:

(*a*) When the growth is situated in the epiglottis or other of the higher portions of the larynx.

(*b*) When the growth is situated in the upper portion of the larynx and involves the pharyngeal wall.

(*c*) When the malignant growth begins in the pharynx and extends to the supraglottic (extrinsic) portion of the larynx.

Technique.—(*a*) Place the patient under chloroform or ether anesthesia per rectum or mouth after the usual preliminary preparations.

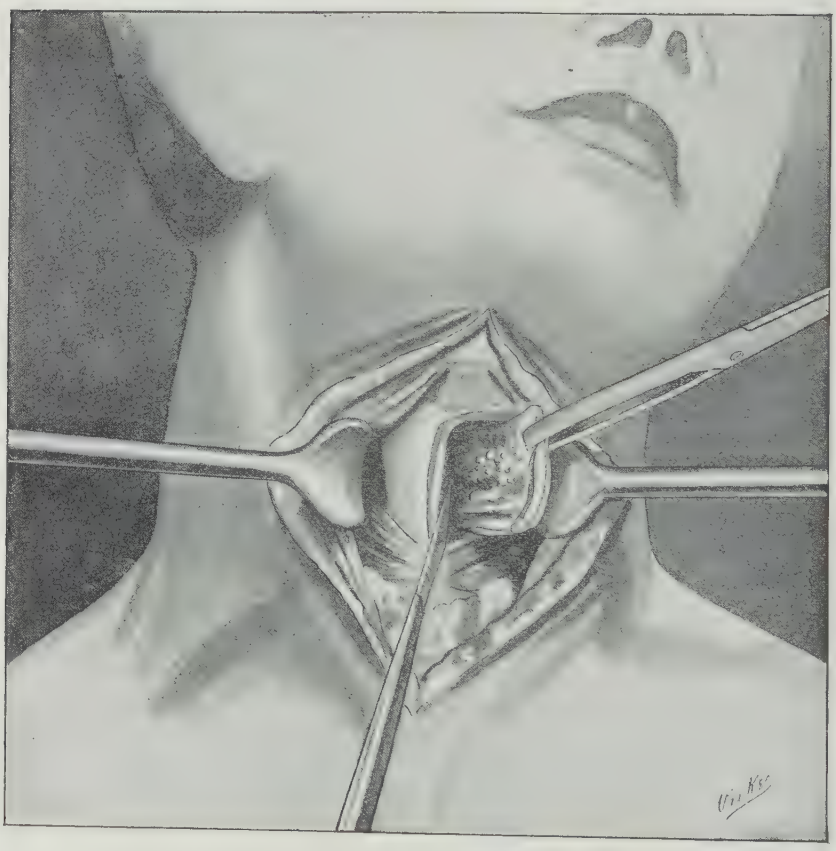
(*b*) Prepare the neck and face by cleansing, etc.

(*c*) Elevate the shoulders of the patient by placing a sand pillow under them, and draw the head well backward to bring the hyoid region into

easy access. Also elevate the foot of the operating table to prevent blood and secretions entering the trachea while the reflexes are abolished by the anesthetic.

(*d*) Make a transverse incision through the skin after Kocher's method, beginning about ½ inch below the inferior border of the hyoid bone, and extend it from the anterior border of the sternocleidomastoid muscle

FIG. 342



Hemilaryngectomy for intrinsic carcinoma of one-half of the larynx.

to the corresponding point on the opposite of the neck. The incision should be from 2½ to 3 inches in length. Then make a perpendicular incision in the median line, beginning above the transverse incision, and extending downward to the prominence of the thyroid cartilage.

(*e*) Divide the superficial fascia, in which the anterior jugular vein is found. The jugular vein should be ligated in two places on each side of the neck and severed between the ligatures.

(*f*) Sever all the muscles, including the sternohyoid, on either side of the median line, and just beneath them the thyrohyoid muscles, thus exposing the thyrohyoid membrane to view.

(*g*) With the finger applied to the membrane, explore for the epiglottis, so as to avoid injuring it in the next step of the operation.

(*h*) Incise the thyrohyoid membrane, thus exposing the diseased area to inspection.

(*i*) Carefully inspect the deeper field, beginning at the anterior surface of the epiglottis, for evidences of a malignant growth.

(*j*) Seize the epiglottis with toothed forceps, and gently draw it outward through the wound, securing it with either a suture through its tip or with locked forceps.

(*k*) Traction upon the epiglottis opens the wound and exposes the deeper parts to view.

(*l*) Through the opening all diseased tissue is removed with scissors, knives, and double cutting forceps, some of the surrounding healthy tissue being also included.

(*m*) The wound is now closed by suturing the thyrohyoid membrane, the muscles, and the superficial fascia with absorbable catgut, and the skin with non-absorbable ligatures.

(*n*) The external wound should be painted with tincture of iodine, to prevent stitch abscesses, and a gauze dressing applied.

(*o*) The dressing should be removed in three to five days and renewed. The stitches in the skin should be removed on about the fifth or sixth day.

(*p*) At the end of ten or twelve days the patient should be able to leave the hospital.

4. **Partial Laryngectomy.**—This operation is often spoken of in the literature as synonymous with laryngofissure, which is but the preliminary step in partial and hemilaryngectomy. Partial laryngectomy is a more extensive operation than simple laryngofissure. In laryngofissure only the soft parts and the growth are removed, whereas in partial laryngectomy a portion of the cartilaginous framework is removed with the growth.

Indications.—The indications for partial laryngectomy are somewhat different from those for laryngofissure. For example, it is not indicated for the removal of foreign bodies in the larynx, benign neoplasms, or of cancerous growths, which only involve the soft structures.

The following are the chief indications:

(*a*) When malignant growths are limited to the soft parts on one side of the larynx, and when it is suspected that the cartilage is also involved, a partial laryngectomy may be done.

(*b*) When malignant growth is limited to one side and involves a portion of the cartilaginous framework of the larynx. The removal of the growth and the portion of the cartilage involved is regarded as sufficient to obliterate all traces of the growth. If partial laryngectomy will not obliterate the growth, complete laryngectomy should be performed.

(*c*) If for any reason there is a suspicion of involvement of the deeper structures, partial laryngectomy is indicated.

Technique.—The technique is so little different from that given in laryngofissure, that a detailed description is unnecessary. The chief difference consists in the removal of the affected portion of the cartilaginous framework in addition to the procedures practised in laryngofissure, in which only soft tissues are removed. The additional fact that partial laryngectomy is usually indicated in extrinsic cancers also implies a more serious condition, with earlier glandular involvement. Hence, the anxiety and desire to be certain to include all the diseased tissue, even at the expense of some healthy tissue.

5. **Complete Laryngectomy.**—The removal of the larynx is a formidable and sad procedure. The death rate in the hands of the average operator is high. The condition of the patient, should he recover from the operation, is often pitiable indeed, though this fact does not always appear in the published reports. However, from the patient's point of view he would rather be alive without his larynx than dead with it. Complete laryngectomy may, therefore, be done when simple and less radical measures hold out little or no hope of success.

Indications.—In a general way, it may be said that the total removal of the larynx is indicated in those cases in which the disease involves a large portion of the soft and cartilaginous structures in both lateral halves of the larynx. It may also be indicated when one side is involved in its entirety and there is a strong suspicion that it has also invaded the opposite side.

The following fairly represents the chief indications for complete laryngectomy:

(*a*) The involvement of one-half of the larynx, with a strong suspicion that it has invaded the opposite side, the glands of the neck not being involved.

(*b*) The involvement of both sides of the larynx, especially if the cartilaginous framework is included in the process, the glands of the neck not being involved.

(*c*) The involvement of the extrinsic areas of the larynx on both sides. If the intrinsic portions only, as the vocal cords, are invaded by the cancerous growth, it may be successfully operated on by laryngofissure.

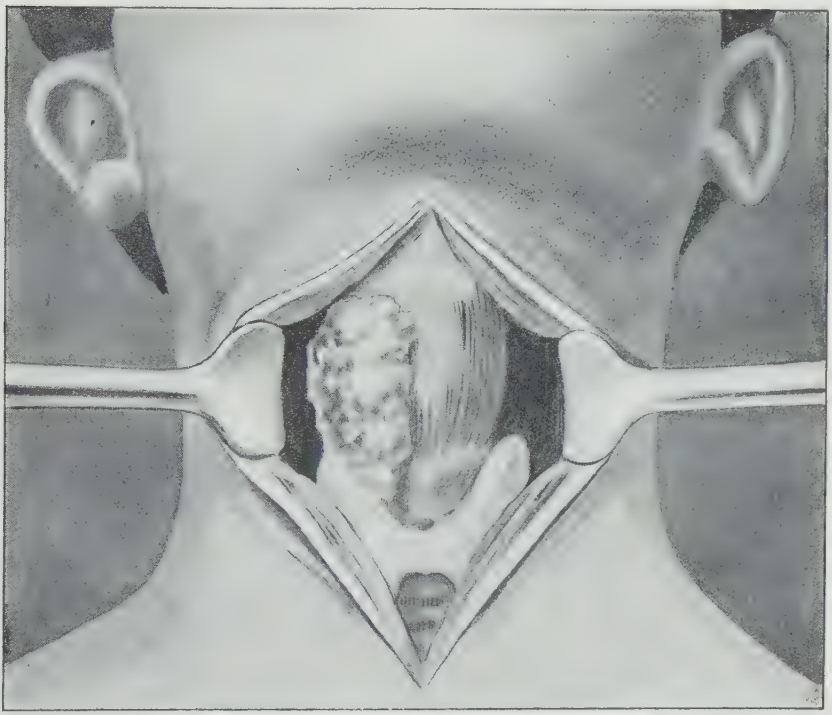
(*d*) The involvement of the extrinsic portions of the larynx on both sides, together with the contiguous tissues, as the pharynx, necessitates the total extirpation of the larynx, together with the other structures that are cancerous.

(*e*) When both sides are extrinsically more or less involved, together with the glands of the neck, total laryngectomy and the ablation of all the lymphatic glands on both sides of the neck are indicated, though a fatal result will probably follow.

Technique.—The method of W. W. Keen is probably the simplest, safest, and most thorough which has yet been devised, and is the one used by me. It is given in the following description:

(*a*) The preparation of the patient for the operation bears an important relation to the success or failure of the surgical procedure. If the patient's general health is bad the prognosis is correspondingly bad. It is essential, therefore, that the general condition of the patient be improved by a short course of forced feeding and tonic remedies. The operation should be performed in the morning, when the vital forces are at their best. On the evening prior to the operation, a cathartic should be given, and a saline given early the following morning. The face (adult male) and neck should be shaved and cleansed the day before the operation, and a moist carbolic acid dressing applied.

FIG. 343



The superficial soft tissues dissected from the larynx preparatory to the complete removal of the carcinomatous larynx. The remaining soft tissues should be dissected from the larynx before separating the posterior wall of the larynx from the esophagus.

(*b*) On the following morning the patient should be placed upon the operating table in the Trendelenburg position, with the foot of the table raised to prevent the aspiration of blood into the trachea. The patient should have his head lowered throughout the operation, and for three days after it.

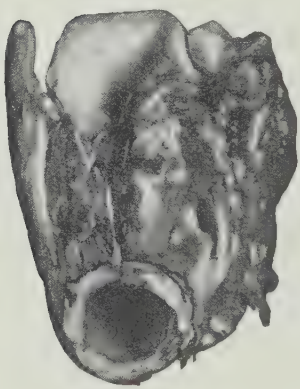
(*c*) Ether vapor, per rectum, as recommended by Cunningham and Stucky, is, perhaps, the best method of inducing anesthesia, as the anesthetist and his apparatus (Cunningham's) are removed from the field of operation.

The anesthetic may be administered by the mouth or the tracheotomy tube (in case a preliminary tracheotomy has been performed), or, if tracheotomy is performed during the operation, it may be given by the mouth until tracheotomy is performed, and then through the tracheotomy tube.

If tracheotomy is not done either before or during the operation, the anesthetic may be given by mouth until the trachea is severed from the cricoid cartilage, and then through the stump of the trachea.

(*d*) The incision should be made in the median line, beginning at the hyoid and extending downward to the sternal notch (Fig. 339). The only vessels of any consequence encountered are the superior and infe-

Fig. 344



Carcinoma of the larynx removed by complete laryngectomy. Posterior view (Author's case.)

Fig. 345

Carcinoma involving all of one and part of the other half of the larynx. Complete laryngectomy was performed by the author by Keen's method without tracheotomy. Anterior view. (Author's case.)

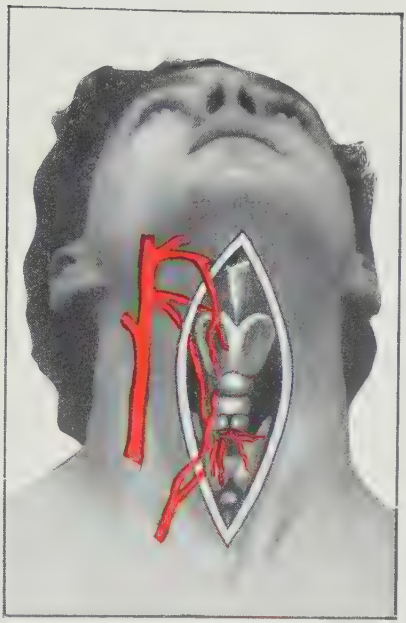
rior laryngeal arteries and their branches. The arteries and veins should be ligated as they are exposed (Plate XIV). The venous hemorrhage may be controlled by pressure, or the larger trunks may be tied.

(*e*) Separate the soft structures (Fig. 343), including the muscles in the median line, and dissect them from the larynx down to the esophagus on the posterior wall of the larynx.

(*f*) Introduce a heavy anchor suture between the first and second cartilaginous rings of the trachea on either side, and pass one end of the suture through the adjacent skin, as shown in Fig. 346. This is done to prevent the trachea dropping into the mediastinum when it is severed from the larynx.

(*g*) Tie the anchor sutures described in the preceding paragraph, and sever the trachea from the cricoid ring of the larynx with a sharp scalpel.

PLATE XIV



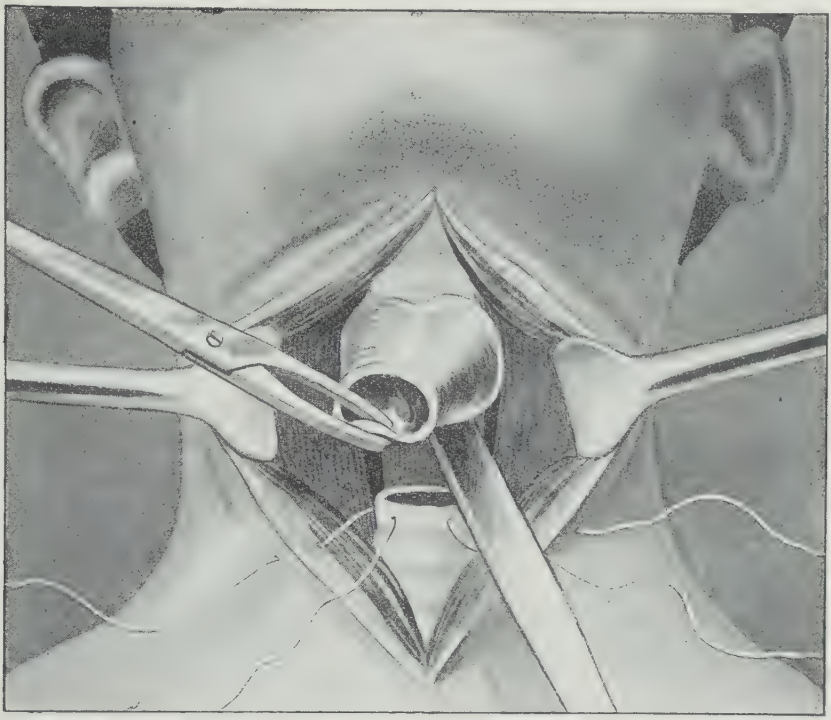
Arteries of the Larynx.

The superior laryngeal and the inferior laryngeal arteries, branches of the superior and inferior thyroid arteries, respectively, supply the walls, glands, muscles, and mucous membrane of the larynx.

If the anesthetic has been given by the mouth, it should be transferred to the trachea.[1]

(*h*) Dissect the posterior wall of the larynx from the esophagus with the finger or blunt instrument, as shown in Fig. 346. This is often a difficult task, as the adhesions are firm. Every effort should be made to avoid tearing the wall of the esophagus, as it is difficult to repair it by suture.

FIG. 346



Complete laryngectomy. The larynx has been severed from the trachea at the junction of the first ring and the cricoid cartilage. The larynx is being separated from the anterior wall of the esophagus by blunt dissection.

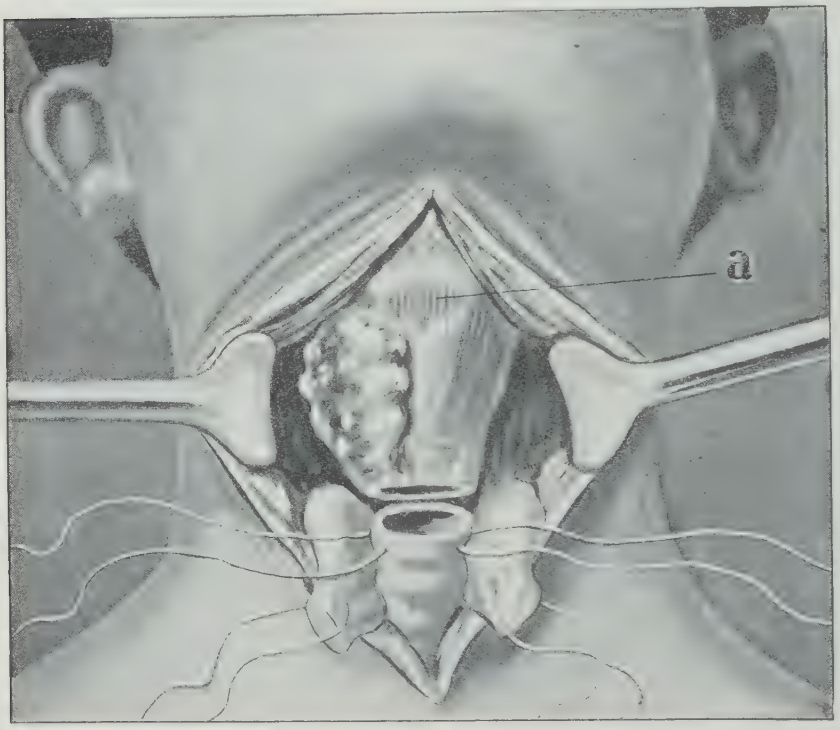
(*i*) Having separated the esophagus from the larynx as high as the arytenoid cartilages, it should be severed from the larynx by transverse incision (Fig. 348).

[1] In this description, it is presumed that the removal of the larynx is done without tracheotomy either prior to or during the operation, as suggested by Dr. W. W. Keen. I performed the operation in this manner in August, 1905, with satisfaction. The larynx and carcinoma thus removed are shown in Figs. 344 and 345. The patient died six days after the operation from exhaustion. He rallied after the operation, progressed very favorably for five days, took food per rectum for four days, and by mouth for one. He was then unable to retain food on his stomach. Rectal feeding was again tried, but was not retained. Death occurred the following day. The patient was fifty years old, and had been a heavy whisky drinker for twenty-five years. The carcinoma was extrinsic and large, and while chiefly limited to the right half of the larynx, it had extended to the left side of the epiglottis. There was no enlargement of the glands of the neck. Only one enlarged lymphatic gland was found, and that was in the glosso-epiglottic space.

(*j*) The only attachment remaining is the thyrohyoid membrane in front. This should also be severed by a transverse incision (Fig. 348). The larynx and the neoplasm are thus extirpated, leaving the pharynx open in front.

(*k*) The lower pharyngeal membrane should now be sutured to the thyrohyoid membrane below the hyoid, as shown in Fig. 348, thus closing the wound in the anterior wall of the pharynx.

FIG. 347



Complete laryngectomy. The thyroid glands turned aside with ligatures through them. The trachea severed below the cricoid cartilage preparatory to dissecting the larynx from the esophagus and other deep soft tissues. Anchor sutures passed through the upper ring of the trachea to prevent the trachea dropping into the mediastinum. *a*, thyrohyoid membrane.

(*l*) The soft tissues should be brought together in the median line by buried absorbable catgut sutures.

(*m*) The stump of the trachea should be securely sutured to the skin, as the breathing must in future be carried on through it.

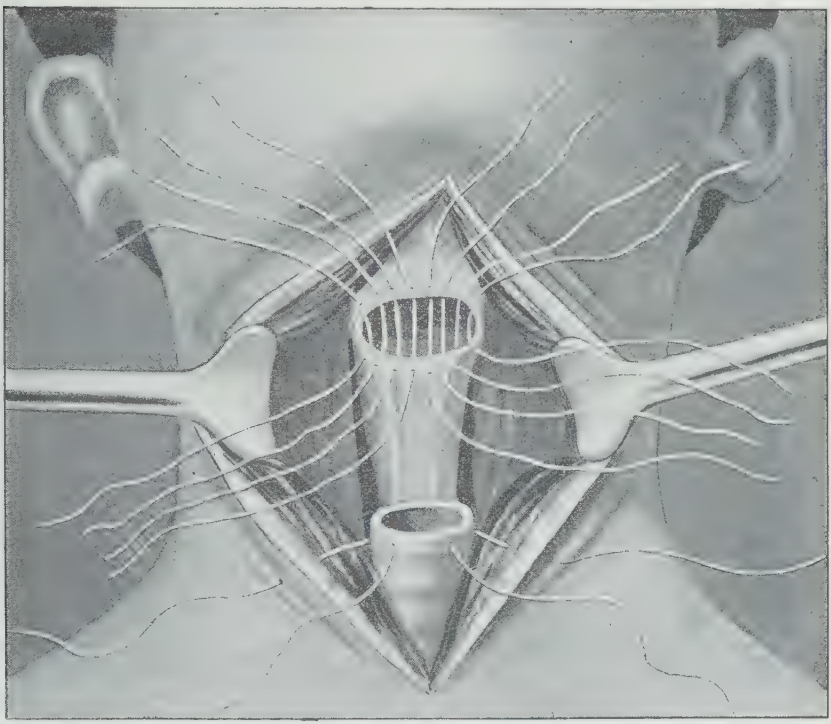
(*n*) The skin should be closed by sutures except around the stump of the trachea, as shown in Fig. 349.

(*o*) A dressing should be applied over the line of skin sutures. A few folds of thin moist gauze should be placed over the tracheal stump to filter and moisten the air inspired through it. This portion of the

dressing should be frequently changed, as it becomes soiled by the secretions coughed out through the trachea.

After-treatment.—Keep the foot of the bed elevated a foot or more for three days, to promote drainage of the trachea, or until the patient can take food by the mouth. Sustain the patient by rectal feeding at intervals of three or four hours for four days. At the end of this time the pharyngeal wound is usually united, and food may be given by mouth. In from twelve to fourteen days, the patient should be able to leave the hospital, if he is not dead.

FIG. 348



Complete laryngectomy. The larynx has been removed, leaving an opening in the anterior wall of the pharynx. The sutures are in position ready to close the wound.

Axioms.—1. Early diagnosis and an early operation in laryngeal cancer means a probable cure.

2. An early provisional diagnosis of cancer may be made if three clinical facts are borne in mind, namely, a patient forty or more years old, complaining of continued hoarseness without cough, with sudden sharp pains in the larynx, pharynx, or ears.

3. The operation of choice should be the one that will insure the complete removal of malignant tumor with the least destruction of normal healthy tissue and the least damage to the function of the larynx.

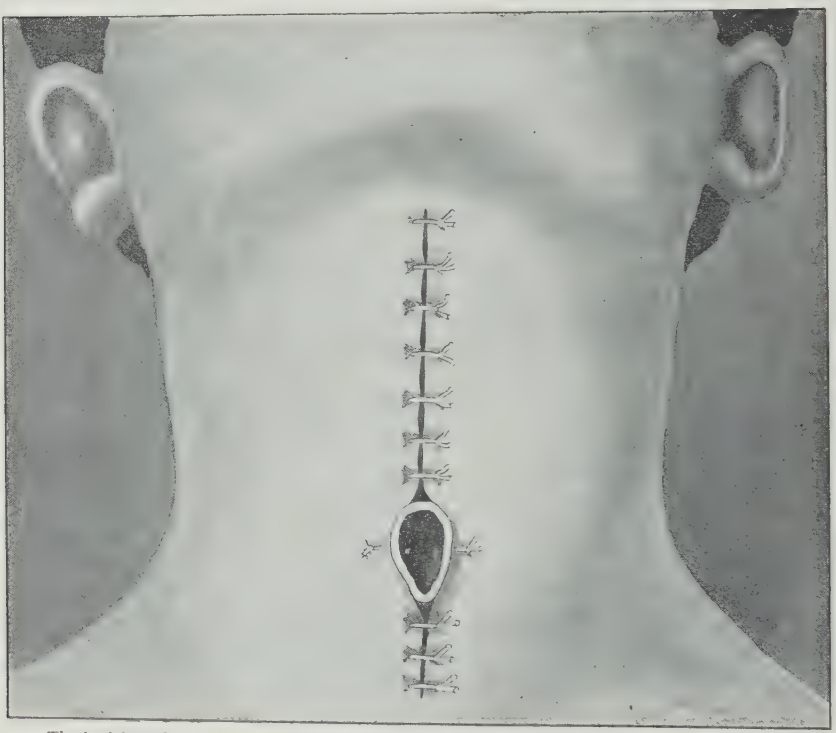
4. Intrinsic cancer of the larynx is successfully treated by laryngofissure, a simple and comparatively safe method.

5. Complete removal of the larynx is a formidable and dangerous operation, only suited to extensive involvement of the soft and the cartilaginous portions of the larynx in both lateral halves.

6. Extensive involvement of the larynx and of the adjacent structures means certain death without an operation, and probable death with an operation.

7. If the diagnosis of cancer of the larynx is only made at an advanced stage, the physician is guilty of "ignorance," when it is easy to be "wise."

FIG. 349



The incision after complete laryngectomy. The end of the trachea is sutured to the skin.

Postoperative Considerations.—The surgeon's responsibilities are by no means ended when the operation is completed. There are several conditions which are either present or likely to arise that demand his thoughtful attention. Among them are the following.

1. *Shock and Sudden Death.*—Stoerk attributes death by shock to the severing of the fibers of the inhibitory cardiac branches of the pneumogastric nerve. They are given off, and pass forward to the larynx, thence downward back of the trachea, where they may be injured in separating the esophagus from the larynx and the trachea. It is, therefore, well

to keep close to the posterior wall of the trachea, and to avoid undue manipulation and traumatism in making the separation.

Crile, by experimentation upon the lower animals, arrives at the conclusion that sudden death in laryngectomy and intubation is due to an irritation of the middle and the upper portion of the larynx, the irritation exciting a reflex inhibition of the cardiac branches of the pneumogastric nerve. He therefore recommends a preliminary incision through the cricoid membrane, through which the interior of the larynx may be brushed with a 5 per cent. solution of cocaine. After that is done, the operation of election is continued. He also suggests that an injection of atropine helps to prevent the reflex influence upon the heart. He makes the following distinction between asphyxia and reflex action on the respiratory organs and the heart:

(*a*) In asphyxia there are more or less violent efforts to breathe, the heart momentarily beating stronger; whereas,

(*b*) In reflex disturbances the breathing stops suddenly and the heart immediately becomes weak.

The above distinctions are peculiarly applicable to impending death during intubation in diphtheria and pseudomembranous croup. During intubation, the patient is suddenly asphyxiated, or is thrown into a state of shock, the characteristics of each being given in the above paragraph.

Treatment of Cardiac Reflexes.—(*a*) Instantly lower the head without further manipulation of the larynx.

(*b*) Slap the chest with a cold, wet towel, then immediately dry the surface and repeat the cold applications.

(*c*) Artificial respiration should, in the meantime, be kept up.

Treatment of Asphyxia.—(*a*) Remove the intubation tube or the obstruction to the larynx and clear it of membrane.

(*b*) The patient will then, in all probability, cough out more membrane or obstructing secretions, thus clearing the lumen of the trachea.

(*c*) Reintroduce the cannula (in diphtheria), and no further trouble will be likely to occur.

While the foregoing remarks upon shock and sudden death do not, in all respects, have a direct bearing upon the operation for cancer of the larynx, they nevertheless have an indirect relationship, and may prove of value in the study of this subject.

2. *Inspiration pneumonia* is a common sequel of the operative treatment of laryngeal cancer, and is a frequent cause of death. In laryngofissure, one of the simplest external laryngeal operations, the death rate is about 4 per cent. In complete laryngectomy the mortality from pneumonia alone is much greater.

3. *Rectal Alimentation.*—After complete laryngectomy the patient should be sustained by rectal alimentation for three or four days, after which he may be given food by the mouth. In the simple operations, the rectal feeding may be discontinued somewhat earlier, proportionate to the extent of the operation. Indeed, in simple laryngofissure it may be dispensed with altogether.

4. *The Voice.*—After laryngeal operations, the voice may be good, if the cords are not greatly damaged in the removal of the growth or

the larynx is not removed in its entirety. If the tumor arises from the cords, and has penetrated deeply into their substance, they must be removed, and the voice is consequently weak and otherwise impaired. After laryngofissure for laryngeal cancer, the voice is usually more or less impaired, while in benign growths it is usually very good. After hemilaryngectomy and partial laryngectomy, one cord remains, and gives a husky though useful voice. After complete laryngectomy, when the trachea is stitched to the skin, there is no voice except in rare cases, where the tissues around the tracheal opening are thrown into vibration. When the trachea is stitched to the pharyngeal wound there may be more or less voice. This is obtained by the peculiar conformation of the parts after the healing process is complete. The larynx being removed, the base of the tongue drops backward and downward, approximating the posterior wall of the pharynx. The cavity below the base of the tongue forms an air chamber, which is utilized to force air through the constriction formed by the base of the tongue and the pharyngeal walls, thus throwing the tissues at this point into vibration. The union of the trachea to the pharyngeal wound is not often practised, as the tension is so great that the tissues tear apart, slough away, or undergo gangrenous degeneration.

5. *Recurrence.*—Recurrence of the cancerous growth is common on account of failure to make an early diagnosis. Intrinsic growths are less malignant than the extrinsic, hence recurrence in this variety is not so common.

It may be said, then, that recurrence of laryngeal cancer is largely dependent upon the following factors:

(*a*) Intrinsic cancers of the larynx do not recur as frequently as the extrinsic.

(*b*) Conversely, extrinsic cancers more often recur than the intrinsic.

(*c*) Extralaryngeal cancers, involving the larynx, have a still greater tendency to recurrence.

(*d*) An early diagnosis and operation by laryngofissure, in intrinsic cancer of the larynx, should result in a death rate of only 10 per cent., and 5 of the 10 die of pneumonia rather than of recurrence.

(*e*) Complete laryngectomy in cancer of the larynx was, up to 1889, attended with a death rate of 44 per cent., but since antiseptic surgery and an improved technique have been attained, it is reduced to about 15 per cent. When I speak of a death rate of 15 per cent., I mean death within three years after the operation. Quite a number die within a few months from pneumonia, septicemia, gangrene, exhaustion, or other sequelæ. In still others recurrence brings on a fatal issue.

(*f*) The ligation or injection of the external carotids and their branches should only be done when the cancer is inoperable, as it does not cure, but only holds out the hope of retarding the growth of the tumor by diminishing its nourishment.

(*g*) Tracheotomy should be reserved for inoperable cases in which the cancerous tumor obstructs the breathing and threatens the life by suffocation.

CHAPTER XXXI

FOREIGN BODIES IN THE LARYNX, TRACHEA, BRONCHI, AND ESOPHAGUS[1]

Etiology.—The lodgement of foreign bodies in the air passages is most common in infants and young children, as they have an instinctive desire to test all substances with their mouths. Coughing, laughing, crying, and ineffectual attempts to swallow draw the foreign bodies into the lower air tract. The small caliber of the larynx and air tubes in infants and young children increases the chance of lodgement of foreign bodies. The smaller size of the larynx and air tubes in infants and young children renders the obstruction greater than in older subjects from the same foreign bodies, hence the danger is correspondingly greater in young subjects.

The nature of the foreign bodies ranges anywhere from particles of food to marbles, coins, safety pins, burrs, and false teeth.

Symptoms.—The symptoms of a foreign body in the respiratory passages are those of obstructed breathing, laryngeal, tracheal, bronchial or pulmonary irritation, and inflammation. The patient is suddenly seized with a violent choking and suffocative attack, characterized by cyanosis, aphonia, beads of perspiration on the forehead, and a weak pulse. These symptoms usually subside within a few minutes, but return again in a few hours or days. After the foreign body remains in the larynx for several weeks the spasmodic symptoms cease and the cough, etc., become more constant, often leading to a diagnosis of tuberculosis. A negative finding upon examination of the sputum removes the suspicion of tuberculosis. A positive finding does not, however, exclude a foreign body. A history of spasmodic cough and dyspnea and hoarseness followed by a persistent cough should excite suspicion of a foreign body in the respiratory tract if the patient is a small child. If the foreign body lodges in the ventricle of the larynx or in the subglottic space, hoarseness or aphonia is usually present. When the foreign substance changes its position, or a fresh irritation arises, new suffocative attacks are excited. If the foreign body lodges in the trachea, bronchus, or one of the bronchioles, the voice remains clear. Bronchial rales or pneumonia may subsequently develop. In some instances the movements of the foreign body when in the bronchus may be detected by auscultation (Halstead). Blood-stained expectoration is occasionally a symptom. In some instances all symptoms and physical signs are

[1] Revised by Dr. Chevalier Jackson, whose distinguished work in this field is unique and unequalled.

strangely absent even when the bodies of considerable size are present. Dyspnea, attended with an elevation of temperature, often leads to an erroneous diagnosis of tracheal diphtheria. A laryngoscopic examination may not reveal the foreign body, even though it is lodged in the ventricle of the larynx. By direct laryngoscopy (Fig. 351), a better view of the larynx may be obtained. To Gustav Killian belongs the credit of devising instruments whereby almost all of the respiratory tract may be clearly inspected for foreign bodies. This alone is enough to immortalize him in the scientific annals of medicine and surgery, though he has in many other ways made his name equally famed in rhinology and laryngology.

Indications.—The indications are to remove the foreign body as soon as possible, as it may become dislodged and migrate to a new and more dangerous location. The continued presence of the foreign body may also give rise to considerable local irritation and subsequent edema or septic inflammation. Pneumonia is a rather frequent complication. In prolonged cases serious septic absorption may occur. Cases are recorded wherein the foreign body remained in the air passages for years without causing death. It should not be deduced from this fact that the early removal of the foreign body is not desirable, as the risks attending its continued presence in the air passages are infinitely greater than those incident to its early removal.

The indications are, therefore, to institute proceedings for its removal, either by (*a*) holding the child's head downward and thumping it on the back (a dangerous procedure), the surgeon being prepared to perform a tracheotomy should suffocative symptoms supervene; (*b*) the titillation of the larynx with the finger, in the hope of dislodging the foreign body or of exciting a coughing spasm, during which it may be expelled (a dangerous procedure); (*c*) the indirect removal with instruments by the aid of a laryngoscopic mirror; (*d*) the removal of the foreign body by the direct method with the Killian or the Jackson tubes; (*e*) tracheotomy to relieve the suffocative dyspnea; if cyanosis is marked, tracheotomy may also be done to establish a new avenue of inspection and for the instrumental removal of the foreign body; (*f*) and, finally, the indications are to have a skiagraph made to accurately locate the foreign body. If it is a metallic or bony substance, its location is easily shown; whereas if of vegetable matter, it is less easily shown on the skiagraphic plate, and in any case we must not be misled by negative radiographic findings. Many cases of failure to show even metallic bodies on otherwise excellent skiagraphs have been reported.

Having located the foreign body, practise bronchoscopy or tracheoscopy, and remove it with suitable instruments, by either upper or lower bronchoscopy, upper bronchoscopy being preferable when practicable.

Treatment.—It is generally understood among the laity that pounding a child on the back, especially when held head downward, will often dislodge a foreign body from the respiratory tract. These procedures have, therefore, usually been performed before a physician is called, provided it is known that a foreign body has been inhaled. Even though

PLATE XV

Lower Bronchoscopy.

a, the electric wire supplying the lamp at the distal end of the bronchoscopic tube; *b*, the conduit for aspirating the secretions and blood from the distal end of the tube; *c*, the tracheotomy wound; *d*, the distal end of the tube; *e*, the larynx; *f*, the foreign body; *l l*, the lungs.

the foreign body is not thus removed, the suffocative symptoms often subside within a few minutes, and the incident is often forgotten. This method of procedure is dangerous, as the foreign body may be inspired deeper into the air passages instead of being expelled. If the physician is present, he should prepare to do a tracheotomy if the suffocative symptoms demand it. If the child is in a fairly comfortable condition, it should be removed to a hospital and all arrangements for any emergency be made, before an attempt is made to remove the foreign body. When the symptoms recur a few hours or days later, without the marked strangulation and coughing which characterized the initial attack, the family often sees no connection between the two, and fails to report the occurrence of the first one to the attending physician. If the foreign body assumes a new location, the violent spasmodic seizures are repeated.

If suffocation is imminent, tracheotomy should be performed at once, for, as Chevalier Jackson says, if this is not done, the child may never breathe again. When this is done, it may be necessary to employ artificial respiration even if the foreign body is in the larynx. If it is in the trachea or bronchus, it may not relieve the distress unless the foreign body is expelled through the tracheal wound. As a matter of fact, it is frequently thus expelled the moment the edges of the severed tracheal rings are retracted. If it is not voluntarily expelled, the lining mucous membrane of the trachea should be titillated, a procedure that sometimes causes its expulsion. Having performed tracheotomy, which is not attended with voluntary expulsion of the foreign body, proceed to pass a probe upward through the tracheal wound into the larynx, to locate it if it is there. If lodged in the ventricular pouch or in the subglottic space, its removal is not difficult. Having located it, introduce slender forceps, seize it, and remove it through the tracheal wound.

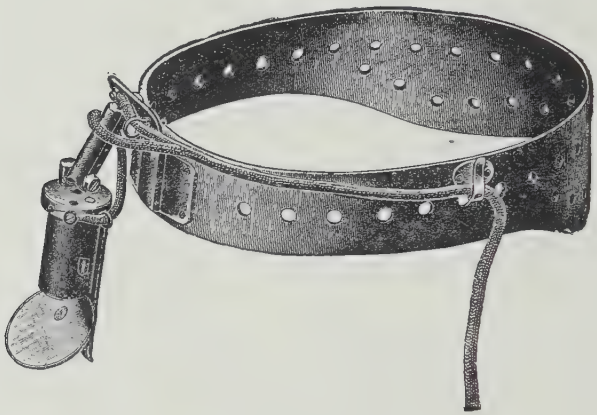
If the foreign body is lodged in the trachea at its bifurcation, it may be easily seen through a tracheoscopic tube introduced through the tracheotomy wound (Plate XV). For illumination, a Kierstein head lamp (Fig. 350) or a small electric lamp at the distal end of the tube, as devised by Jackson (Fig. 351), may be used. If a Killian or Jackson tube is not available, the foreign body may be detected with a probe introduced through the wound, after which slender forceps may be introduced through the wound without a tracheoscope and the foreign body removed. This method is inexact and crude, and should only be used as an emergency measure.

If the foreign body is in one of the bronchi, its removal is more difficult. Indeed, if it is not voluntarily expelled upon making the tracheal opening, or upon titillating the tracheal mucosa, a bronchoscope should be introduced through the mouth or the tracheotomy wound.

I am greatly indebted to Dr. Chevalier Jackson for personal instruction and for the description of the technique of tracheobronchoscopy given in his classical treatise upon this subject. In describing the technique of the various procedures for the removal of foreign bodies from the upper respiratory tract, I have adhered to his methods and largely to the instruments devised by him. In so doing, I am not unmindful

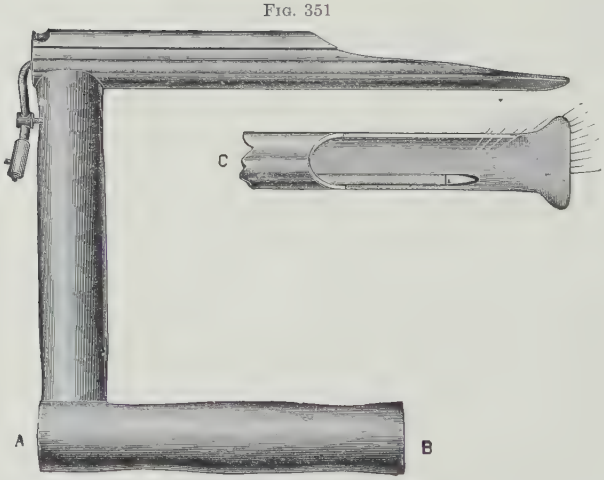
of the fact that the greatest credit is due to Prof. Gustav Killian, of Freiburg, who was the first to remove a foreign body from a bronchus by upper bronchoscopy, and who has, through his writings and demonstrations, made bronchoscopy available to every specialist throughout

Fig. 350



Kierstein's lamp.

Fig. 351



Jackson's slide speculum for direct laryngoscopy. The handle *B* gives great leverage and greatly aids in overcoming the resistance of the muscles at the base of the tongue when the epiglottis and tongue are lifted forward.

the world. Jackson's illuminated bronchoscopic tubes are, however, easier for the inexperienced surgeon to use, and for this reason I recommend them in this work, though the latest apparatus, devised by Killian's assistant, are most ingenous and admirable, and in many instances are better adapted for the work than Jackson's tubes.

Much credit is also due to Dr. Ingals, one of the first Americans to adopt bronchoscopy, for his writings, wherein he reports thirteen foreign bodies searched for or removed by bronchoscopy. Two deaths have followed the removal of foreign bodies in his practice, the cause of death being attributed to reflex irritation of the vagus nerve.

Tracheoscopy and Bronchoscopy.—*Preparation of the Patient.*—If a general anesthetic, preferably ether, is used, the patient should be prepared as for a surgical operation. The morning hour, before the patient has had breakfast, is, therefore, the most favorable time, though in many cases the imminent danger in which the patient is placed leaves no choice in this respect. If time permits, the bowels should be emptied. If the tracheobronchoscope is to be used through a tracheal wound, the neck should be shaved and cleansed. This route, as suggested by Jackson, is aseptic immediately after the tracheotomy, as the instruments may be introduced through a sterile wound; whereas if they are passed a day or two later, after the inevitable tracheotomy wound infection, the danger of septic infection of the deeper air passages is more likely to occur. Upper bronchoscopy should be practised when feasible, and with the skill that comes with practice, tracheotomy will never be necessary except for dyspnea. Any foreign body that has gone down through the glottis can and should be safely brought out by the same route.

The Anesthetic.—Stolid adults tolerate the introduction of the tubes under cocaine anesthesia, whereas more excitable ones, and children, require a general anesthetic. The larynx and trachea may be cocainized by cotton-wound applicators before the introduction of the tubes, whereas bronchi and secondary and tertiary bronchioles can only be reached after the tube is introduced (Jackson). Ether is the safest anesthetic. Ethyl chloride should not be used, as it is not well tolerated by the lower respiratory tract. Profound anesthesia may be induced, though it is an advantage to retain enough of the reflexes for the patient to aid in disposing of the secretions, thus preventing the occurrence of aspiration pneumonia. Morphine, codeine, and heroin, hypodermically, lessen the amount of general anesthetic needed, though they increase the risk of pneumonia by paralyzing the cough reflex (Jackson).

Position of the Patient.—Killian usually passes the tubes with the patient in the lateral recumbent position under chloroform anesthesia. Jackson prefers ether anesthesia, with the patient in the recumbent posture (Fig. 352), as it is less tiresome for the operator to sit than to stand during what is often a prolonged ordeal. The head of the patient is also steadied more readily in this position. Jackson prefers the recumbent posture, also because the patient is in position for tracheotomy should suffocation occur during the attempted upper bronchoscopy. The head should be supported over the end of the table, in Boyce's position, and should be firmly grasped by an assistant, as shown in Fig. 352. The head should not be turned to one side, but should be held exactly in the middle line during introduction of the tubes. If the tube is to be introduced through the tracheal wound, the head should be turned to one side to remove the chin from the axis of the tube.

Introduction of the Tube.—A tube should be selected of the proper length and size to reach the required depth and to correspond with the caliber of the respiratory tract to be explored. The length of the tube will depend somewhat upon whether it is to be introduced through the mouth or through the tracheal wound. The shorter the tube, the clearer will be the field of inspection, though with Jackson's illuminated tubes the length makes no difference; in fact, it is impossible to tell by looking through the tube whether it is 10 centimeters or 80 centimeters in length. The size of the tube will depend upon the age of

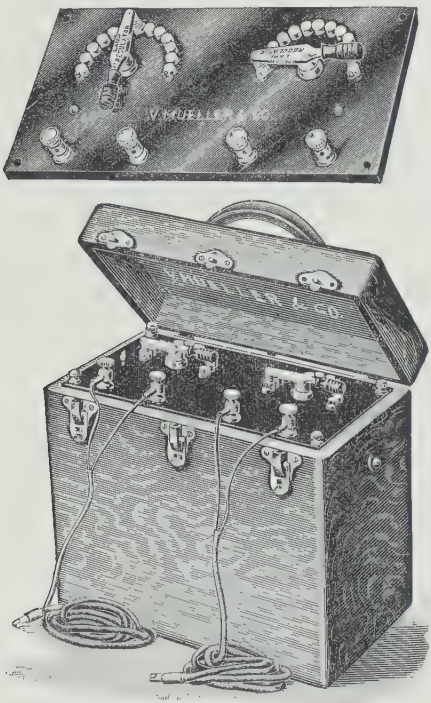
FIG. 352

The position of the patient and assistant in upper tracheobronchoscopy devised by Dr. John W. Boyce. (After Jackson.)

the patient and whether the trachea, bronchus, or one of the bronchioles is to be explored. The secondary and tertiary bronchi may only be explored with small tubes. Having selected a tube of the proper size and length, an assistant should regulate the light and hand it to the operator. The tube should then be passed to the desired depth. This assistant should have entire charge of the tubes and dry-cell battery (Fig. 353), which furnishes the energy for the electric light at the distal end of the tube. The bayonet catch is used by the operator himself to turn the light on and off, as needed. A second assistant stands at the instrument

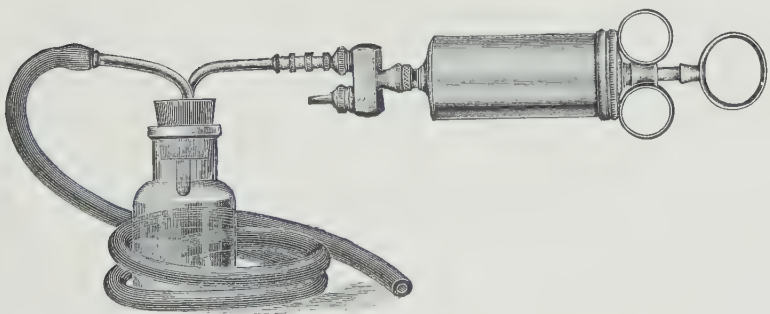
table to pass to the operator, forceps, hooks, and sponge holders armed with little folded gauze sponges, with which the field is kept clear of secretions. The third assistant should hold the patient's head in position.

Fig. 353



Battery for illuminating Jackson's tubes.

Fig. 354



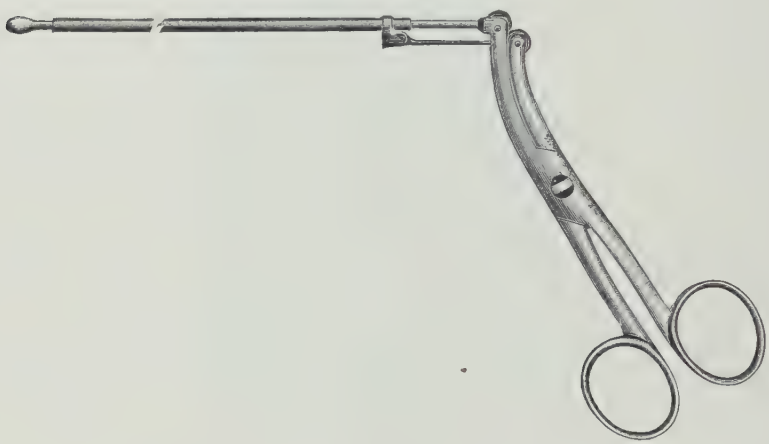
Jackson's exhaust pump for removing secretions in esophagoscopy and rarely in tracheobronchoscopy.

The anesthetist should closely observe the pulse and respiration, as they may stop through reflex irritation excited by the presence of the bronchoscope in the trachea.

Inspection.—The tumor or foreign body should be sought for at the depth of the tube by direct inspection through it. The illumination is brilliant, and a clear view may be obtained in most subjects if the secretions are removed by cotton-wound applicators, or sponge holders.

The Removal of a Foreign Body or Growth.—Long shanked hooks and forceps (Fig. 355) are introduced through the tube, the growth or foreign body seized and withdrawn. It often requires patience and perseverance to accomplish the purpose in hand. If the tube has been either carelessly or roughly introduced, the mucosa may be injured, and the blood will be a worse obstacle to the view than the secretions. It is sometimes necessary to spend an hour or more in exploring the deeper air tract for a foreign body. Even then it may not be located.

Fig. 355



Long forceps for the removal of foreign bodies in bronchoscopy.

Having completed the exploration successfully, the tracheotomy wound, if one has been made, may be allowed to close at once, even though the obstruction to breathing is not completely relieved. The embarrassment which still remains is usually due to the congestion of the respiratory tract in the region formerly occupied by the foreign body, and will disappear in from three to seven days. If the foreign body is not found, or, if found, is not removed, the tracheotomy tube may be left in place indefinitely, or until such time as the foreign body is found or is expelled voluntarily.

Complications and Sequelæ.—When tracheoscopy and bronchoscopy are performed through the mouth under a general anesthetic, pneumonia is occasionally a serious sequela. If performed through the mouth under partial general anesthesia, or under cocaine anesthesia, such a sequela does not so often occur. When performed through a tracheotomy wound under strict aseptic precautions, pneumonia rarely follows except as a result of a septic condition established by the presence of the foreign

body. That is, bronchoscopy *per se*, when performed under good surgical conditions, does not often cause pneumonia.

General Considerations.—According to Killian, foreign bodies in the larynx, trachea, and bronchi may be divided into (1) hard and (2) soft varieties. He still further subdivides them for clinical purposes into (*a*) slender, (*b*) flat, (*c*) round, (*d*) cubical, (*e*) irregular, (*f*) metallic, (*g*) non-metallic, (*h*) friable, and (*i*) those likely to swell. These subdivisions are of clinical significance, because the size, shape, consistency, and chemical composition have much to do with the location and the technique of removing the foreign bodies.

(*a*) Slender objects, as needles, pins, nails, splinters, etc., usually lodge with the point turned upward, and they lie diagonally across the lumen of the tube. Needles and pins usually cause little inflammation; hence, mucus and large granulations are not present to obstruct the view. Slender foreign bodies should be grasped with forceps (Fig. 355) near the point buried in the tube wall, pushed downward to disengage the buried point, and then removed through the bronchoscopic tube. Small nails may be removed with a rod-magnet introduced through the bronchoscopic tube.

(*b*) Flat objects, as coins, buttons, pebbles (flat), usually lodge in the trachea, though small buttons may enter the bronchi. Coins are usually found in adults, as they are too large to enter the lower air tubes in infants and children. Children from three to six years old have a fascination for small flat pebbles. These usually lodge in the trachea near the bifurcation. Flat objects usually stand diagonally across the lumen of the trachea or bronchus, and are easily grasped with forceps. They may be removed by upper bronchoscopy in nearly all cases.

(*c*) Round objects, as glass beads, cherry stones, coffee beans, etc., are frequently coughed up before assistance is called. They remain movable for quite a while, changing position from time to time. As Killian says, they are difficult to grasp with the forceps on account of their shape and the ease with which they elude the forceps, as it pushes the foreign body before it. A bead or other round object is, therefore, more easily removed if it is first pushed down to the bifurcation of the trachea, where it may be grasped with the forceps. Oval seeds, as prune stones, are rough, and are easily grasped with the forceps. When present in children, prune stones are usually near the bifurcation of the trachea, as they are too large to enter the bronchi.

(*d*) Cubical foreign bodies are difficult to grasp with forceps on account of their width. Killian recommends the use of his hook or hook forceps for this purpose. He also recommends lower bronchoscopy (through a tracheotomy wound) after failure by upper bronchoscopy.

(*e*) Irregular objects, as bone fragments, are usually found in adults. When present in children they lodge in the trachea. If small, the fragments may enter the right bronchus. As the bone fragment is usually rendered sterile by cooking, infection attending its presence is somewhat delayed. If allowed to remain in the bronchus or trachea too long, bronchitis, bronchiectasis, pulmonary abscess, or gangrene may develop.

The bone fragments are irregularly flat, and vary in size from 14 to 16 mm. long by 8 to 9 mm. wide.

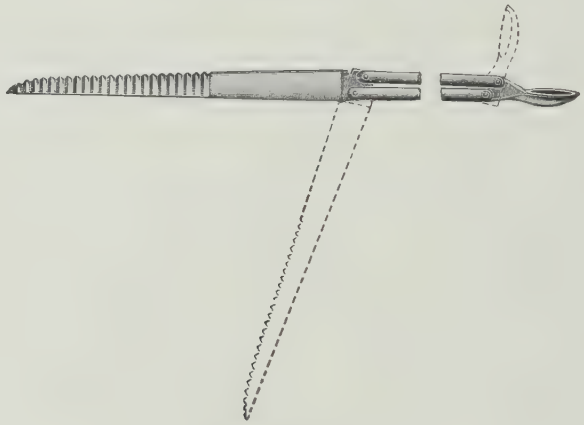
Carious teeth are occasionally aspirated into the trachea or bronchi, and when present quickly excite infective reaction. They should, therefore, be removed as quickly as possible.

Collar buttons are difficult to remove, especially when the larger flat end is turned upward. When the button lies crosswise of the air tube it may be grasped by its neck with forceps or a hook and removed.

False teeth are usually too large to pass below the vocal cords, though Wild reports a case in which a plate with two false teeth entered the left bronchus. It was removed eleven days after the accident by lower bronchoscopy, after being observed by upper bronchoscopy.

(*f*) Metallic substances may be clearly demonstrated by skiagraphy, whereas (*g*) non-metallic substances are less clearly defined. The skiagraph may, therefore, be used to locate the foreign body in many subjects.

Fig. 356



Jackson's extractor.

(*h*) Friable substances, as a fragment of an apple or a swollen and partially disintegrated bean, are difficult to remove, as they break into smaller fragments when seized with forceps. When thus broken the smaller particles are often coughed up, though it is sometimes dangerous to depend upon this mode of ejection, as the particles may be aspirated into one of the secondary or tertiary divisions of the bronchus. Should this accident occur, one lobe of the lung may be deprived of air and rapidly undergo retrograde changes, and become the seat of infection and inflammation. Furthermore, the foreign body is less accessible and more difficult to remove when in one of the smaller bronchi. Killian has constructed forceps, modelled somewhat after an obstetric forceps, with which friable substances, as a swollen bean, fragments of apple, etc., may be grasped and removed without leaving fragments in the air tube. Jackson has devised a bean extractor (Fig. 356), for all soft bodies, such as beans, peanuts, pieces of potato, etc.

Barbed cereal spikes of wheat, rye, etc., are often difficult to remove, as the barbs usually point upward and engage in the mucous membrane when attempts are made to remove them. They have a tendency to descend gradually to the deeper tubes. A forceps that will grasp the entire length of the spike should be used, to prevent fragmentation.

(*i*) A swollen bean, or other substance likely to swell from the absorption of the moisture of the lower respiratory tract, may gradually close the lumen of the bronchial tube (secondary) and thus shut off the air supply to a portion of the lung. The secretions are retained and undergo decomposition, and finally cause serious inflammatory reaction, as violent fever, pneumonia, and atelectasis. According to Killian, 39 per cent. of these cases have died.

Killian has collected 164 reported cases of foreign bodies in the lower respiratory tract, which were treated by bronchoscopy. Of these, 8 coughed the foreign body up. The result is unknown in 5, leaving 159 cases in which the results are known.

Twenty-one (13 per cent.) died, 2 from cocaine poisoning, 2 from stenosis, 16 from pulmonary complications, 5 with the foreign body *in situ*, and 11 in spite of removal.

Upper bronchoscopy was fully successful in 54 cases.

Lower bronchoscopy was fully successful in 63 cases.

Of the first 18 cases occurring in Prof. Killian's practice, one died six months after the removal of the foreign body from severe pulmonary complications.

In two he failed to find the foreign body.

Upper bronchoscopy was performed in 12 cases.

Upper and lower bronchoscopy in 5 cases.

Lower bronchoscopy in 1 case.

Dr. Jackson has extracted 61 foreign bodies from the trachea and bronchi all by upper bronchoscopy. He failed to find the foreign body in the bronchi in three cases where it showed plainly in the radiograph.

Direct Laryngoscopy.—Direct laryngoscopy should be done as a routine procedure in the examination of the larynx, as by it a better view of the parts is obtained, and it is the only means by which the larynx of little children can be examined. It has rendered possible the positive diagnosis or exclusion of laryngeal diphtheria in infants, and the laryngologist who does not use it in these cases does not do his duty. It may be done in the office under cocaine anesthesia, though it is a very disagreeable procedure. Foreign bodies and neoplasms may also be removed by direct laryngoscopy; indeed, this should be the method of choice, especially in papilloma of the larynx, as repeated operations are often necessary to eradicate the disease.

Anesthesia.—Cocaine anesthesia is usually sufficient for office examinations and for the removal of growths and foreign bodies from the supraglottic portion of the larynx. First brush the larynx with a 4 per cent. solution of cocaine to lessen the reflex irritability, and after waiting a minute, swab the larynx with a 20 per cent. solution of cocaine, under the guidance of a laryngeal mirror. One to three such applications at

intervals of from three to five minutes generally induce local anesthesia profound enough to permit of an operation. Cocaine is not well tolerated by children, and should be used with caution. Anesthesia is not necessary for simple inspection of the larynx in children if they are properly held.

Posture of the Patient.—The sitting posture is generally used. The patient should be seated upon a stool 8 inches high; an assistant, sitting behind the patient, should hold his head *forward*, the head rotating back-

FIG. 357

Direct laryngoscopy with Jackson's self-illuminated tube spatula: *a*, electric cord supplying the lamp at the distal end of the spatula; *b*, the conduit for the electric cord; *c*, the tip of the tube spatula holding the epiglottis forward against the base of the tongue; *d*, the conduit for the removal of the secretions and blood from the larynx during examinations and operations by direct laryngoscopy.

ward on the occipito-atlantal joint, to bring the mouth in line with the axis of the trachea. The patient's inclination is to throw his head and neck backward. This defeats the whole object. The head and neck should be pushed as far forward as possible. The assistant should also steady the mouth gag in the patient's mouth. The surgeon should retract the upper lip with the index finger to prevent its being injured between the upper teeth and the slide speculum. The surgeon should stand in

front of and over the patient, with his eye in line with the tube spatula and the larynx (Fig. 357).

Introduction of the Slide Speculum.—Pass the instrument into the throat until the distal end of the instrument is behind the tip of the epiglottis. Then draw the epiglottis forward against the base of the tongue, as shown in Fig. 357. If the spatula is placed too low, against the cricoid ring, the patient has a pronounced sense of suffocation; whereas if the instrument is withdrawn a little higher the dyspnea is relieved and the patient breathes with a "brassy" tubular sound.

Examination through the Slide Speculum.—Forcibly draw the epiglottis forward against the base of the tongue to bring the anterior portion of the larynx into view. This is very difficult to do in some patients and comparatively easy in others. If the illuminated instrument is used, the light should be turned on before introducing it into the mouth. If a non-illuminated tube is used, a Kierstein head lamp should be utilized to illuminate the larynx.

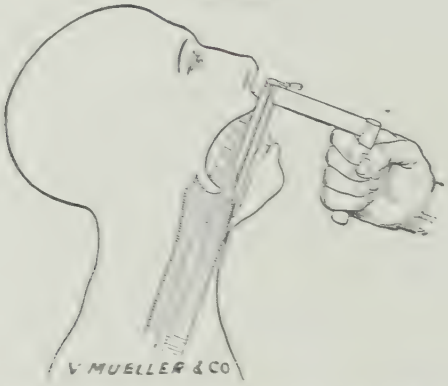
Upper Tracheobronchoscopy.—Upper tracheobronchoscopy is used for diagnostic and therapeutic purposes. By it the condition of the trachea, bronchi, and bronchioles may be observed, and treated by cotton-wound applicators moistened with the medicine. Jackson has observed and successfully treated ulcers of the trachea by upper tracheobronchoscopy. Persistent cough that resisted all other methods of treatment was quickly cured when the diseased tracheal mucous membrane was brushed with a mild solution of the nitrate of silver, argyrol, etc., *via* the tracheobronchoscope. Foreign bodies in the trachea, bronchus, or one of the smaller bronchioles may be diagnosticated and removed through the tracheobronchoscope.

Preparation of the Patient.—If a general anesthetic is to be given, the patient should be prepared as for a major surgical operation if time permits.

Anesthesia.—A general anesthetic, preferably ether, should be administered. The larynx, trachea, and bronchi should also be brushed with a 20 per cent. solution of cocaine. The larynx may be brushed with cocaine before the introduction of the bronchoscope, and the trachea and bronchi as the tube is passed downward. The local anesthetic should not be administered until the general has attained its full effect, as it is safer to preserve the reflexes, so that the patient will aid in disposing of the secretions; otherwise, aspiration pneumonia may result. The use of cocaine in the larynx and trachea prevents the reflex phenomena due to irritation of the vagus nerve. After the bronchoscope is introduced the anesthetic should be given through the tube or by rectum after Cunningham's method. Dr. T. Drysdale Buchanan has devised an anesthetic attachment (Fig. 359) to the Jackson bronchoscope, and Dr. Jackson now uses this. The anesthetic tube ends in the proximal end of the tube and chloroform is blown in from a dosimetric apparatus at each inspiration of the patient. The anesthetic is started with a mask in the ordinary way and preferably with ether. The new method requires an anesthetist familiar with the apparatus. A Brophy vaporizer may be used.

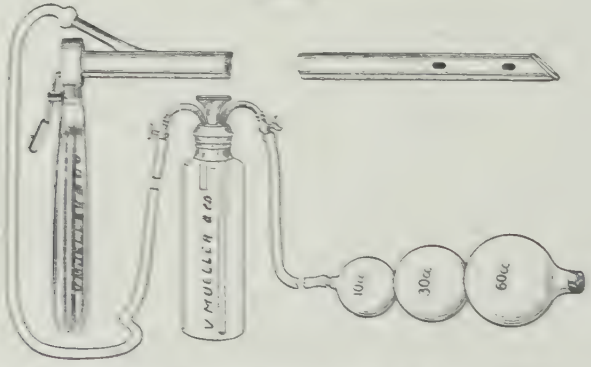
Position of the Patient's Head.—Have an assistant seated on a stool at the right side of the head of the patient, with his left foot on a low stool. The patient's head and neck are drawn beyond the end of the table, and are supported and controlled by the assistant. His right arm is passed beneath the neck of the patient, the hand grasping the

FIG. 358



mouth gag and side of the face. The assistant's left arm rests upon his left knee, and his hand supports the patient's head. The head and neck are thus under the complete control of the assistant (Fig. 352). By raising his right arm, the neck is raised; by raising the left hand, the

FIG. 359

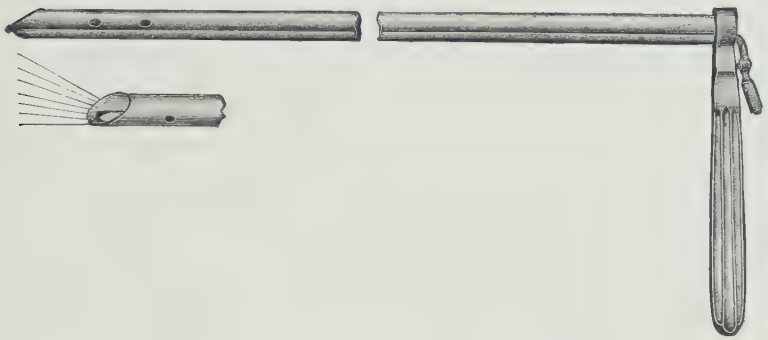


Dr. T. Douglas Nathanson's anesthetizing dosimetric attachment for Jackson's bronchoscope. The chloroform is delivered into the proximal end of the bronchoscope at each inspiration.

head is raised, and by reversing the movements of the arm and hand, the opposite effects are produced. With the right and left hands the head may be rotated on its vertebral axis. The foot of the table should be 15 inches lower than the head, and the top of the footstool 26 inches lower than the top of the table.

Introducing the Slide Speculum.—The slide speculum should be introduced to expose the chink of the glottis while the tracheobronchoscope (Fig. 360) is being introduced. This procedure is identical with that described in the section on Direct Laryngoscopy, the only difference being the recumbent posture of the patient and the use of the slide speculum spatula. Jackson's slide speculum (Fig. 351) is so constructed that it may be easily removed after the tracheobronchoscope has entered the trachea.

FIG. 360



Jackson's self-illuminated tracheobronchoscope.

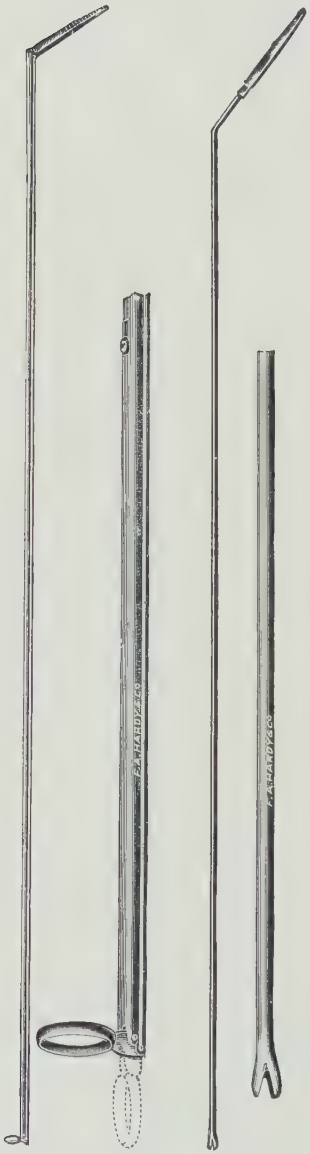
Introducing the Tracheobronchoscope.—Having properly introduced the slide speculum and exposed the cords of the larynx to view through it, the tracheobronchoscope is introduced through the tube spatula to the larynx. The light is turned on and regulated by an assistant before handing it to the operator and the operator's eye is placed at the proximal end of the tracheobronchoscope to watch the respiratory movements of the vocal cords. The tracheobronchoscope should be passed through the glottis during an inspiratory movement of the vocal cords, as they are separated at this time.

Having passed the vocal cords and a good distance into the trachea, the slide speculum should be separated and removed from the mouth after the thimble gag (Fig. 371) has been inserted to prevent the patient biting the bronchoscope. The slide speculum being of thick metal does not need it. Wide gagging renders exposure of the larynx and passing a bronchoscope almost impossible.

The tracheobronchoscope resting in the angle of the mouth and trachea should be pushed downward (cocaine being applied to the mucous membrane with a long sponge holder) until it reaches the foreign body, morbid process, or the bifurcation of the trachea. The tracheobronchoscope should rest in the right angle of the mouth. The entire head is moved to the right when the left bronchus is to be entered, and to the left when the right bronchus is to be entered. The operator should constantly guard the upper lip of the patient with his index finger, to prevent it being pinched between the upper teeth and the bronchoscope, and for this purpose and the steadying of the bronchoscope the

little and adjoining finger of the left hand are kept in the patient's mouth while the thumb and index fingers steady the bronchoscope.

Fig. 361

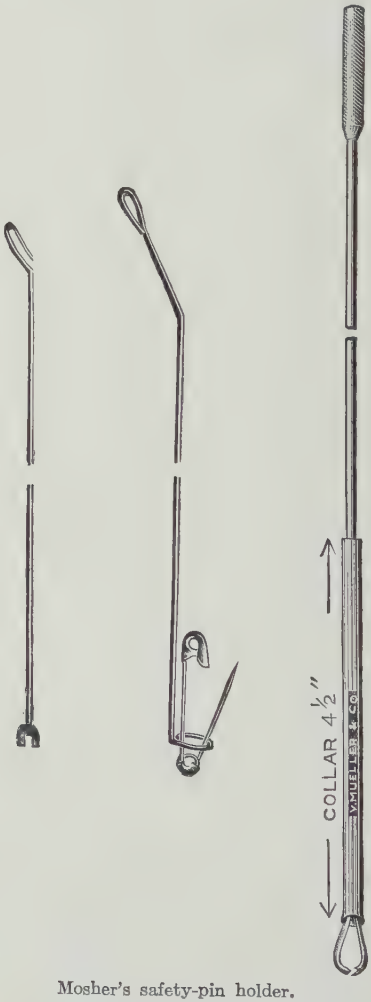


Safety-pin closer.

Fig. 362

Jackson's safety-pin forceps for holding the ring of an open safety-pin securely while the forceps carries the pin down into the stomach, where the pin is turned over for safe removal. Employed in cases of open safety-pin lodged point up in the esophagus.

Fig. 363



Mosher's safety-pin holder.

Having entered the right or left bronchus, the tube is passed downward, the operator watching for the secondary bronchi, morbid lesion, or

the foreign body. By using the smallest-sized bronchoscope the terminal bronchioles may be explored for abscess or other morbid lesion, and if the diseased area is not accessible to bronchoscopic treatment, it may be accurately diagnosticated and located and operated through the chest wall by a general surgeon.

The Removal of the Secretions and Blood.—In cases of very excessive secretion the secretions and blood may be removed with Jackson's pump or aspirator (Fig. 354), which is attached to the conduit for this purpose. An assistant should have entire charge of the aspirator, and use it as directed by the operator. Ordinarily mounted long sponge holders are used to remove the secretions. According to Ingals, the preliminary use of atropine prevents excessive secretions. It also guards against reflex shock. According to Jackson, morphine lessens secretion very much, and it also lessens the cough, but is not as safe as atropine, because it prevents the patient ridding his air passages of secretions and infective materials after the bronchoscopy is over.

FIG. 364

Jackson's forceps, curved jaws.

FIG. 365

Jackson's forceps, cupped jaws.

FIG. 366

Specimen forceps tip to fit universal handle. The side jaw will bite into a flat lateral wall. The cross forms the bottom of a basket to hold the tissue removed.

The Removal of Foreign Bodies.—Variously shaped forceps, hooks, screws, etc., are used to remove foreign bodies (Figs. 361 to 365).

Topical Applications.—Ulcers and other local morbid lesions of the mucous membrane of the trachea and bronchi may be brushed with a weak solution of the nitrate of silver through the tracheobronchoscope.

Remarks.—The trachea and bronchi are elastic and expansile, and tolerate the straightening and dilatation with the bronchoscope.

The illuminated tubes should be boiled after the electric light carrier is removed. The light carriers should be immersed in alcohol. The unilluminated tubes should not be boiled, as the lustre of the interior of the tube is thus destroyed, and its capacity to carry the reflected rays from the head lamp is diminished.

Do not use instruments in lower bronchoscopy that have just been used in upper bronchoscopy. Have freshly sterilized instruments ready for the purpose. Have sterile lamps in a sterile tube ready for use should a lamp burn out.

The patient's head and face should be prepared as for a major opera-

tion about the head. The teeth and mouth should be cleansed with soap and alcohol. The operator and assistants should be dressed in sterile gowns and caps, a precaution especially necessary in handling the long instruments.

The patient should be allowed to sit up as soon as possible, to prevent the occurrence of pneumonia.

Lower Tracheobronchoscopy.—Lower tracheobronchoscopy consists in introducing the tracheobronchoscope through a tracheotomy wound, as shown in Plate XV.

Indications.—Lower tracheobronchoscopy is indicated when direct laryngoscopy or upper tracheobronchoscopy fails. A larger tube may be used in lower bronchoscopy, an advantage in removing large foreign bodies, though Jackson states that he has never yet performed a tracheotomy for the purpose of lower bronchoscopy, having always been able to remove the foreign body through the glottis. He believes that any foreign body that has gone down through the glottis can be and should be removed by the same route.

Position of the Patient.—Primary lower bronchoscopy should always be done in the dorsal position, as tracheotomy is to be performed. The patient should be placed in Rose's position, with the head extended beyond the end of the table.

FIG. 367

Trousseau's dilator.

Low Tracheotomy.—Low tracheotomy should be performed, as the chin is thus farther removed from the operative field and is not so much in the way of the long instruments. The tracheobronchoscope may, however, be introduced through a high tracheotomy wound.

Stop all bleeding before introducing the tracheobronchoscope.

The trachea should be swabbed with a 20 per cent. solution of cocaine through Trousseau's dilator (Fig. 367).

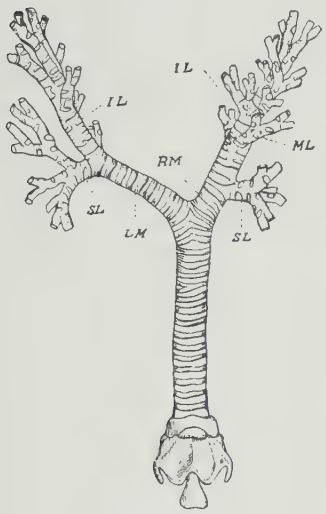
If the right bronchus is to be entered, have the patient's head turned to the right and *vice versa.*

Introduction of the Tracheobronchoscope.—Jackson's illuminated short tracheobronchoscope should be introduced through the tracheotomy wound, the operator's eye being at the proximal end of the tube watching for the bifurcation of the trachea (Plate XV). The end of the bronchoscope usually lodges against the bifurcation, so that both bronchi are visible. Lateral pressure in either direction will allow the tube to pass into one of the bronchi. The moment the tube enters the bronchus, cough is excited. A cotton-wound applicator moistened with a 10 per

cent. solution of cocaine should be applied through the tube and the tube passed to the secondary bifurcation (Fig. 368, *SL*). When a secondary bronchus is entered cough is again excited, and cocaine should be applied as before. It is impossible to maintain anesthesia deep enough to abolish entirely the cough reflex for any length of time, unless rectal anesthesia is used, and even then it is not advisable to abolish all the reflexes, as the patient is thereby subjected to the danger of aspiration pneumonia.

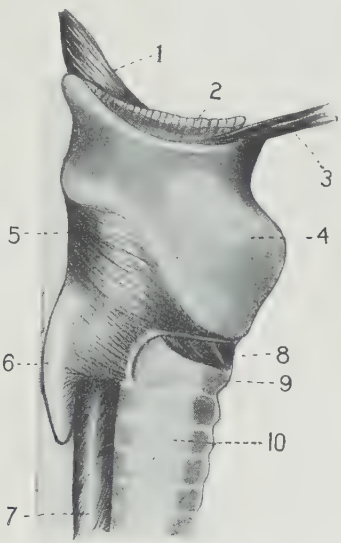
Having introduced the tracheobronchoscope, the foreign body and morbid lesions should be studied, treated, or removed.

Fig. 368



Tracheobronchial tree: *LM*, left main bronchus; *SL*, superior lobe bronchus; *ML*, middle lobe bronchus; *IL*, inferior lobe bronchus. (Jackson.)

Fig. 369



Pouch of the posterior wall of the hypopharynx.

After-treatment.—The tracheotomy wound should not be sutured except at its upper and lower angles. The tracheotomy tube should be worn for a few days, but should be abandoned before the patient leaves the hospital. The tracheotomy wound should be cleansed every three hours with a warm 1 to 5000 bichloride solution. The wound should heal from the bottom, beginning with the severed tracheal rings. If the fleshy portion of the wound tends to heal first, it should be prevented.

Diverticulum or Pouch of the Hypopharynx.—The inferior constrictor muscle of the pharynx forms the posterior and lateral walls of the hypopharynx, and it is in the median or posterior wall of this muscle that the pouching occurs. The lower fibers are attached to the cricoid cartilage and extend in a horizontal direction. The remainder of the muscle fibers radiate in an upward and median direction (Fig. 369), and it is in the central and lower portion of this part of the muscle that the pouching occurs. Various theories have been advanced to explain the pouching

in this region, but the one advanced by Wilson is probably correct, namely, the occasional congenital absence of muscle fibers in this region. He has found the muscle fibers absent in a considerable percentage of the cadavers examined by him. When the pouch is present it may be the seat of lodgement for a bolus of food or a foreign body. When such a condition is present, it may be examined by direct pharyngoscopy with the Killian or Jackson tube spatula and the food or foreign body removed through the same instrument.

Spasm of the inferior constrictor muscle, especially the circular portion which forms the mouth of the esophagus, may occur and prevent the swallowing of food for a few hours or days.

In attempting esophagoscopy in a patient in whom the pouch is present, the esophagoscope may enter the pouch and lead to the erroneous impression that the esophagus is closed by stricture. A careful observation and manipulation should lead to a correct diagnosis.

ESOPHAGOSCOPY; FOREIGN BODIES IN AND STRICTURES OF THE ESOPHAGUS

The examination of the esophagus through the mouth is now an established procedure, and should be considered in connection with bronchoscopy, as foreign bodies may lodge in either tube. The differential diagnosis between a foreign body in the trachea or bronchi and the esophagus must, therefore, be made. Not only this, but the foreign body should be removed, whether it is in the bronchi, the trachea, or the esophagus. A brief description of esophagoscopy will, therefore, be given in this work.

The sizes of tubes required, according to Chevalier Jackson, are, for infants, 7 mm., and for adults, 10 mm. in diameter.

The normal appearance of the esophageal lumen with the Jackson self-illuminated tubes is a whitish grayish pink, in strong contrast to the white and red rings of the tracheal membrane.

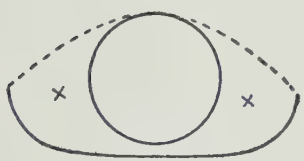
Examination of the Upper End of the Esophagus.—This is the easiest of all the examinations with the straight tubes, and is accomplished by the same technique as described under Direct Laryngoscopy. According to Jackson, the slide spatula speculum (Fig. 351) should be passed back of the base of the tongue until the epiglottis appears, after having cocainized the *introitus esophagi* with a 10 per cent. solution. Having engaged the tip of the epiglottis, a straight cotton-wound applicator, dipped in a 10 per cent. solution of cocaine, should be passed through the slide speculum and applied to the epiglottis, the laryngeal and the esophageal orifices; a few minutes should be allowed for anesthesia to supervene. The speculum is then passed down back of the epiglottis and the cricoid cartilage, and lifted forward against the base of the tongue. The larynx and the esophageal depression are thus brought into view. The spatular end of the speculum is inserted into the esophageal depression to a point below the arytenoid cartilages,

and far enough to engage the posterior portion of the cricoid cartilage. The cartilage should then be lifted forward, thus exposing the pyriform fossæ and the esophageal lumen.

ESOPHAGOSCOPY

According to Dr. Chevalier Jackson, preliminary to passing a tube into the lumen of the esophagus the upper end of the esophagus should be examined, as described in the preceding paragraph, to learn the pathological conditions present in this region. This procedure will prevent the making of a false passage through an ulcerated surface and will locate a foreign body if present at the entrance of the esophagus. In passing the long tube extreme gentleness should be practised. If the tube does not readily pass, it is either not correctly placed or it is improperly directed. The tube should be lubricated with sterile vaseline. The proximal end should be held lightly with the right hand, the handle directed horizontally to the right. The forefinger of the left hand is passed into the right glosso-epiglottic fossa, posteriorly to the lateral glosso-epiglottic fold and posteriorly to the tense pharyngo-epiglottic fold, and, if possible, into the right pyriform sinus.

Fig. 370



Schema showing relation of the cricoid cartilage (the circle) to the posterior hypopharyngeal wall, in the dorsally recumbent patient, observer looking down the esophagus. The pyriform sinuses are at the positions marked *X*.

Fig. 371

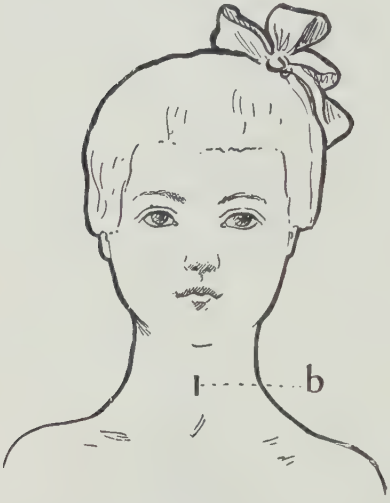
Thimble gag or bite block for bronchoscopy and esophagoscopy.

The tube should then be made to follow the same route, while the finger slides toward the median line and lifts the tongue and anterior pharyngeal tissues upward (dorsal decubitus). When the cricoid cartilage can be reached, which is possible only in children, it is better to lift upon it directly rather than upon the soft tissues. When possible, as it usually is in adults, the cartilage should be lifted indirectly by traction upon the tissues at the extreme point reachable with the finger, often the right glosso-epiglottic fossa. With practice the whole instrument is pointed by the operator's general sense of direction into the axis of the pyriform sinus, just as a billiard cue is used. (Jackson.)

The head of the patient should be held in extreme extension with the mouth widely open, as shown in Fig. 357.

After the *introitus* is passed the obturator is removed, and the cord is attached to the light carrier by the bayonet fitting. The tube must be guided by the eye so as to follow the esophageal lumen by sight. In the thorax the lumen opens up at each inspiration, when the axis is easily followed. After passing the *introitus*, the head of the patient should be raised slightly to prevent the tube pressing on the trachea. The obturator should never be used in case of foreign bodies, as the foreign body may be overridden. The right pyriform sinus is found by sight and the general sense of direction and the well-greased esophagoscope is insinuated through it.

Fig. 372



The probable position assumed by a penny when lodged in the subglottic space.

Fig. 373

The position assumed by a penny, as shown by skiagraphy, when lodged in the mouth of the esophagus of a child, aged three years. (Author's case.)

The entire lumen of the esophagus may be examined for stricture or other pathological lesion, and for foreign bodies. When a foreign body is found it may be removed as by bronchoscopy. By using a longer tube almost the entire surface of the stomach may also be inspected with great clearness of illumination with Jackson's self-illuminated gastroscope.

In one of my cases the skiagrapher reported the foreign body, a penny, to be located at the bifurcation of the trachea. As it was impossible for me to get to the studio to examine the plate, I acted upon his diagnosis and attempted to locate the foreign body in the trachea. At one time I passed the tube into the esophagus and heard a slight metallic click. Further search failed to elicit the metallic sound. When I viewed the skiagraphic plate a few days later I found the shadow of the penny on a level with the cricoid cartilage, instead of at the bifurcation of the trachea, as reported by the skiagrapher. Nine days after the attempted removal

by bronchoscopy the penny was passed per rectum, thus showing the penny to have been in the upper portion of the esophagus, from which place it was probably dislodged at the time I heard the metallic click. Another point of diagnostic interest in this case was the position of the penny. Its flat surface stood at right angles to the vocal cords, a fact which immediately attracted my attention when I saw the plate a few days later. Had the penny been in the subglottic space, its edge would probably have presented anteriorly. The location and position of the penny led me to inform the parents that it was not in the trachea, but was in the upper part of the esophagus at the time the skiagraphic plate was made. This diagnosis was later verified by the passage of the penny (Figs. 372 and 373).

INSTRUMENTS FOR DIRECT LARYNGOSCOPY, BRONCHOSCOPY AND ESOPHAGOSCOPY

Dr. Jackson has reduced his tubal armamentarium to four tubes and two slide speculums. It is impracticable to have one tube for adults and children, or the same tube for the esophagus and the bronchi. This means one bronchoscope for adults and one for children; one esophagoscope for adults and one for children. A good working set would be the following:

1 bronchoscope, 5 mm. by 30 cm., for children.
1 bronchoscope, 7 mm. by 40 cm., for adults.
1 esophagoscope, 10 mm. by 53 cm., for adults.
1 esophagoscope, 7 mm. by 45 cm., for children.
1 adult's slide speculum.
1 child's slide speculum.
1 aspirator for the esophagoscopes.
1 specimen forceps, long and short.
1 foreign-body forceps.
3 Coolidge sponge holders with Jackson's long collar.
1 Sajous' cotton-holding laryngeal forceps, for cocainizing the pharynx and upper laryngeal orifice.
1 double bronchoscopic battery.

An extra large adult size bronchoscope, 9 mm. by 40 cm., and an extra small infant size, 4 mm. by 30 cm., are very useful.

PART IV

THE EAR

CHAPTER XXXII

THE CLINICAL ANATOMY AND PHYSIOLOGY OF THE EAR

The organ of hearing is divisible into (*a*) the external ear, (*b*) the middle ear, and (*c*) the internal ear.

THE EXTERNAL EAR

From a clinical point of view the auricle is of interest on account of the destructive inflammatory processes which attack its cartilaginous framework and the perichondrium covering it. Perichondritis and chondritis of the auricle occurring in the insane from traumatism has been frequently observed and reported (Fig. 395). Perichondritis following the mastoid operation occasionally occurs. I have seen but one case in my practice, and it developed several weeks after the mastoid operation; the exciting cause was undoubtedly the influenza bacillus, as it followed an attack of la grippe. In performing the plastic operation upon the meatus, that is, in making the Koerner, Panse, Siebenmann, or the Ballance incisions, the cartilage of the auricle is included; hence, it is necessary to exercise great care as to surgical cleanliness, otherwise infection of the perichondrium and cartilage may occur.

The *external auditory meatus* is divisible into a cartilaginous and an osseous portion. The cartilaginous portion of the meatus (the auricular extension) is attached to the osseous or deeper portion by bands of fibrous tissue. The superior and posterior walls of the cartilaginous meatus are thinner than the anterior and inferior walls. The inferior wall extends deeper along the floor of the meatus than the other walls, and is known as the processus triangularis. The anterior wall of the cartilaginous meatus is crossed by two or three fissures, which are filled with connective tissue and a few muscle fibers. These fissures are called the fissures of Santorini, and they render the auricle more movable. They are of clinical importance, first, because they afford an outlet for the discharge of pus into the meatus in deep abscess of the parotid gland, and secondly, because they render the auricle more elastic and thus permit it to be turned on the cheek during the mastoid operation.

In the newborn the meatus is fibrous throughout its entire length, and its walls are collapsed and in apposition. Bone salts are gradually deposited and the canal assumes its patulous condition.

The sebaceous glands are limited to the cartilaginous portion of the meatus, hence furunculosis of the meatus is confined to this area. The beginner in otology is sometimes confused in making a differential diagnosis between acute suppurative mastoiditis with bulging of the post-superior wall, and furunculosis of the cartilaginous meatus. In the first instance the bulging is in the bony meatus close to the drumhead, and the auricle is not tender or sensitive upon manipulation. In the second instance, the bulging is more external in the cartilaginous meatus, and the auricle is extremely sensitive upon manipulation. The sensitiveness of the auricle in furunculosis is due to the fact that the inflammatory reaction attending the furuncle or boil has extended by continuity of tissue from the cartilage of the meatus to the cartilage of the auricle, and thereby renders the nerve fibers of the auricle exquisitely sensitive.

THE MIDDLE EAR

The membrana tympani forms the outer wall of the middle ear. It is a composite membrane of three layers: the outer one being a reflection of the skin of the meatus, the middle one being fibrous tissue, and the inner a reflection of the mucous membrane of the middle ear. The handle of the malleus is embedded within these structures, hence the sound waves impinging upon the eardrum are transmitted to the handle of the malleus, and thence to the incus and stapes, where the foot plate transmits them to the sound-perception apparatus.

The membrana tympani is of clinical importance chiefly on account of the various changes in its appearance in diseased conditions of the middle ear. These changes are, therefore, of diagnostic value. In order to appreciate fully the abnormal appearances of the eardrum, it is first necessary to know the normal characteristics. A normal drumhead is characterized by the presence of the handle of the malleus, the short process of the malleus, the triangular cone of light, the anterior and posterior folds, and a faint view of the long process of the incus seen through the semitransparent pearly gray eardrum.

When the Eustachian tube is closed, the air within the middle-ear cavity becomes rarefied by the gradual absorption of the oxygen into the blood of the surrounding tissues. As a result of the negative pressure thus brought about, the eardrum is pushed inward—that is, the eardrum is retracted. This changes the contour of the eardrum as viewed through the external auditory meatus. The cone of light is broken or altogether lost, the handle of the malleus is drawn inward and is foreshortened, the short process of the malleus projects more prominently toward the observer's eye, and the anterior and posterior folds which arise from the short process are accentuated.

In retraction due to obstruction of the Eustachian tube, the membrana

tympani is regular or uniform throughout its entire area, with the exception of the part containing the malleus. If the retraction is due to an adhesion to the inner wall of the tympanic cavity, the membrane is irregularly retracted. The membrana tympani, upon suction with Siegle's otoscope, remains fixed at the point of adhesion, and is distended in other areas, giving a blistered appearance.

PERFORATION OF THE MEMBRANA TYMPANI

The clinical significance of perforation of the membrana tympani when due to middle-ear disease is somewhat dependent upon whether it is marginal or central in location. When marginal, it usually signifies bone necrosis; and when central (away from the margin), it signifies a simple middle-ear suppuration without bone necrosis.

Its significance is still further differentiated by its exact location; that is, if it is marginal the bone necrosis is in the immediate vicinity of the marginal perforation. If, for instance, the perforation is in the margin of Shrapnell's membrane (membrana flaccida), immediately above the short process of the malleus, the tegmen antri is necrotic; if it is in the post-superior margin of the eardrum (the part nearest to the antrum), the mastoid antrum is necrosed.

The point to be borne in mind is that the perforation is secondary to the bone necrosis, the necrotic process extending from the ear cavities to the eardrum. Its clinical significance is, therefore, an index to a preëxisting morbid process in the tympanic cavities, the focal point of which is in the neighborhood of the perforation. Leutert, Zaufal, the author, and others have called attention to the significance of the foregoing facts.

The further elaboration of the clinical significance of perforations of the eardrum is given in Fig. 417.

THE EUSTACHIAN TUBE

The second and most common avenue of approach to the middle-ear cavity is through the Eustachian tube. It is through this channel that nearly all middle-ear diseases invade the middle-ear cavity. The tube is about 36 mm. long, the pharyngeal opening being about 15 mm. lower than the tympanic opening. The tympanic opening corresponds to the anterosuperior quadrant of the eardrum, hence it is not in the most dependent portion of the cavity. This does not interfere with drainage under normal conditions, as the ciliæ of the epithelium of the tympanic cavity sweep the secretions to the opening of the tube and through it to its pharyngeal opening. If, however, the ciliæ are impaired in their functional activity by an inflammatory or other morbid process, the elevated position of the tympanic orifice of the tube materially interferes with the drainage. Under these conditions the secretions are

retained, decomposition follows, and further irritation of the mucous membrane results.

The tympanic end of the tube has an osseous framework, and is about 12 mm. long. The pharyngeal end of the tube has a cartilaginous and membranous framework, and is about 24 mm. long. The tube is trumpet-shaped at both extremities, and is narrowest at the junction of the osseous and cartilaginous portions. This is known as the isthmus. The framework is lined with mucous membrane which is covered with ciliated epithelium, which carries the secretions toward the pharyngeal orifice.

Under ordinary conditions, the membranous walls of the tube are in a state of collapse, and only open when certain palatal muscles are contracted. Yawning and swallowing cause these muscles to contract, and air is thus admitted into the tympanic cavity.

The muscles regulating the patency of the pharyngeal orifice of the tube are the tensor veli palati and the levator palati; they also elevate the soft palate and assist in approximating it against the posterior wall of the pharynx in the act of swallowing. As the superior ends of the muscles are attached to the cartilaginous lip and to the membranous portion of the tube, and the inferior end to the soft palate, it is obvious that the contraction of the muscles will produce a twofold result—namely, the pharyngeal orifice of the tube is opened and the soft palate is elevated.

When, for any reason, the act of swallowing does not open the tube sufficiently to admit air into the tympanic cavity, the oxygen is absorbed from the contained air by the blood in the surrounding tissues, and a partial vacuum, or negative pressure, results. The blood in the surrounding tissues is drawn to the parts by the negative pressure, and congestion results. The retained secretions undergo decomposition and irritate the lining mucous membrane. The hyperemia induces overnutrition. As a result of the combined irritation and increased nutrition the mucous membrane becomes thickened, either by hypertrophy or hyperplasia. The secretions are not only retained in excesive quantity, but are changed in character. This condition is known as middle-ear and tubal catarrh.

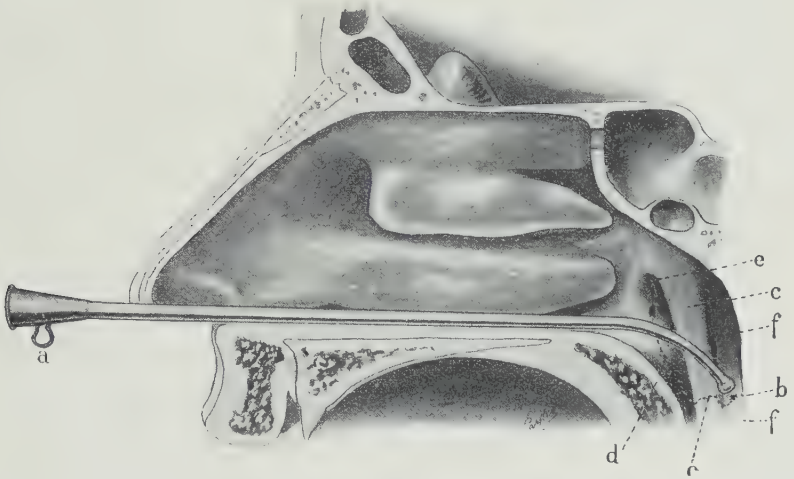
Anything that obstructs the flow of secretions of the Eustachian tube predisposes the mucous membrane of the tube and middle ear to infection and inflammation. The two great underlying principles relating to the etiology of inflammation of mucous membrane lined cavities are: (*a*) The exciting cause of inflammation is almost always a pathogenic microörganism. The microörganism is powerless to grow upon healthy tissue, hence the second great underlying principle relates to the conditions which favor their growth. (*b*) The predisposing cause is usually an obstructive lesion interfering with the drainage and ventilation of the cavity, thereby lowering the resistance of the tissues. The pathogenic microörganisms then flourish, and with their toxins excite the reaction of inflammation.

The action of the tensor and levator veli palati muscles is so intimately associated with that of the muscles of the palate and pharynx, that it is somewhat difficult to estimate the influence of the other muscles on the

patency of the tubes. The pharyngopalatinus (posterior pillar of the fauces) has its upper attachment in the soft palate, and it contracts during deglutition, and thus indirectly exerts a tensive action upon the tubal muscles. In inflammatory processes involving the tonsils and the faucial pillars, the swollen condition of the palatopharyngeus muscle indirectly interferes with the action of the tubal muscles. In this way, disease of the tonsil causes tubal and middle-ear disease; that is, drainage and ventilation are interfered with. The microörganisms causing the tonsillar disease find a lowered resistance of the tubal membrane, grow there, and cause catarrhal or suppurative inflammation.

The anterior wall of the pharyngeal end of the tube is membranous, while the upper and posterior walls are cartilaginous. The tensor and levator veli palati muscles are attached to the membranous portion of the tube, hence when they contract the tube is opened to its isthmus.

FIG. 374



Showing a method of catheterization: *a*, the ring indicating the direction of the tip of the catheter; *b*, the posterior wall of the pharynx; *c*, *c*, the ridge forming the posterior lip of the mouth of the Eustachian tube; *f*, *f*, Rosenmüller's fossa; *b*, *d*, *e*, the route traversed by the tip of the catheter to enter the mouth of the Eustachian tube.

Much has been written concerning the normal patency of the Eustachian tube, and the preponderance of the evidence is in favor of the view that it is closed except during the act of deglutition. Politzer's experiment, consisting of a vibrating tuning fork held in front of the nose shows that it is but faintly heard except during deglutition, thereby proving that the tube is closed under ordinary conditions and is open during deglutition. This permits of the interchange of air between the pharynx and the middle ear, and maintains an equilibrium of pressure on the inner and outer surfaces of the membrana tympani.

The pharyngeal end of the tubal cartilage (posterior and superior walls) forms a projecting lip or tubal prominence on the lateral wall of

the epipharynx. Just behind this is a groove known as Rosenmüller's fossa. The fossa and tubal prominence are the landmarks used in the introduction of the Eustachian catheter. The tip of the catheter is first lodged in the fossa of Rosenmüller, then drawn forward, gliding downward and inward over the prominence, and thence upward and outward into the tubal orifice (Fig. 374).

To inflate the tube and middle ear, the compressed air should be applied at the beginning of the act of deglutition, as the tubal muscles are then contracted and the tube open. The Eustachian tube of an infant is shorter, straighter, and more easily inflated than that of an adult. In an adult the tube is sharply bent at the isthmus, whereas in an infant the tube is nearly straight. A lower degree of air pressure should, therefore, be used for infants than for adults. Earache in infants and young children is often quickly relieved by inflation, as it is due to tubal congestion and obstruction, or to a plug of tenacious mucus in the lumen of the tube.

THE TYMPANIC CAVITY; TYMPANUM; CAVUM TYMPANI

The tympanic cavity is the space between the tympanic orifice of the Eustachian tube and the mastoid antrum. Its lining mucous membrane is continuous with that of the Eustachian tube, and extends to the antrum and mastoid cells. It is covered with ciliated epithelium, the wave-like motion of which carries the secretion to the Eustachian tube.

The upper wall (tegmen tympani) of the tympanic cavity forms a portion of the floor of the middle fossa of the cranial cavity; the outer wall is composed of the eardrum, and in its upper portion (outer wall of the attic) of bone. The wedge of bone forming the outer wall of the attic should be removed in the radical mastoid operation to fully expose this space to inspection and treatment during and after the operation. The inner wall of the tympanic cavity is contiguous to the outer wall of the cochlea and vestibule; the posterior wall separates the tympanic cavity from the antrum and mastoid cells; the anterior wall is very thin and covers the internal carotid artery; and the lower wall separates the tympanic cavity from the jugular bulb. The facial nerve runs across the upper and posterior wall and is usually enclosed in a bony covering, though numerous instances are on record in which the bony covering was absent.

The foregoing description of the relations of the walls of the tympanic cavity to the contiguous vital organs is of great clinical significance in the middle ear and mastoid infections and inflammations.

Contents of the Tympanic Cavity.—The tympanic cavity contains the chain of ossicles, the tympanic muscles, and the chorda tympani nerve. The handle of the malleus is attached to the membrana tympani, and the foot plate of the stapes is attached to the membrane of the oval window. The incus is suspended between the malleus and stapes, and completes the anatomical connection between the membrana

tympani and the labyrinth. The chain of ossicles transmits the sound waves from the membrana tympani to the labyrinth, though there is little doubt that some waves are transmitted through the air in the tympanum to the round or oval window without the intervention of the ossicles. I recall one patient on whom I did a radical mastoid operation, removing the malleus and incus, who heard whispered speech at ten feet, showing that good hearing is possible though all the ossicles were removed except the stapes.

The Chain of Ossicles and the Membrane of the Oval Window.—It is shown by the case just cited that all the receiving apparatus may be removed except the contents of the oval window without greatly impairing the hearing, though this is exceptional. Orientation of hearing is greatly diminished, as is also the faculty of keying the perception apparatus to catch sounds accurately. The tensor tympani and the stapedius muscles are rendered ineffective by the removal of the malleus and incus, hence the ear has lost its focussing apparatus. The membrana tympani receives a larger number of sound waves than the foot plate of the stapes, hence the hearing is more acute with the eardrum and the ossicles intact than it is without them.

A Physiological Law.—It may be laid down as a physiological law that *anything that interferes with the normal tension existing between the membrana tympani, ossicles, and the contents of the oval window will cause tinnitus and deafness.* Hence, pathological changes in the eardrum, thickening or other change in the mucous membrane which covers the ossicles, ankylosis of the ossicles, especially of the foot plate of the stapes, as in spongifying of the bony capsule of the labyrinth, etc., result in tinnitus and deafness. Catarrhal inflammation of the mucous membrane of the middle ear and Eustachian tube induces a negative pressure in the tympanic cavity, and disturbs the normal tension between the eardrum and the oval window; the mucous membrane of the walls of the tympanic cavity and ossicles is thickened, and tinnitus and deafness follow. The inflation of the tympanic cavity in tubal and middle-ear catarrh restores (in a degree) the normal tension and decreases the congestion of the mucous membrane, and thereby lessens the tinnitus and deafness.

The heads of the malleus and incus and their ligamentous attachments to the walls of the tympanic cavity divide the cavity into two compartments—namely, the atrium, or middle ear proper, and the attic. When there is a suppurative process in the attic or the antrum and mastoid cells for a considerable time, adhesive bands form and still further increase the barrier between the atrium and the attic. The drainage of the secretions is blocked, and gives rise to retention and decomposition of the secretions and to pressure symptoms, as pain and tenderness. Necrosis is also augmented by the increased pressure from the retained secretions. Suppuration in the attic, and in the antrum and mastoid cells in old chronic cases, is, therefore, a more serious condition than suppuration with its focal centre in the atrium.

The *chorda tympani* nerve passes through the upper portion of the

atrium between the handle of the malleus and the long process of the incus, and is usually severed or destroyed in the radical mastoid operation. As a consequence, the sense of taste at the base of the tongue and the neighboring parts of the fauces is impaired; indeed, it is perhaps best to destroy the nerve, as the irritation during the application of post-operative dressings would otherwise excite a disagreeable sense of taste.

Walls of the Tympanum.—The superior wall, the tegmen tympani, is a thin plate of bone forming a portion of the middle fossa of the skull, and it is frequently the seat of necrosis in suppurative inflammation of the middle ear. The necrotic process often extends through it, and thus exposes the dura to infective bacteria which may be present. Ordinarily a wall of granulation tissue is formed in Nature's effort toward repair and protection. Such a perforation may, therefore, exist for years without involving the cranial contents. On the other hand, if the secretion is blocked by the ossicles, their ligaments, and the adhesive bands at the floor of the attic, the infective bacteria may be forced through the granulation tissue into the cranial cavity and excite meningitis or brain abscess.

One of the strongest arguments against curettage of the attic through the external auditory meatus is, that the granulation tissue may be removed and the dura exposed to the pathogenic bacteria. The same objection does not hold to its removal during the radical mastoid operation, as perfect drainage is thereby established.

The inferior wall or floor of the tympanic cavity is of clinical interest, on account of its proximity to the jugular bulb. It is only in exceptional cases, however, that the floor is thin, hence the jugular bulb is ordinarily in no danger in the curettage of the floor. Occasionally the floor is so thin that in curetting granulations from it there is danger of injuring the jugular bulb and causing serious or even fatal hemorrhage. When the jugular bulb is thrombosed, necrosis of the floor of the tympanic cavity may occur, and granulations spring from this point. Granulations of the floor of the tympanum in cases of lateral sinus thrombosis are significant of the involvement of the jugular bulb.

The outer wall of the tympanum is chiefly composed of the membrana tympani, though at its upper and lower portions it is composed of bone. The bony wall at its upper portion forms the outer wall of the attic, or the recessus epitympanicus (Fig. 375). The handle of the malleus is embedded in the membrana tympani, as is also the short process, located at the upper extremity of the handle.

The inner wall of the tympanum is of interest because it also forms the outer wall of the labyrinth, and because of the presence of important structures concerned in the function of sound conduction (Fig. 377). The most important of the structures concerned in sound conduction are the oval window (fenestra vestibuli), the stapedius muscle, the tensor tympani muscle, and the round window (fenestra cochlea). The other important structures are the promontorium, a projection due to the beginning of the basil turn of the cochlea; the prominentia canalis facialis, which forms the upper and posterior border of the fossula

fenestræ cochleæ, and the prominentia canalis semicircularis lateralis. The prominences of the facial nerve canal and of the lateral semicircular canal form the median boundary of the attic (recessus epitympanicus), and they lie in close relation to the deep portion of the postsuperior wall of the external auditory meatus. The removal of this wall in the radical mastoid operation is likely to result in injury to these two structures. The Stacke protector is sometimes used to protect these structures by passing it from the middle ear upward and backward into the aditus ad antrum.

The facial nerve is usually covered by bony tissue, though in exceptional cases it is not. In necrotic processes it is frequently exposed, hence extreme caution is necessary in removing the postsuperior wall of the meatus, lest the nerve be injured. The nerve comes sharply outward from the cranium and then turns downward, forming a rather sharp knee, without coming near the mastoid surface. Hence, the outer portion of the posterior wall of the meatus may be removed without danger of injuring the facial nerve. T. Passmore Berens reported a case in which the facial nerve came near the surface, and in which it would have been injured if the posterior wall of the meatus had been removed as completely as usual. The bone of the postsuperior wall of the meatus is often spoken of as a "wedge of bone," from the fact that it is triangular in shape. The point of the wedge is at its deepest portion, while the pole is the external portion. The point of the wedge forms the outer wall of the aditus ad antrum, the constriction which marks the boundary between the attic and the antrum.

The malleus and incus are also removed in the radical mastoid operation, and the obstruction to the drainage of the mastoid cells and the antrum is thus completely removed. The chief objection to the ossiculectomy alone for the cure of chronic suppurative ear disease is that neither is free drainage thereby established, nor is all the morbid material removed; that is, the necrosis and granulations are usually present in the antrum and cells as well as in the attic, hence the removal of the malleus and incus does not give relief except in the attic. If the disease is limited, or focalized in the attic, ossiculectomy may be all that is necessary to do.

The Antrum.—The antrum is embryologically a part of the middle ear, while the mastoid cells are not. It communicates with the attic through the aditus ad antrum. The mastoid cells drain into it. The ciliated epithelium lining the cells, antrum, tympanum, and the Eustachian tube propels the secretions successively through these parts to the pharyngeal orifice of the tube. In severe acute inflammation, and in prolonged chronic inflammation, the epithelium is denuded in certain areas of its ciliæ, and the drainage of the secretions is interfered with. The superficial destruction of tissue thus started may extend to the deeper tissues, as the epithelium, mucous membrane, periosteum, and the bone. Necrosis may be thus established. When such extensive destruction has become established there is little probability of a cure except by the radical, or the meatomastoid operation.

The Mastoid and Temporal Bone Cells.—A knowledge of the possible distribution of the mastoid and temporal bone cells is sometimes a matter of extreme importance in the successful treatment of mastoiditis. In many chronic cases it is absolutely necessary for the surgeon to remove all morbid tissue, and to establish free drainage of the remotest air spaces in the temporal bone. The pneumatic cells are not always confined to the mastoid process, but may be in the posterior root of the zygoma, the squamous plate of the temporal, in front of the external auditory meatus and in the posterior wall of the pyramid of the petrous portion of the temporal bone. When in the latter position they are not easily reached, though as Jansen has shown, they may be exenterated. I have seen cases in which pus-discharging cells were in front of the meatus with a canal of communication leading to the antrum. Had they not been opened and exenterated in the course of the radical operation, the operation would have been a failure. Hence, it is necessary in all chronic cases to make careful search for pneumatic cells in other regions than the mastoid process. In one of Dr. Wale's bony specimens, the mastoid cells communicated with the sphenoid sinus.

The Arteries of the Middle Ear.—The middle ear receives its chief blood supply from branches of the internal carotid artery. The branches pass backward through the canaliculus carototympanici to the mucous membrane of the middle portion of the tympanic cavity. The middle meningeal artery sends a branch to the upper portion of the middle ear, while the A. stylomastoidea sends a branch to the postinferior portion and to the mastoid cells. As all these branches are quite small, they have no special clinical significance.

PHYSIOLOGY OF THE EAR

I. **Membrana Tympani.**—The eardrum is stretched across the inner end of the external meatus, and is elastic enough to undergo considerable movement when the air in the meatus is alternately condensed and rarefied with Siegle's otoscope. The membrane is attached to a groove in the annulus, the sulcus tympanicus, by an extension of the periosteum, of which the middle or fibrous layer is composed. The annulus tympanicus does not extend completely around the meatal opening, but is absent at the upper portion, the Rivinian segment. The part of the membrane attached to the annulus is known as the pars tensa or the membrana tensa.

The part attached to the Rivinian segment is not stretched, but is loosely drawn, and is known as Shrapnell's membrane, the pars flaccida or the membrana flaccida. This portion of the membrane forms the outer wall of Prussak's space, while the pars tensa forms the lower portion of the outer wall of the tympanic or middle-ear cavity (Fig. 370).

The membrana tympanum is not placed perpendicularly across the opening of the meatus, but forms an angle of about 140 degrees with the postsuperior wall, and one of 45 degrees with the antero-inferior wall.

This is of clinical importance in the removal of foreign bodies from the meatus.

The function of the membrana tympani is to receive and convey sound waves to the chain of ossicles, and thence to the labyrinth. That it is not absolutely essential to fair hearing is shown by the fact that good hearing is often present when the membrane is perforated or entirely absent. The eardrum also protects the tympanic mucous membrane from the deleterious effects of the air and from the entrance of morbific germs and foreign bodies.

When the normal tension of the drumhead is disturbed, there is an impairment of hearing; hence, any morbid condition of the Eustachian tube which interferes with the ventilation of the tympanic cavity, or any inflammatory disease of the mucous membrane which interferes with the mobility of the ossicular chain, or any morbid condition of the drumhead which interferes with its elasticity or motility, will cause more or less deafness.

II. **Eustachian Tube.**—The function of the Eustachian tube is twofold, namely: (*a*) To ventilate, (*b*) to drain the tympanic and mastoid cavities. When these spaces are healthy, the Eustachian tube is adequate for the purpose. When, however, the spaces are inflamed, and the secretions are greatly increased in quantity, it is not large enough to accommodate the passage of the secretions into the epipharynx. When its capacity is thus overtaxed, the retention of the secretions causes pressure necrosis in the direction of least resistance, namely, the membrana tympani. Perforations thus arise in the course of infective inflammations of the tympanic cavity, the antrum, and mastoid cells. The Eustachian tube is generally large enough to carry off the secretions from the tympanic cavity, even when in a diseased state; but when in addition the antrum and mastoid cells are involved, it is not capable of disposing of the secretions, retention occurs, and the pressure symptoms (pain, tenderness, and swelling) of mastoid inflammation ensue. If the excess of secretions from the antrum and the mastoid cells are diverted from the tympanic cavity, the morbid process tends to subside because the tube is large enough to drain the secretions from the tympanic cavity. In other words, the retention of the secretions in any cavity tends to foster inflammatory processes in the mucous membrane, which may, in time, extend to the periosteum and the bone to which it is attached. (See Diseases of the Nasal Accessory Sinuses, the Clinical Anatomy of the Tonsils, and Meatomastoid Operation.)

Tympanic Cavity.—The function of the tympanic cavity and its contents is to transmit sound waves to the labyrinth. It also forms a channel of communication between the Eustachian tube and the epipharynx, on the one hand, and the antrum and mastoid cells on the other. The cavity is divided into two spaces by the interlocking heads of the malleus and incus. The lower space is called the atrium, or the middle ear proper, while the upper is called the attic. The attic is still further subdivided by the heads of these bones into an inner and outer attic. The outer space is divided into an upper and a lower

space by the external ligament of the malleus (Fig. 375). The lower space is called Prussak's space, suppurative inflammation of which is difficult to cure. (See Suppuration of Prussak's Space.)

The inner wall of the tympanic cavity presents two anatomical features of physiological and clinical interest, namely, the oval and round windows. The oval window, the fenestra vestibuli, receives the foot plate of the stapes, which is surrounded by the annular ligament and communicates with the vestibule of the labyrinth. The round window opens into the cochlea, and the membrane closing it forms an elastic valve to relieve the shock to the cochlea in the presence of excessive sound waves.

Fig. 375

Coronal section through the tympanum. *a*, extremity of the upper; *b*, extremity of the lower bony wall of the meatus; *d*, tegmen tympani; *e e*, attic, external portion, internal portion; *f*, malleus and superior ligamentum mallei; 2, incus; *h*, stapes within the fenestra vestibuli; *i*, promontory; *k*, Prussak's space; *m*, hypotympanic recess (cellar); *l*, scar in the lower half of the drumhead in apposition with the promontory; 2, incudostapedial junction. (After Brühl-Politzer.)

Intrinsic Muscles of the Ear.—The tensor tympani muscle pulls the *handle of the malleus inward*, thus increasing the tension of the drumhead. This movement of the malleus is communicated to the long process of the incus, which in turn acts upon the stapes and compresses it into the oval window. Prolonged retraction of the membrana tympani is attended with a shortening of the tendon of the muscle, a condition which materially interferes with the cure of the deafness resulting from these conditions. The stapedius muscle acts in antagonism to the tensor tympani, and counterbalances the compression of the foot plate of the stapes in the oval window. The membrana tympani, the circular ligament of the oval window, and the interposed chain of ossicles are thus poised to receive the sound waves and transmit them to the cochlea, where the impression is received by the delicately attuned organ of Corti, which in turn transmits the impression through the auditory nerve to the auditory centre of the brain, where it is perceived as sound.

It is apparent from the foregoing physiological data that it is of great therapeutic value to maintain free drainage and ventilation of the middle ear and its accessory cavities, and to prevent the morbid changes incident to the inflammatory processes of the middle ear.

Physiology of the Sound-perceiving Apparatus.—The sound-perceiving apparatus is composed of the terminal nerve filaments of the labyrinth, the acoustic (auditory) nerve, and the auditory centre in the brain.

Auditory Nerve.—The auditory nerve arises between the facial and glossopharyngeal nerves in the medulla oblongata, and passes into the internal auditory canal, in the fundus of which it divides into two branches; the vestibular branch (nerve) enters the vestibule, where it sends twigs to the utricle and the superior ampullæ of the semicircular canals; the cochlear branch (nerve) passes into the cochlea and gives off twigs to the saccule and to the ampulla of the superior semicircular canal.

The distribution of the auditory nerve in the cochlea forms a spiral ganglionic ribbon, the ganglionic cells being connected by medullated nerve fibers, the whole being supported on the membranous cochlea, which is attached to the osseous cochlea by fibrous bands. The membranous labyrinth is filled with a fluid called endolymph, and is surrounded by a fluid called the perilymph. The cochlear distribution of the auditory nerve is called the organ of Corti.

Function of the Vestibular Apparatus.—Within the vestibule (saccule and utricle) the otoliths, acting upon the delicate hair-like prolongations within the ampulla, preside over the sense of the position of the head (body) in space. The angle of the impact of the otoliths upon the hair-like processes (the relative bending) creates a sensation which, being interpreted by the brain centres, gives conscious knowledge of the relative position of the head (body) to the line of gravity and consequently to the plane of the earth. In other words, they aid in the maintenance of equilibrium. (See Functional Tests of the Vestibular Apparatus.)

Function of the Semicircular Canals.—These canals are the organs of coördinated movements, or statical sense; hence, they are also a part of the apparatus presiding over the sense of equilibrium. (See Functional Tests.)

Function of the Cochlea.—Corti's cells constitute the true terminal acoustic (auditory) nerve apparatus. They are about 2000 in number, and are ciliated. The function of the cochlear apparatus is to perceive and differentiate sound waves, and convey them to the auditory nerve trunk, thence to the acoustic centres of the brain, where they are perceived as sound.

Shambaugh controverts the theory of Helmholtz that the basilar membrane is the resonator of the internal ear. According to Helmholtz, the fibers of this membrane vibrate in sympathy with the sound waves as they react upon the labyrinth, and thus stimulate the hair cells of the organ of Corti. Shambaugh's conclusions are ingenious, and are as follows (Plate XVI):

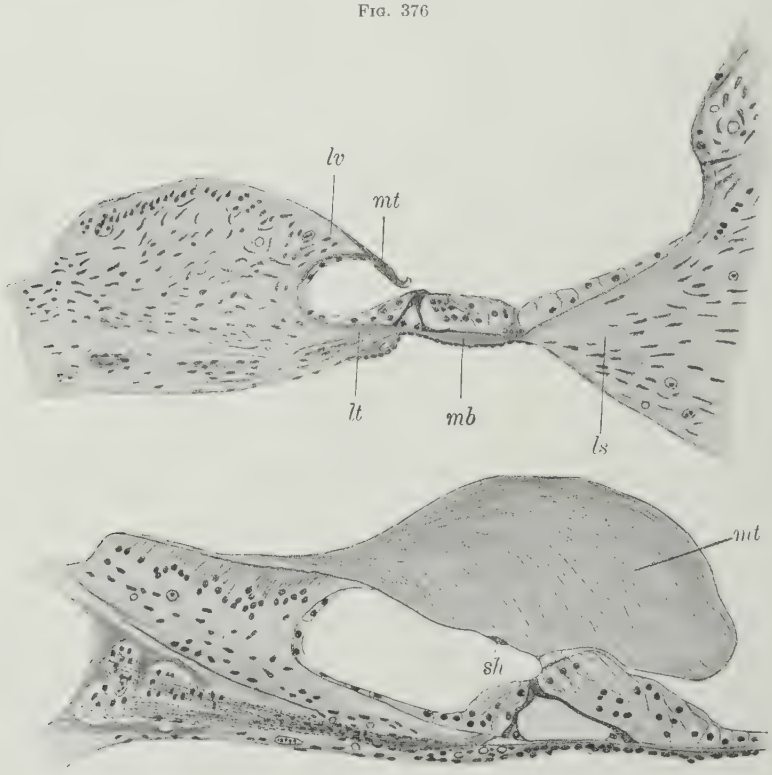
1. "The hair cells of the organ of Corti are the real end organs wherein the physical impulses of sound waves are transformed into the nerve impulses, which result in tone perception.

2. "The perception for the various tones takes place in different parts of the cochlea, those of higher pitch being taken up by the hair cells located near the beginning of the basal coil, those of lower pitch by the cells near the apex of the cochlea.

3. "The stimulation of the hair cells is effected only through the medium of their projecting hair.

4. "The hypothesis that each hair cell acts as its own agent in selecting its stimulus from the impulses passing the endolymph is shown to be untenable for a number of reasons, chiefly, however, because the relation existing normally between the hair cells and membrana tectoria will not permit of these impulses in direct contact with the hair cells. I have shown conclusively that the hairs of the hair cells project normally into the under surface of the membrana tectoria.

Fig. 376



lv, labium vestibularis; *mt*, membrana tectoria; *lt*, labium tympanæ; *mb*, membrana basilaris; *ls*, ligamentum spirale; *sh*, streifen of Hensen. (Shambaugh.

5. "The stimulation of the hair cells is accomplished only through an interaction between the hairs of the hair cells and the membrana tectoria.

6. "The hypothesis of Helmholtz that this stimulation is brought about through the vibration of the fibers of the membrana basilaris is untenable, especially for the following reasons: In tracing the membrana basilaris toward the beginning of the basal coil in the vestibule this structure is found at a considerable distance from the lower end of the coil, and where a perfectly formed organ of Corti it still present, to become so stiff and rigid as to render it incapable of vibrating. Even a complete absence

of a basilar membrane in this locality is sometimes noted. The logical conclusion is that since the stimulation of the hair cells in this locality is accomplished without the intervention of a vibrating membrana basilaris, therefore the stimulation of the hair cells throughout the cochlea is not dependent on the vibration of this membrane.

7. "The logical conclusion is that the stimulation of the hair cells is accomplished through vibrations of the membrana tectoria transmitted to it by impulses passing through the endolymph.

8. "The membrana tectoria is shown to be so constituted anatomically as to be capable of responding to the most delicate impulses passing through the endolymph. Furthermore, the great variation in size of this membrane from one end of the cochlea to the other, together with its lamellar structure, suggests the probable physical basis which renders it capable of acting the part of resonator by responding in one part to impulses of a certain pitch, and in another part to impulses of another pitch (Fig. 376).

9. "Finally, the pathological phenomena of 'tone islands,' 'diplakousis binauralis of disharmonica,' and of 'tinnitus aurium,' are all plausibly accounted for in this conception of the physiology of tone perception.

10. "To restate briefly the process by which the phenomenon of tone perception is accomplished: The sound waves conducted from the air impinge upon the membrana tympani, producing vibrations in it. These vibrations conducted along the chain of ossicles transmit impulses to the intralabyrinthine fluid through the medium of the foot plate of the stapes. The impulses originating in the fluid in the vestibule pass directly into the scala vestibuli and through the membrane of Reissner to the endolymph, where sympathetic vibrations are imparted to the several parts of the membrana tectoria, depending on the pitch of the tone. The vibrations in turn stimulate the hairs of the hair cells which normally project into its under surface. The nerve impulses originating from all the hair cells thus stimulated by a particular tone come together in the brain centre in the cortex when the tone picture forms the final step in the process of tone perception."

CHAPTER XXXIII

THE FUNCTIONAL TESTS OF HEARING[1]

Physiological Facts.—(*a*) **Range of Hearing.**—The normal range of hearing, in man, for musical tones is from 16 to about 22,000 double vibrations per second. After the fiftieth year the upper limit of hearing is somewhat lowered.

(*b*) **Paths through Which the Sound Waves Reach the Labyrinth.**—1. Sound waves reach the labyrinth chiefly through the tympanic membrane, the ossicles and the oval window into which the foot plate of the stapes is inserted. The foot plate is attached to the oval window by a fibrous membrane which allows it to vibrate therein. Politzer demonstrated that the malleus performed the greatest excursions, the incus less, and the stapes least of all. The relation of the area multiplied by the excursion of the foot plate of the stapes to the similar factors of the membrana tympani was found by Bezold to be as 1 to 778. Example (the product of the area of foot plate of stapes x its movement distance) is to (the product of the area of membrana tympani x its movement distance) as 1 to 778. This is about the relation of the specific gravity of air to that of water (1 to 774). The mass vibrations of the lower tones in their transmission through the drum membrane and the chain of ossicles to the perilymph are transposed to this degree into molecular vibrations. Hence the loss of hearing for low tones in disease of the conduction apparatus.

2. Sound waves also reach the labyrinth through the fenestra cochleæ (round window), therefore the function of the ear is not altogether destroyed when the foot plate is fixed, as in spongifying of the bony capsule of the labyrinth.

Sound waves are also carried to the labyrinth to a considerable extent through the bones of the skull (Fig. 377). This explains why some deaf persons hear tolerably well over the telephone. Weber's well-known experiment demonstrates that when a tuning fork is placed upon the skull and the external meatus is artificially closed with the finger, the vibrating fork is heard much better on that side. In other words bone conduction is thus increased. In the normal ear, hearing by bone conduction for tuning forks of medium pitch is about one-third to one-half of that by air conduction. The relative duration of hearing by bone and air conduction varies greatly with different forks. It also varies with the point of contact made with the fork. Bone conduction is best over the mastoid antrum. Politzer, Bezold and Andrews have called attention to the varying results obtained by different forks of the same number of vibrations. Each fork should, therefore, be carefully and

[1] This section has been entirely rewritten by Dr. Alfred Lewy.

repeatedly tested upon normal persons to establish its normal register. By *normal register* is meant the length of time the fork is heard by normal ears by bone conduction when placed over the mastoid antrum, and the time it is heard by air conduction when held as near as possible to the auditory meatus (Fig. 382).

FIG. 377

Air and bone conduction (schematic): 1, cranium; 2, cerebrum; 3, auditory nerve going to temporal lobe; 4, labyrinth; 5, tympanum and ossicles; 6, auditory meatus; 7, pinnæ; *a*, tuning fork placed on the vertex; *a b*, osteal bone conduction; *a c*, craniotympanal bone conduction; *d*, tuning fork held in front of the ear; *d c*, air conduction. (After Brühl-Politzer.)

5. The Bezold-Edelmann set of forks and whistles has become standard. It is constructed upon scientific principles and should be used by all otologists. It covers the range of hearing of the human ear. The forks are weighted and free from overtones. With them deaf mutes may be tested for "islands of hearing," and when found the islands or areas of the organ of Corti which are functionating may

be utilized to teach speech if within the range of tones used in articulate speech. They are indispensable for scientific work. No other set of forks and whistles meets all the demands. One may usually, though not always do diagnostic work with three well-selected forks, for instance the Reiner set as used by Neumann of Vienna. This set consists of one C (64 d.v.) for estimating the low tones, one d♯ (153.8 d.v.) for the relative bone and air conduction, and c^4 (2048 d.v.) for estimating the high tones. While one may not determine the low or the high limits with these, a loss of hearing for low tones or for high tones may be determined by the shortening of the time the C or c^4 respectively are heard as compared with the normal.

(*c*) **Tension.**—The tensor tympani draws inward the hammer handle and the drum membrane and tilts the hammer head and incus so that the foot plate of the stapes is pushed into the oval window, thus increasing the pressure on the perilymph. The stapedius by its contraction tilts the anterior and broader end of the foot plate outward, the narrower, posterior portion of the membrane of the oval window acting as the fulcrum, the power being applied at the head of the stapes and incudostapedial joint. Thus the stapedius is the antagonist of the tensor tympani. Their exact function in the process of hearing has not yet been satisfactorily demonstrated.

(*d*) **Perception.**—The normal ears perceive sound in its actual pitch. Both ears perceive it exactly alike in pitch, timbre, and intensity. In certain pathological conditions one or both ears may be "out of tune," a condition known as diplacusis, when both ears are not in consonance.

Principles Underlying the Tests of Hearing.—1. The normal range of hearing is from 16 to 22,000 double vibrations per second, that is, from C_2 (16 d.v.) to about f^7 (22,097 d.v.).

2. When the conduction apparatus is diseased or obstructed the hearing is impaired or lost principally for the lower tones of the scale.

3. When the perception apparatus is diseased the hearing for all tones is impaired, and is entirely lost (as a rule) for the high tones.

4. The normal ear hears the tuning fork about two or three times as long by air as by bone conduction. The ratio varies with different forks.

5. When the conduction apparatus is diseased or obstructed, bone conduction is increased and air conduction is diminished; bone conduction may be so much increased that the fork is heard longer by bone than by air conduction (Negative Rinne).

6. When the perception apparatus is diseased bone conduction is diminished. Hearing for the tuning fork by air conduction is diminished to a less degree, so that it appears relatively exaggerated.

The Functional Tests of the Auditory Apparatus.—In considering the physiological tests of the functional ability of the cochlea it is to be remembered that no one of the tests described is alone sufficient for an accurate diagnosis, as a rule. Each test has its value as a part of the examination and in corroboration of the other tests, and the diagnosis is made from the entire picture. While it is not always

necessary to go through all the tests as described in the schema below, it is the opinion of the writer that a diagnosis made without at least the more fundamental ones is merely a guess. As will be seen by a careful study of the preceding paragraph the fundamental principle is the bone conduction. Markedly diminished bone conduction nearly always means disturbance or disease of the perception apparatus. Markedly prolonged bone conduction means disturbance or disease of the conducting apparatus. The other tests and the physical examination, both local and general, are necessary to give more detailed information upon which intelligent treatment and prognosis may be based. The otologist should make constant use of the tests, in order that he may become skilled in their application and in his deductions therefrom. It is necessary, therefore, to make a routine practice of applying them to all or nearly all cases coming under observation. This is the practice of the author in both private and clinical work and he feels well rewarded for his trouble. The portion of the Politzer clinic examination schema referring to the functional tests is as follows:

Right	Left
Whisper	
Conversation	
Acoumeter	
Weber test	
Schwabach test	
Rinne test	
Low limit	
High limit	
Whisper, conversation or acumeter after inflation.	

The Voice Test.—The practical test of hearing is the ability to hear conversation, but as the spoken voice is usually too loud for the distance obtainable in an ordinary office, and as there is a great difference in the carrying quality of different consonants and vowels, the whispered voice is more applicable, provided the deafness is not of such degree that the whisper is heard with great difficulty or not at all. In using the whisper it should be given with only the residual air, so as to obtain the greatest degree of uniformity, and the surgeon should train his voice to this end. In a perfectly quiet hall the whisper may be heard by a normal ear at about 40 meters. In 1871 Oscar Wolf published his conclusions as to the voice as a means of testing the organ of hearing. He found the letter R the lowest in the scale, while the highest number of vibrations were produced by the letter S. In the same manner some words are high pitched while others are low. Examples of high-pitched words are: six, seize, tease, message, shady; low-pitched words: horror, rural, moon, rude; medium-pitched words: table, Mary, baby.

To a certain degree the voice test may indicate the form of deafness, as, for instance, in conduction deafness the high-pitched words are heard much better than the low ones, as a rule. The reverse is not true to any degree in nerve deafness; however, in this condition Wolf

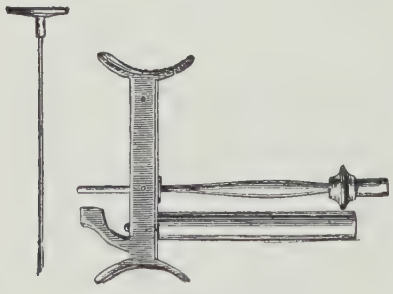
states that F sounds are not heard. It should be remembered that in pathological conditions of the hearing that neither the whisper nor any other test is a correct indication of the hearing for conversational voice, and that some voices are heard better than others. It is a common experience for people with a pure conduction deafness to find that they hear high-pitched feminine and children's voices better than they do a low masculine one.

Technic.—(*a*) Place the patient at one end of a quiet room with the ear to be tested toward the examiner's end of the room. The patient should not see the lips of the physician during the test. Some deaf people become very expert in lip reading.

(*b*) Have the patient moisten the tip of his index finger and insert it firmly into the meatus of the ear which is not being tested. The physician should himself see that this is properly done.

(*c*) The physician begins the test from without the range of the patient's hearing, approaching quietly until patient repeats correctly what is spoken or whispered to him, and the distance so found is entered on the record. If the room is not long enough the physician should turn his back to the patient. If the distance is still too short the patient should turn his open ear to the opposite wall. Each of these maneuvers is supposed to indicate an increased distance of about one-third. The spoken or the whispered voice is used according to the degree of deafness, and the record should state which style of speech is used. Repeat test with the other ear, using different words or numbers.

Fig. 378



Politzer's acoumeter.

(*d*) Inflate the ears.

(*e*) Repeat the tests and record the difference, if any, following inflation.

The Politzer Acoumeter.—(Fig. 378). This instrument was designed to give an accurate mechanical standard of measurement for the hearing distance. All the instruments are supposed to be of the same pitch and timbre, and the hearing distance for them in a quiet room should be about 40 feet. Politzer and Lucae claim that this instrument more nearly corresponds with the voice tests than either the watch or the distance test with the tuning fork.

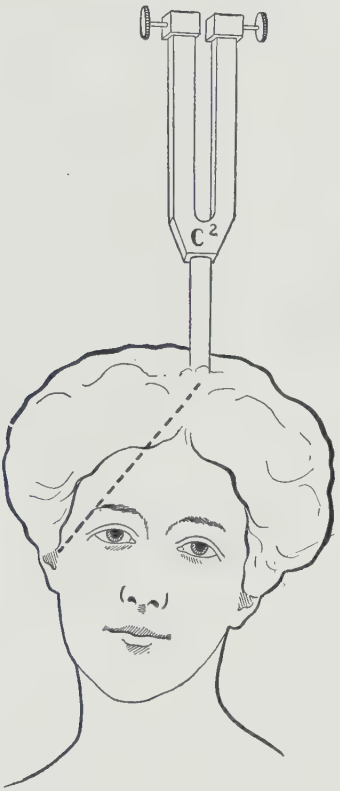
The Watch Test.—This instrument has long been used to test the acuteness of hearing. As a diagnostic aid it is far inferior to other tests, but is mentioned because of its common use. It may be used to measure the hearing distance (*a*) by approaching the ear with it; (*b*) by firm contact with the auricle if not heard by air conduction; (*c*) by contact with the mastoid process; (*d*) by placing it between the teeth and noting in which ear it can be heard most plainly, as in

the Weber experiment; (*e*) for comparison from time to time during treatment. Its drawbacks are: Watch-ticks are not standardized; the hearing for the watch is no indication whatever of the hearing for conversation; the patient becomes accustomed to the sound of one watch and apparently hears it better when there is no real improvement in hearing; the patient gets the habit of using his own watch

Fig. 379

The Weber experiment. The patient is deaf in the left ear and the sound lateralizes to the left ear, thus indicating disease of the sound-conduction (middle ear) apparatus to the left ear.

Fig. 380



The Weber experiment. The patient is deaf in the left ear and the sound lateralizes to the right or good ear, thus indicating disease of the perception apparatus (labyrinth) of the left ear.

frequently, and in incurable deafness his condition may be much the same as that of the neurasthenic entrusted with a clinical thermometer.

The Weber Test.—E. H. Weber first found that a normally vibrating tuning fork (Fig. 379) placed upon the skull is much more distinctly heard in that ear the external meatus of which is closed by the finger. In other words, the sound is referred to that ear in which a conduction

deafness has been produced. Clinically it has been shown that when the middle ear alone (including the Eustachian tube) is diseased, or when the external canal is obstructed, the sound of the vibrating tuning fork when on the median line of the skull, as the vertex, forehead, teeth or chin, is lateralized toward the affected ear; and that when the internal ear alone is affected the sound is lateralized toward the unaffected ear. This test is not altogether dependable in bilateral deafness nor in unilateral deafness when both the middle and internal ear are affected, as here are two opposing conditions, one tending to increase while the other tends to decrease bone conduction. Often, also, patients do not lateralize the sound, or are inaccurate. Hence this test should be accepted only in corroboration of the other tests; if inconsistent with them it should be ignored. In suppurative disease of the ear if the sound is first referred to the sick ear but subsequently changes and is referred to the well ear, it is very suggestive of involvement of the labyrinth.

Technic.—The Bezold large *A* fork (108.7 d.v.), or any fork between this and c^2 may be used (Dr. Alfred Lewy thinks c^2 is rather high, but it is used by many others). The vibrating fork is placed in the median line on either the vertex, forehead, glabella, teeth, or chin, and the patient asked to indicate in which ear the sound appears to be loudest. This is entered on the record. Patients often have the preconceived notion that they must hear it louder in the better hearing ear. This should be overcome. In order to test the accuracy of the answer the following simple procedure will often suffice: If the patient says, "I hear it louder in my right ear," the surgeon closes the right ear with his finger (the fork meanwhile remaining in place and vibrating) and asks, "Now where do you hear it?" If the patient then imagines the sound is referred to the open ear he may be known as inaccurate.

The Schwabach Test.—Schwabach first noticed that the sound of the tuning fork through the cranial bones in conduction deafness was heard longer than normal. The explanation of this is still open for discussion. It is at least partially due to the interference with the entrance of adventitious sounds from without, and with corresponding interference with the egress of some of those received through the cranial bones. The practical application of this is the bone conduction for the tuning fork as compared with the known normal for that fork. This test is very valuable in connection with the relative air and bone conduction test (Rinne test). Markedly prolonged bone conduction indicates a conduction deafness. Markedly shortened bone conduction indicates an internal ear lesion or disturbance, *i. e.*, nerve deafness. That this is not necessarily an organic lesion is shown by cases described by Alfred Lewy (*Laryngoscope*, March, 1913). Sometimes when the patient ceases to hear the fork by bone conduction, if the fork is removed for a few seconds and then replaced without having been struck again the patient again hears it. This is known as the "fatigue symptom," and is supposed to be due to fatigue or

exhaustion of the nerve, and to occur in neurasthenia and hysteria. In combined cases, *i. e.*, cases of mixed conduction and perception deafness, the Schwabach test may show the bone conduction somewhat shortened, slightly lengthened, or approximately normal. On account of the affection of the perception apparatus the disturbance of the conduction apparatus fails to bring about the usual increase in bone conduction.

Technic.—The fork for this purpose should be free from overtones, not so low that the vibrations are transmitted as concussions to the skull nor so high that it is difficult to distinguish between the air and bone conduction. The best fork is the Edelmann-Bezold *A* (108.7 d.v.) as recommended for the Weber test. Any good fork of sufficient intensity and duration, free from overtones, between *A* and c^2 may be used, but the Reiner d♯ (153.8 d.v. with clamps) is next choice. The normal register of the fork must have been ascertained previously by trials on normal persons. In order to reproduce even results the fork must always be struck on the same object, in the same manner and with the same force, and applied to the skull of the patient with the same degree of pressure. For instance, in using the Bezold *A*, the fork may be dropped from the vertical to the horizontal by its own weight, striking on the examiner's knee (the examiner's thigh is flexed to a right angle with his body and the handle of the fork just touches the thigh at the beginning of the fall), the fork is then rested by its own weight on the patient's skull. Or one may use a small rubber hammer made for the purpose of striking the tuning fork, or the rubber hammer used by neurologists for obtaining tendon reflexes will do. The number of seconds from the stroke till the patient no longer hears the fork is noted, preferably on a stop watch, and entered on the record "Schwabach—seconds." (If preferred one may record the per cent. of normal, *e. g.*, S. 40 per cent. or 150 per cent.) The patient must be instructed to raise his hand or otherwise indicate the moment he no longer *hears* (not feels) the fork.

The Rinne Test (Combined Testing of Bone and Air Conduction).—This is a very valuable test. If one holds the handle end of a vibrating tuning fork against the mastoid process until the tone is no longer heard, and then brings the prongs near the external auditory meatus (Fig. 379 and 380), the sound will again be heard, the length of time the tone is heard through the air being double or treble, according to the fork used, the hearing time through the bone. This is the "Positive Rinne." It occurs normally. It also occurs in nerve deafness, though in this condition both the bone and air conduction are shortened—"Shortened Positive Rinne." In a pure conduction deafness the bone conduction is relatively lengthened while the air conduction is relatively shortened. When this condition advances to a point where bone conduction exceeds air conduction we have a "Negative Rinne." Negative Rinne, but with both bone and air conduction very much shortened, may also occur in severe nerve deafness. Plus-minus Rinne is a term applied when bone and air con-

duction are equal. Indefinite Rinne, when air conduction is entirely absent. False Rinne, when one ear is totally deaf and the fork apparently heard on the mastoid of the deaf ear is really heard in the other ear. The bone conduction as found with the Rinne test should corroborate the Schwabach test. Occasionally bone conduction is prolonged for the *A* fork, and shortened for the a^1. This condition has been found in syphilis by Norval Pierce and Alfred Lewy in a few cases.

Fig. 381

Showing the Rinne *a'* fork in position on the mastoid process in the Rinne test.

Fig. 382

Showing the Rinne *a'* fork held close to the ear in Rinne's test; indeed, the prong tips should be within the concha.

Technic.—The best fork for this purpose is the Edelmann a^1 (435 d.v.). It is free from overtones and of sufficient intensity and duration, and yet the tone is not carried through air conduction to the opposite ear from the one being tested, c (128 d.v.), d♯ (153.8 d.v.), c^1 and c^2 may also be used if they fulfil the above indications. V. Mueller & Co., have recently devised a c^2 fork that appears practicable. The fork for this test should be carefully selected, as it is the most frequently useful one. The same degree of force and the same object (non-metal) for striking should always be used. Striking the fork on the knee-cap does very well. It is then firmly placed with the end of the handle on the mastoid process over the antrum, being held by the handle near the prongs. Care should be observed to use uniform pressure and to avoid contact with the auricle or hair; when the patient indicates that the sound is no longer heard, the fork is held suspended with the prongs flatwise toward and as near as possible to the concha without touching. In this position the sound is heard the best and longest. If abundant hair prevents the fork being held in this manner it may be held prongs up. If the Schwabach test shows greatly increased bone conduction it often saves time to get the air conduction first in making the Rinne test, as it will probably be a "negative." The length of

time the fork is heard by bone conduction and by air conduction is measured in seconds, preferably with a stop watch, from the time the fork is struck, one stroke sufficing for both parts of the test, and is entered on the record "Rinne +12:35" or "Rinne −20:15" for example; the bone conduction first; or if written as a fraction ($\frac{12}{35}$) the bone conduction is the numerator. The normal register for the fork used must be known.

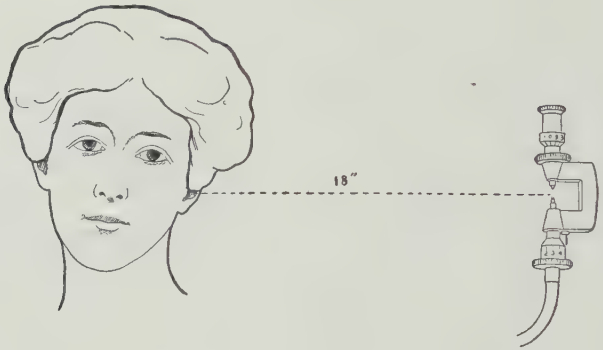
The Low Limit.—Normally the low limit is about C_2 (16 d.v.), but some persons with otherwise normal hearing fail to distinguish this tone. However, failure to hear G_2 (24 d.v.) may be safely put down as indicating some loss of hearing for lower tones. Bezold states that failure to hear C_1 (32 d.v.) in conduction deafness indicates ankylosis of the stapes. Loss of hearing for low tones practically always occurs to some degree in conduction deafness; the greater the loss the greater the probability of stapes ankylosis. It rarely ocurs in pure nerve deafness (except congenital), but of course occurs in combined cases. The mass vibrations of the lower tones are transposed by the system of levers comprising the membrana tympani and chain of ossicles into molecular vibrations, in which form they are transmitted to the perilymph. Anything interfering with the function of the conducting apparatus hinders the transmission of low tones.

Technic.—The examiner begins with the lowest fork; if this is not heard the next one higher is used, first before one ear than the other, until the patient, whose eyes should be closed during the procedure, indicates that he hears the tone. The lowest fork heard by each ear is entered on the record. If one uses but one fork for estimating the hearing for low tones C (64 d.v.) is a practicable one. It must be weighted to prevent overtones, and its normal register must be known. One can then enter on the record the fraction or percentage of time as compared with the normal that this fork is heard. Shortening of the hearing time indicates loss of hearing for low tones.

The High Limit.—Edelmann states that the educated ear can distinguish f^7 (22,097 d.v.) on a good Galton whistle (Fig. 381), and d^7 (18,581 d.v.) on a Schulze monochord. The writer also found d^7 the limit on the Struycken-Schaefer monochord, but many normal ears do not hear beyond c^7 (16,554 d.v.). After the age of about 50 years the high limit declines, owing to senile changes. According to Zwaardemaker at the beginning of senility the limit is about a^6; in old age about g^6. These data should be borne in mind in estimating the significance of tests for the range of hearing. Any marked loss of the upper range indicates some pathological process in the internal ear. When associated with a conduction deafness it indicates the probability of beginning degeneration in the basal turn of the cochlea. A moderate loss for high tones occurs quite commonly in spongification of the labyrinthine capsule. The writer is of the opinion that any marked loss of the upper tone limit is of unfavorable prognostic import when it occurs in any form of deafness except hysterical and in acute suppurative otitis media.

Technic.—(*a*) The Galton whistle (Edelmann's) (Fig. 383). This whistle has an adjustable aperture and graduated pipe-length, both operated by screws, and is blown by compression of a bulb. A scale for translating the tones into their proper musical designation accompanies each instrument. The whistle must be blown gently as it is difficult to exclude the opposite ear even when the meatus is occluded.

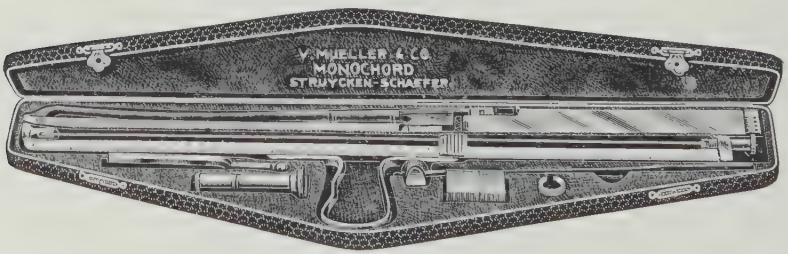
Fig. 383



Testing the hearing with the Galton-Edelmann whistle at eighteen inches.

Begin above the high limit and gradually lengthen the pipe by the screw until the sound is heard as a clear whistle (as distinguished from the blowing sound). The number of the line and the aperture distance, or its equivalent in musical terms is entered on the record. The small whistles of American make are practically useless except in cases with very marked loss of upper tone limit. All whistles of course test only air conduction.

Fig. 384



Monochord.

(*b*) **The Monochord** (Fig. 384).—This instrument consists of a metal frame on which is stretched a piano wire. On the frame and wire is fitted a block, which by its position regulates the pitch. A bone button can be attached to one end of the frame, which is held in contact with the mastoid process to test bone conduction for high

tones, an advantage which this instrument has over the whistle. Transverse vibrations are caused by striking the wire with a small hammer or drawing a violin bow across it. For the highest tones longitudinal vibrations are used, and these are obtained by rubbing the wire lengthwise with a felt pad moistened with turpentine and benzole, or carbon tetrachloride. (A felt-tipped bottle which keeps automatically moist is furnished with the instrument.) The range of the Struycken-Schaefer monochord is from g^1 to above the high limit. The frame is calibrated so that the pitch can be read directly for longitudinal vibrations, and in centimeters for the transverse vibrations, which requires reference to a scale for translation into musical terms. In obtaining the transverse vibrations (low tones), the instrument must be rested on a table to act as resonator, as otherwise the tone is too thin. In obtaining the high tones the patient must distinguish between the rubbing and the clear tone, but this is not difficult. The directions for use which accompany this instrument as well as the Galton whistle must be carefully studied.

(*c*) The c^4 (2048 d.v.) fork.—By using the large size c^4 fork of either the Bezold, Hartmann, or the Reiner set, one may test the high limit quite satisfactorily and more simply than above described. The fork is stroked gently, tapped with the finger or struck on metal according to the degree of loss for the upper tones, or the examiner can by alternately holding the vibrating fork before the patient's and his own ear (if normal) determine if the upper limit is normal, slightly short, moderately short or very much short, and so enter it on the record. The small size c^4 forks seldom vibrate long enough to perform this test satisfactorily.

Unilateral Total Deafness.—If both ears are occluded by the moistened fingers a loud fork or voice can still be heard. It is evident that though one ear be totally deaf the other cannot be entirely excluded from hearing by simple occlusion of the meatus, therefore it is necessary to use one of several methods that have been devised, all of which operate both by occluding and producing noise in the ear which it is desired to put out of commission temporarily, while the supposedly totally deaf ear is tested. When one of these devices is properly applied to one ear, if the other is totally deaf the patient will not hear even a loud voice (unless shouted directly toward the head).

The Neumann Noise Apparatus. — This is an electrical device which operates with either a direct or an alternating current. It consists of a rheostat, and interrupter and two telephones, fitted with ear-pieces, which fit snugly into the external meati. A switch causes the noise to be heard in either or both ears at the will of the operator. The rheostat controls the intensity of the noise. The instrument can also be used to discover malingerers who claim unilateral deafness.

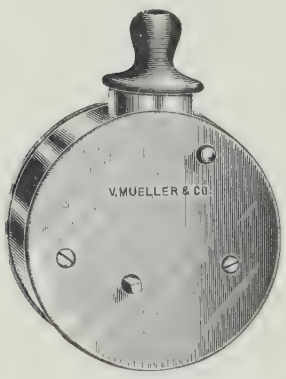
The Barany Noise Apparatus (Fig. 385).—This is a clockwork buzzer, which when wound up is operated by pressing a button while the ear-piece is in place.

The Pierce Method.—A *C* fork (64 d.v.) of sufficient loudness and duration (the one recommended for the low limit or a Koenig C fork will do) is fitted on the handle end with a conical ear-piece, which is moistened, and while the fork is vibrating loudly placed firmly in the external meatus.

Other methods are the running of a stream of water or air under slight pressure into the canal of the ear to be excluded.

The Gellé Test.—If the air is compressed in the external canal of the normal ear (using an air bag with a snug fitting ear-piece), the tone of a vibrating tuning fork placed on the vertex or mastoid will be perceived greatly diminished. According to Gellé if the stapes is ankylosed the pressure in the external canal cannot be transmitted to the labyrinthine fluid and the test is then negative. A more practicable method of performing this test, as devised by Barany, is as follows: A branched or "T"-shaped auscultation tube is used; two ends carry snug-fitting ear-pieces, the third a mouth-piece. One of the ear-pieces is held tightly by the patient in his external meatus, so that no air escapes; the other likewise by the examining physician; the third is used to compress the air in the tube and in the external canals by the physician blowing into it. The stem of the vibrating tuning fork is placed about the middle of the rubber tubing. If the stapes is not ankylosed, both the patient and the physician will hear the sound greatly diminished during compression of the air in the tube (unless the hearing is already very poor). Thus the examiner has a control test.

Fig. 385



Barany's noise apparatus.

Bing Test.—No. 1.—The small end of a speaking trumpet is fitted into the free end of a catheter, which is inserted into the Eustachian tube so that the sound waves enter the cavum tympani and come into direct contact with the foot plate of the stapes and the membrane of the round window. If the speech is heard decidedly better in this way than with the end of the speaking trumpet in the external meatus, the inference is that the interference with conduction is outside the stapes, that is, in the incus, malleus, or drum membrane.

No. 2.—Bing claims that after the tone of a vibrating tuning fork on the vertex becomes inaudible it is again heard if the meatus is occluded with the finger, if there is a labyrinthine affection. As this occurs normally, the test is useful only in severe deafness. If there is a conduction deafness the sound is not again heard when the meatus is occluded.

The various forms of deafness are described in detail under their respective headings.

The Function Tests of the Vestibular Apparatus.—The whole question of the labyrinth will be considered together, as there must be such a constant reference, back and forth, to the theoretical and practical problems that the student will find it expedient to have all the data grouped. (See Chapters XLIX and LI, *The Labyrinth: its Physiology, Functional Tests, Pathology, and Disease.*)

CHAPTER XXXIV

GENERAL ETIOLOGY OF DEFECTIVE HEARING

Defects of hearing may arise from any condition that affects the functional integrity of the conduction or the perception apparatus of the organ of hearing. It may be stated, as a general law, that the deeper (nearer the acoustic centre) the lesion, the more profound is the disturbance of hearing.

A. **Defects of Hearing Due to Lesions of the Auricle.**—This division of the subject may be passed by without analysis, as there is but slight impairment of hearing, even from the total loss of the auricle.

B. **Defects of Hearing Due to Affections of the External Meatus.**—(*a*) Inspissated cerumen. (*b*) Furunculosis. (*c*) Dermatitis. (*d*) Eczema. (*e*) Foreign bodies, animate and inanimate. (*f*) Exostosis of the meatus. (*g*) Collapse of the cartilaginous meatus. (*h*) Congenital artresia of the meatus. (*i*) Congenital absence of the meatus. (*j*) Cholesteatoma.

A glance at the foregoing analysis makes it apparent that hearing is diminished on account of the obstruction to the transmission of sound waves through the external auditory meatus and by the congenital absence of this canal. Congenital absence of the external auditory meatus is nearly always attended with absence of the middle and the internal ears, hence the deafness may be attributed more to the latter than to the former.

Cholesteatoma within the meatus is usually coincident with the same process in the middle ear and the pneumatic cells of the mastoid, hence the defect of hearing is largely due to the condition of the middle-ear and the mastoid spaces.

With these exceptions, the obstructions in the meatus account for deafness. It should be said, however, that inspissated cerumen in the meatus is often a sign of middle-ear catarrh, and the deafness may be partially due to this condition.

Collapse of the cartilaginous meatus is usually found only in the aged. The deafness in such cases may be due in part to senile changes in the middle ear and labyrinth.

C. **Defects of Hearing Due to Affections of the Drumhead.**—(*a*) Perforation. (*b*) Thickening. (*c*) Calcareous deposits. (*d*) Cicatricial tissue. (*e*) Cicatricial bands extending to the ossicles and the wall of the middle ear. (*f*) Retraction. (*g*) Bulging or pouching. (*h*) Inflammation (myringitis). (*i*) Herpes. (*j*) Traumatic rupture. (*k*) Fracture of the handle of the malleus. (*l*) Atrophy (lack of normal tension).

It may be stated as a general acoustic law that anything which disturbs the normal tension existing between the drumhead, the ossicles, and the labyrinthine fluid will result in an impairment of hearing. It should be noted that in nearly all of the foregoing conditions the normal tension is disturbed, hence the deafness.

In a number of lesions of the drumhead there are, of necessity, pathological changes in the middle ear which in part account for the deafness. For example, perforation of the drumhead is nearly always attended with either chronic suppuration or cholesteatoma of the middle ear, and possibly of the attic, the antrum, and the mastoid cells. In thickening, scars, cicatricial bands, calcareous deposits, retraction, and atrophy, middle-ear disease, usually of a chronic inflammatory nature, is present, and in a large measure accounts for the defective hearing. In simple myringitis, herpes, traumatic rupture, and fracture of the handle of the malleus, the middle ear may not be involved and the deafness is transitory.

D. **Defects of Hearing Due to Affections of the Middle Ear.**—(*a*) Simple catarrhal otitis media. (*b*) Catarrh with adhesions. (*c*) Sclerosis of the mucous membrane. (*d*) Cholesteatoma. (*e*) Acute suppuration. (*f*) Chronic suppuration. (*g*) Ankylosis of the ossicles. (*h*) Ankylosis of the foot plate of the stapes to the oval window (fenestra of the vestibule). (*i*) Adhesive bands uniting the ossicles to each other, to the walls of the tympanum, and to the drumhead. (*j*) Atrophic otitis media. (*k*) Anemia of the mucosa occurring with general anemia and debility. (*l*) Loss of tonicity of the stapedius and the tensor tympani muscles. (*m*) Congenital defect or absence of the middle ear. (*n*) Granulations in the middle ear. (*o*) Serous and mucous accumulations. (*p*) Caries of the ossicles. (*q*) Caries of the walls of the tympanum. (*r*) Polypus. (*s*) Otosclerosis or spongifying of the bony capsule around the oval window.

In the foregoing conditions we find the more common causes of deafness. The acoustic law given in the preceding section (C), namely, that the condition which disturbs the normal tension between the drumhead, the ossicles, and the labyrinthine fluid will cause deafness, applies with special force to the affections mentioned in this section. All or nearly all the pathological lesions named materially interfere with this tension, and thereby interfere with the transmission of the sound waves to the labyrinth. A study of these lesions will verify the general law enunciated at the beginning of this chapter, that as a general thing the deeper the lesion the more profound the deafness. For instance, a lesion affecting only the drumhead does not produce as profound deafness as does ankylosis of the foot plate of the stapes.

Sclerosis of the mucosa of the middle ear is often complicated with the same process in the bone beneath it. Chronic suppuration of the middle ear is also often attended with sclerosis (eburnation) of the bone.

This process may extend to the mastoid or to the bony capsule of the labyrinth, and thus augment the deafness.

The author has often seen cases in which the hearing was improved only after the administration of iron and arsenic. These patients were

anemic and suffered from general debility of a chronic type. Whether the improvement was due to an increased tone of the stapedius and the tensor tympani muscles, or to an increased tone and vital energy of the whole organ of hearing, would be difficult to determine. T. M. Rumbold believed that the trouble was in the muscles. This may be true, as there may be a lack of muscular tonicity here as well as elsewhere in the body. It may be said with equal certainty that all the tissues of the body, including those of all parts of the auditory apparatus, are lowered in tone and vital energy. We therefore think that the deafness due to or existing with general anemia, accompanied by seeming loss of muscular tone of the tension muscles of the middle ear, is probably due to a lowered vitality of all the parts concerned in audition.

Granulations and polypi in the middle ear not only interfere with the transmission of sound waves through the middle ear, but they often also obstruct the external meatus. They usually signify necrosis of the bony walls of the tympanum and an involvement of either the cranial cavity, the mastoid cells, the sigmoid sinus, the jugular vein, or the labyrinth.

Ankylosis of the foot plate of the stapes is a serious condition, inasmuch as it is usually impossible to permanently overcome it. The deafness and tinnitus are great and exert a depressing influence upon the patient. Great care should be exercised by the otologist in giving the prognosis in this class of cases. He should not hold out false hope of ultimate recovery, but he should so couch his language that the patient will not entirely abandon hope. It is the physician's office to cheer as well as to treat his patients. This is doubly true in hopeless cases, as they are often despondent to the point of suicidal mania. Fixed attention arouses the benumbed organs, and even though a course of office treatment is not advisable, the patient should be told to observe under what conditions he hears most clearly and to seek to adapt himself to his environment. Expectant attention is thus aroused, and the usefulness of the auditory apparatus is maintained at as high efficiency as is possible. In addition to the above, rest is beneficial and the organic salts of iron should be administered.

E. **Defects of Hearing Due to Affections of the Eustachian Tube.** —(*a*) Catarrh. (*b*) Fibrous thickening of the mucosa. (*c*) Fibrous bands across the lumen of the tube. (*d*) Fibrous rings or stricture of the tube. (*e*) Lymphoid hypertrophy within the tube. (*f*) Hypertrophy of the mucosa. (*g*) General sclerosis of the mucosa. (*h*) Paralysis of the palatine muscles which regulate the patency of the mouth of the tube.

The chief function of the Eustachian tube being to maintain the equilibrium of air pressure between the air in the middle ear and that external to it, an obstruction to the normal passage of air destroys the equilibrium. The normal tension of the drumhead, the ossicles, and the labyrinthine fluid is disturbed, and deafness and tinnitus result.

It is not usually recognized that lymphoid hypertrophy plays a prominent part in Eustachian obstruction. This must be true, however, as there is a considerable quantity of such tissue in the mucosa of the tube,

especially near its pharyngeal end. The same pathological processes which cause hypertrophy of the pharyngeal and the faucial tonsils will also cause hypertrophy of the tubal lymphoid tissue. We may, then, speak of a tubal or "Eustachian tonsil" as a cause of Eustachian obstruction.

In long-continued catarrhal or suppurative inflammation of the middle ear, fibrous thickening or fibrous bands may form in the Eustachian tube and give rise to persistent deafness and tinnitus unless relieved by suitable treatment. If air is not admitted to the middle ear in sufficient quantity, the drumhead becomes retracted on account of rarefaction of the air within the middle ear, the handle of the malleus is drawn inward and rotated on its axis, and the chain of ossicles is forced inward and compresses the labyrinthine fluids. Perhaps a more correct statement would be to say that the normal tension between the drumhead and the labyrinth is lost, and deafness and tinnitus result.

Tubal catarrh (salpingitis) is much more common than is generally supposed, and no doubt many of the so-called cases of middle-ear catarrh are in reality of this type.

Since the normal patency of the tubes is controlled by the palatine muscles, any condition which affects their innervation or motility will cause defective hearing. These conditions will be considered in the next section.

F. **Defects of Hearing Due to Affections of the Epipharynx and the Fauces.**—(*a*) Adenoids. (*b*) Epipharyngeal catarrh. (*c*) Polypi or other neoplasms. (*d*) Disease of the faucial tonsils. (*e*) Adhesions of the anterior and the posterior pillars of the fauces to the tonsils. (*f*) Suppurative inflammation of the epipharynx. (*g*) Paralysis of the palatine muscles (*e. g.*, postdiphtheritic). (*h*) Infections occurring during the course of exanthematous fevers.

In this category are conditions which are sources of diseases of the ear which are attended with impairment of hearing. All inflammatory conditions which involve the mucosa about the pharyngeal orifices of the tubes sooner or later extend within their lumens and cause more or less obstruction. If the inflammation is of a suppurative type, the germs enter the tube and the middle ear, and may cause an acute suppurative otitis media. This may become chronic, and permanent damage to the entire middle-ear apparatus result.

Postnasal adenoids are recognized as frequent antecedents of tubal and middle-ear catarrh and deafness.

There has been much discussion as to whether adenoids extended over the mouths of the Eustachian tubes. The free extremities of the lateral adenoid masses do, no doubt, often occlude them. Perhaps a more important pathological factor is that postnasal adenoids are usually attended with severe postnasal catarrh, which in many cases becomes purulent in character. This often causes obstruction of the tubes and thus gives rise to disturbances of hearing as well as to structural changes in the middle ear and its appendages.

The etiological relationship existing between hypertrophy of the faucial tonsils and disease of the Eustachian tube and the middle ear has long

been recognized, although not as fully as it should be. Their relationship cannot be considered apart from that of the postnasal space, however, as the same conditions which affect one affect the other also. Thus the presence of enlarged faucial tonsils is usually attended with adenoids. Both being lymphoid tissue, they respond to the same irritation and enlarge simultaneously. Notwithstanding this fact, there are some conditions of the faucial tonsils which cause tubal obstruction independently of any effects due to the adenoids (C. R. Holmes).

The presence of diseased or enlarged tonsils produces chronic hyperemia of the mucosa of the epipharynx, and oftentimes a chronic catarrhal or suppurative inflammation is present. Enlarged-and diseased tonsils do not always stand out beyond the pillars of the fauces. A normal tonsil can neither be seen nor felt. Many of the pathological tonsils are flat and lie hidden behind the anterior pillar. Pynchon has called them "submerged tonsils." He has also suggested that if they are examined "on the gag," they will bulge forward and inward and come into full view. When thus examined they appear broad and flat, with an irregular surface. In some cases the lacunæ are filled with debris, epithelium, bacteria, and pus, while in others no such accumulations are to be seen. This does not prove that they are not present in the pockets or lacunæ, as upon introducing a tonsil hook into them, yellowish, round masses may be removed. In others the masses are encysted, probably from inflammatory closure of the mouths of the crypts. The point I wish to make is that even though the tonsils do not project beyond the pillars and are not apparently much diseased, they may be the seat of foci of infection, irritation, and septic material, which gives rise to chronic catarrh of the epipharynx and the Eustachian tubes. The material in the lacunæ affords a good medium for the growth of bacteria, the toxins of which enter the lymphatic and the blood-vascular systems and cause disturbances in remote parts of the body.

G. **Defects of Hearing Due to Mastoid Affections.**—As these conditions are secondary to and associated with pathological changes within the middle ear, they will not be discussed here.

H. **Defects of Hearing Due to Labyrinthine Affections.**—Defects of hearing due to labyrinth affections vary from slight to total deafness. The labyrinth affections causing deafness are: (*a*) The infectious inflammatory processes, such as acute diffuse suppurative labyrinthitis, circumscribed labyrinthitis, and diffuse serous labyrinthitis; (*b*) fracture through the petrous portion of the temporal bone; (*c*) hemorrhage in the labyrinth (Ménière's disease); (*d*) atrophic degeneration of the cochlear nerve endings as in adhesive process of the middle ear; (*e*) atrophic changes in the labyrinth as in boilermaker's deafness; (*f*) syphilis and tuberculous disease of the labyrinth; (*g*) change due to drugs, such as quinine, narcotics, alcohol, etc., and (*h*) otosclerosis affecting the cochlea.

CHAPTER XXXV

FOREIGN BODIES IN THE EAR. CERUMINOUS PLUGS IN THE MEATUS

Children often introduce foreign bodies into the ear for very different reasons from those which may be ascribed to adults. For example, children in their play and in the spirit of imitation will do what they conceive is being done by others. Their elders, in order to excite wonderment and admiration, will do sleight-of-hand performances, pretending to remove a knife or other object from the nose, mouth, or ears. Children are thus led to introduce various objects into their ears. Peas, beans, beads, gravel, buttons, bits of sealing wax, chewing gum, cherry pits, etc., are commonly found in the ears of children. Burnet relates a case of a woman from whom a bead was removed that had been introduced sixty years previously. Children are fond of the sensation of a smooth body, as a bead or bean, rubbed over the skin, and in this way they sometimes accidentally introduce them into the external meatus.

These may remain in place for a long time without causing any serious symptoms, and be overlooked by their parents and unnoticed by the child.

In *adults* the introduction of foreign bodies into the external meatus is more apt to be accidental, or the result of some treatment, as the introduction of a bit of cotton which is allowed to remain long after it has served its original purpose. Bits of pencil, toothpicks, twigs, and straw may be introduced into the meatus during efforts to remove cerumen or moisture, and remain in the meatus until symptoms arise which cause them to seek relief from their physicians.

Animate objects, such as roaches, fleas, flies, rosebugs, bedbugs, ixodes humanus, house-fly maggots, Texas screw-worms, and other living parasites are the source of great agony and discomfort when they enter the external meatus, on account of the clawing and twisting motion incident to their efforts to get food or gain egress from the cavity. The mode and place of sleeping influences the introduction of such objects into the meatus, as sleeping outdoors in a hammock or upon the ground, thereby inviting living insects to make their abode in this cavity.

J. F. Church narrates a case in which a sheeptick had been in a stockman's ear for two years. It was embedded beneath a mass of cerumen and blood, and was still living when removed. The sensation was that of an intolerable scratching, accompanied by excruciating pain and deafness, which would suddenly pass away. There would be intervals of a month or more in which there would be no pain or discomfort in the ear. At times blood clots admixed with cerumen were removed.

When he came under the observation of Dr. Church the pain was, and had been, severe for about four days, and extended to the mastoid region. There was a feeling of numbness over the corresponding side of the face. The meatus was filled with cerumen and epithelium, which was removed with a spud and a syringe. This being done, the deeper portion of the meatus was exposed to view, and a moving body was seen. It presented the appearance of a perforation in the drumhead, with slender maggots protruding through it.

The Texas screw-worm fly, or Compsomyia Lucilla macellaria, has been thought to be of Mexican or South American origin, although Dr. Williston, of Yale College, writes that "It grows especially from Canada to Patagonia." Its chief *habitat* in the United States, however, has been in Texas, hence its name.

Its ravages among cattle are common, and often occasion heavy financial loss by the destruction of its victims. It more rarely invades the human family, but has been known to cause death in a number of instances. Its favorite point of attack in man is the ear or the nose. This is easily understood when it is known that the insect is attracted by foul-smelling odors. Those, therefore, affected with ozena or chronic otorrhea are especially likely to be invaded. The worm in the act of invading the tissues performs a sawing motion, and can penetrate bone. Mackenzie reports cases in which the cranial cavity was penetrated by them, and death from meningitis resulted.

FOREIGN BODIES IN THE EAR

Treatment.—It is important that caution be given as to the great harm that may be done by unwarranted, unskilful, or untimely efforts to remove foreign bodies from the external meatus. It should be remembered that foreign bodies, especially inanimate ones, can do little or no harm so long as they are left undisturbed in the meatus. This, of course, is not true for an indefinite period of time, but it is true in the sense that there is no need of haste on the part of the attending surgeon. More harm has been done to patients by the efforts to remove foreign bodies than has ever been produced by the presence of bodies in the meatus. If a foreign body is smooth and causes no pain or discomfort, there is certainly no occasion for its hasty removal; if it is rough, and causes considerable pain and discomfort, there is more excuse for its immediate removal; but even then it may be much wiser to allay first the irritation and swelling, after which it may be removed with comparative ease with either the syringe, snare, or forceps.

I have seen cases in which the meatus was swollen and red from the unskilled attempts of members of the family to remove a foreign body. While thus swollen it was impossible for me to remove it immediately without a general anesthetic. In such instances I have first used antiphlogistic remedies and soothing applications for a few days, after which it was comparatively easy to remove the foreign body without any great difficulty or pain to the patient. If an insect or other live body gains

entrance to the meatus, the first step to be taken is to render it lifeless, after which its removal can usually be effected with a syringe.

Having thrown out this warning against meddlesome or unintelligent attempts to remove inanimate foreign bodies, we will discuss the best methods of procedure for this removal.

1. First inspect the meatus in order to determine whether or not a foreign body is present, and if present, its probable nature. This is important, as the method of procedure for its removal will depend largely upon the character of the body present.

2. Notice whether irritation or inflammation of the parts is present, and whether it is probable that the foreign body will do harm by remaining a few hours or days longer; and also as to whether it is probable that if immediate steps for its removal are taken, the effort would be rewarded by success. If the parts are swollen and inflamed to such an extent as to make it impracticable to remove it at once, it is better to wait until the swelling and inflammation are reduced by the use of hot, soothing lotions, and the application of leeches to the tragus. After a few hours, or at the most a few days, the swelling and painful condition will have subsided, thereby rendering the removal of the offending object a matter of comparative ease with little discomfort to the patient.

3. Syringing should first be tried, as the stream of water may be forced into the meatus beyond the foreign body, and thus dislodge it from the external auditory meatus. The position of the head should be considered in this and other methods of procedure, as the force of gravity will oftentimes materially aid in the removal of the object. The head should, therefore, be inclined toward the affected ear. Zaufal found, in 109 cases of foreign bodies in the external meatus, that he could remove 92 of them with the syringe, thereby demonstrating that nearly 90 per cent. of foreign bodies may be removed by this method. I fear that in the average practitioner's experience 90 per cent. of the removals have been attempted with either forceps or the so-called "ear hook;" whereas the 90 per cent. of successful efforts should have been accomplished with a syringe, while in the other 10 per cent. it may have been proper to resort to the forceps and ear hook.

4. The agglutination method was recommended by Riverias in 1674, by Celsus in 1806, and was revived by Lowenberg in 1872. It consists in placing heavy glue on the end of a piece of tape or a camel's-hair brush, applying this to the foreign body in the external meatus and leaving it in position until the glue becomes firmly enough fixed to bring the foreign body with it when traction is exerted upon it. This is probably one of the best methods, for most of the cases, after syringing has failed. It is to be recommended on account of the absence of instrumentation, whereby the meatus is so often seriously injured.

A strip of adhesive plaster may be introduced into the meatus, applied to the foreign body and heated by focussing the rays of light upon it with a convex lens. This softens the adhesive material and allows it to become fastened to the foreign body, after which it may be removed by traction upon the adhesive strip.

The agglutination method is not used as often as it should be, as most physicians mistakenly think that a pair of forceps or the foreign body hook, usually present in the pocket case purchased upon graduation, are the instruments *par excellence* for this purpose.

5. The foreign body hook is, perhaps, less harmful in the hands of an inexperienced operator than the forceps, and is, therefore, to be recommended as a better instrument for the removal of foreign bodies from the external meatus. It should be so introduced as to allow the short hook to pass inward with its side against the wall of the meatus until it passes beyond the foreign body, when it should be rotated to bring the hook back of the foreign body. Slight traction should then be made upon it, with the view of dislodging the foreign body from its position in the meatus. If it fails to do this, it should be withdrawn and reintroduced in another position, thereby to find a point at which the body may be loosened. If the foreign body has passed beyond the isthmus of the meatus and is lodged in the recess formed by the membrana tympani and the floor of the meatus, the hook should be introduced above the foreign body, as there is greater space at this point for the outward movement of the impacted mass. The convexity of the floor of the external meatus forms a favorable fulcrum upon which the lower portion of the foreign body rests, while the upper portion makes the outward excursion. It will be necessary, however, in some cases to introduce the hook either posteriorly or anteriorly in order to slowly dislodge the mass from its fixed position. After this has been done, the hook should be introduced above the mass, completely dislodging it from its point of impaction. Its removal through the cartilaginous meatus may then be accomplished with ease and little discomfort to the patient.

6. Various forceps, designed for the removal of foreign bodies from the ear, have been devised and placed upon the market, none of which serve a very useful purpose. Young practitioners have great satisfaction in the thought that they have a full equipment at their command for the removal of foreign bodies from the ear. Beyond the satisfaction they thus afford, the instruments are of little value. It is with such instruments that untold harm and irreparable damage have been done, and not a few lives have been sacrificed to the enthusiasm of their owners, The foreign body has, in many instances, been forced through the drumhead into the middle ear, where the physician has left it, and it was only discovered at a later period during a mastoid operation.

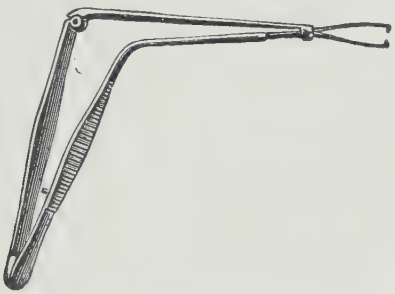
After a time its presence in the middle ear gives rise to necrosis and serious infection, followed by intracranial complications, such as abscess, meningitis, or sinus thrombosis, thrombosis of the jugular vein, labyrinthine necrosis, or transmission of infective thrombi to the lungs, the spleen, or the kidneys.

Having thus briefly, but pointedly, suggested the dangers attending the use of foreign body forceps, it may be said that they have a useful place, limited though it be, in the armamentarium of the physician.

The cautions given above are not for the purpose of discouraging the practitioner from using the foreign body forceps, but are intended to lead

him to use them with great circumspection after having tried all other means for the removal of the foreign body. Those devised by Dr. Samuel Sexton are, perhaps, the best upon the market (Fig. 386). They are so constructed that the toothed tips may be introduced at the sides of the body, while the blades remain practically parallel with the walls of the external meatus; this is a point of no small importance when we remember that most forceps for this purpose are so constructed that when the blades are spread apart the tips are at such an angle as to be easily forced into the meatal wall as they are pushed inward beyond the foreign body. Whatever instrument may be used, great care and delicacy of manipulation should be exercised, to avoid laceration of the meatus. If the foreign body is removed, the laceration will be of small moment, as it can be properly treated and quickly healed; if, however, the efforts to remove the foreign body are unsuccessful, the laceration may become a very serious complication, as the parts cannot, for obvious reasons, be properly treated. Swelling, infection, and inflammation may take place, which will still further interfere with the removal of the foreign body. Great discomfort results, and the condition is a serious menace to the well-being of the patient.

Fig. 386



Sexton's foreign body forceps.

7. *Postauricular incision* for the removal of foreign bodies is a very ancient method of procedure, as Paul of Aegina suggested its use. Von Tröltsch, in his text-book on *Surgical Diseases of the Ear*, suggested that in infants the incision is most effective when made above the auricle in the squamous region, as this area is depressed at that age, thus admitting of easy access to the meatus without injuring the postauricular artery. He thinks the injury to the artery should not be done needlessly, as it is an important source of nutrition to the auricle. With our improved methods of surgery and asepsis, we do not now fear an injury to this artery, and would not, therefore, make the incision above the auricle with this object in view. The incision in this position is, however, undoubtedly best adapted for the removal of foreign bodies which cannot otherwise be removed from the meatus of an infant on account of the oblique angle it forms with the squamous plate. The roof of the osseous meatus is gradually formed by the development

of the squamous bone, and extends inward at an obtuse angle, thus affording a favorable field for the introduction of instruments for the removal of foreign bodies. In adults, von Tröltsch suggests that the incision should be made posterior to the meatus, as its roof is now at right angles to the squamous plate.

With the antiseptic and aseptic methods now in vogue there should be little hesitancy in making a free incision in much the same manner as described for mastoid operations. The wound may be closed at once, union by first intention taking place. The skin of the cartilaginous meatus should be elevated as in the mastoid operation and lifted from its position. The foreign body is thus fully exposed to view, the meatus is shortened and enlarged, and instrumentation for its removal becomes comparatively easy. The patient should be under the influence of a general anesthetic. A portion of the osseous meatus should be chiselled away, if necessary, in order to facilitate the removal of the foreign body.

Urbantschitsch reports a case of an oat husk which entered the Eustachian tube while the patient was chewing an ear of grain. It passed through the tube into the middle ear, and thence into the external meatus.

ANIMATED FOREIGN BODIES IN THE EAR

Treatment.—Great concern is usually occasioned by the entrance of an insect or other animate body in the external meatus, on account of the clawing and scratching and penetrating movements attending its presence. Great noises of the most distressing and horrifying character are sometimes present, due no doubt to the clawing and scratching against the drumhead. On account of the great mental disturbance of the patient, the physician should have well-formulated ideas as to the proper methods of procedure for the removal of the insect, as he will otherwise be led to resort to methods in his haste and anxiety which will probably be unsuccessful and will only add to the pain and discomfort of the patient. I would, therefore, make the following suggestions:

(*a*) Avoid the use of instruments. It has been found by experience that animate objects are not readily removed by the use of forceps or other instruments. They have the power of clinging tenaciously to the skin of the meatus with little hooklets in the case of maggots, and with the feet in the case of fully developed insects.

(*b*) Drown the insect. This can usually be done with oil; if oil is not at your command, water may be used instead. If maggots are within the meatus, a 50 per cent. solution of chloroform should be used for this purpose, as oil or water seems to have little power to cause their death.

(*c*) If for any reason it is desired to remove them immediately without waiting to render them lifeless, the syringe should be used, as in this way they may sometimes be removed with great ease. On the other

hand, the method is oftentimes not successful until they have been rendered lifeless by drowning. If maggots are present, the fumes of chloroform blown into the ear from the bowl of a pipe will almost instantly render them lifeless. Chloroform may also be dropped into the ear for this purpose, with more certain results. After they are rendered lifeless, the insects or larvæ are easily removed with the syringe, and it will rarely be necessary to resort to the use of forceps. Should it become necessary, however, to resort to them, they should be used with great caution, as otherwise the meatus and drumhead may be injured. The use of chlorinated water is of value in rendering them lifeless, especially the larvæ. It is not, however, as efficacious as chloroform.

(*d*) The agglutinative method may be used for the removal of dead insects from the ear, as described under Foreign Bodies in the Ear.

FOREIGN BODIES IN THE EUSTACHIAN TUBE AND MIDDLE EAR

Mayer[1] reports three cases of foreign bodies in the Eustachian tube: one, a grain of corn, was in the bony portion of the tube, while the others were in the cartilaginous end. They may enter the tube either through the middle ear or the epipharynx. If there is a perforation in the drumhead, a small grain or other substance may enter the middle ear through it, and thence pass to the Eustachian tube. Foreign bodies which are unskilfully or roughly handled in the effort to remove them from the external auditory meatus may thus be driven into the middle ear, whence they may gain entrance into the Eustachian tube.

The use of Eustachian bougies has, in the past, been a fruitful source of foreign bodies in the tubes from accidental breaking while being used. Formerly the bougies were armed with feathers, cotton, or hair, for the introduction of medicaments, and were, consequently, more likely to be broken in the tube. Better and smoother instruments are now used, hence the accident occurs less frequently.

Voltolini has recommended the galvanocautery for the removal of firmly embedded organic substances, as beans, etc., from the meatus and the middle ear. At various sittings small portions are thus destroyed, until the whole is finally disintegrated and removed. This method of procedure should be attempted with great caution, as there is considerable danger of exciting inflammation of the contiguous parts.

If the foreign body is so deeply and firmly embedded in the middle ear as to render it impossible to remove it by simple and direct methods, the postauricular incision, such as described under mastoid operations, should be made, and, if necessary, a portion of the bone of the meatus may be chiselled away. When it is thus exposed, an attempt should be made to remove it with a stream of water. Should this fail, forceps may be used.

[1] Monatsschrift f. Ohrenheilkunde, Jahrg. v, Nr. 1.

Foreign bodies in the cartilaginous end of the Eustachian tube may sometimes be seen with a postrhinoscopic mirror as they protrude from the mouth of the tube. In such cases it is often possible to seize the protruding end with a pair of curved forceps through the mouth and thus remove it. If this cannot be done, the drumhead may be perforated by means of a V-shaped incision, if a perforation does not already exist, and air forced into the middle ear by means of a Politzer bag or other compressed-air apparatus with a suitable tip, applied at the external meatus. In this way the current of air may be made to enter the Eustachian tube and force the foreign body from its pharyngeal orifice.

CERUMINOUS PLUGS

Cerumen is the product of the ceruminous glands which are located chiefly in the cartilaginous portion of the external auditory canal. A few glands are also present at the commencement of the osseous portion of the canal. The cerumen is normally thrown off by the movements of the mandible (inferior maxilla) and by the exfoliation of the epidermis which lines the canal. When, however, from any cause the secretion becomes excessive in quantity, more tenacious in quality, or its discharge is mechanically obstructed, ceruminous plugs form in the canal and give rise to more or less disturbance of hearing.

Etiology.—The etiology may be studied under (*a*) diseases of the canal and middle ear; (*b*) obstructive lesions of the canal; (*c*) modifications in the character of the ceruminous secretion; (*d*) foreign bodies in the canal; and (*e*) improper methods of washing the ear.

(*a*) The diseases of the canal and middle ear which cause ceruminous plugs may be subdivided into hyperemia of the skin of the canal, diffuse and circumscribed eczema, and otitis media catarrhalis.

(*b*) Modifications in the character of the cerumen, as in increased adhesiveness and the admixture of epithelium and hairs, cause the retention of the cerumen.

(*c*) Foreign bodies in the external canal form the nuclei of ceruminous plugs. They may be solid substances, as beads, small stones, etc., or they may consist of dust, sand, or other finely divided particles.

(*d*) Improper methods of washing the ears are often responsible for the presence of ceruminous accumulations in the canal. Irritating soap-suds are introduced, the epidermis macerated in it, and the glands overstimulated. A mild dermatitis results. The corner of a towel or a washcloth is often twisted and screwed into the meatus, causing still further irritation, and oftentimes pushing the cerumen into the osseous portion of the meatus, where it remains, forming a nucleus for still more extensive accumulations.

Symptoms.—The symptoms vary according to the degree of occlusion, the position of the plug, the amount of secondary irritation and inflammation, and the preëxisting or associated lesions in the middle ear and labyrinth.

If the occlusion of the canal is incomplete in an ear which is otherwise normal, there will be but little impairment of hearing; if, on the other hand, the canal is entirely closed, there is marked diminution of hearing. If the plug is dislodged into the fundus of the canal against the drum membrane, the disturbance of hearing and the discomfort are much greater. In some cases the plug causes severe *inflammatory reaction* of the tissue immediately contiguous to it, which adds to the discomfort and the impairment of hearing. Reflex pains in the mastoid region are not uncommon in this condition.

If suppurative inflammation of the middle ear and the mastoid cells is present with the ceruminous plug, the symptoms are modified accordingly; that is, there is a mixture of the symptoms of the two conditions.

Pain is a symptom which is present only when the cerumen is hard and exerts pressure on the inflamed walls of the canal.

In general, it may be said that the patient complains of a feeling of fulness in the ears and the head, and occasionally of dizziness, vomiting, headache, stupor, facial paralysis, trigeminal neuralgia, brain irritation, eclampsia, blepharospasm, pain, etc.

The hearing may suddenly change from good to bad, or *vice versa*. When the drumhead is perforated the plug may improve the hearing by acting as an artificial membrane.

Diagnosis.—The diagnosis is made by inspecting the canal, either with a speculum or by lifting the auricle upward and backward. The plug appears as a yellow or brownish mass of greasy or granular material, which, upon probing, proves to be either soft, semisolid, waxy, solid, or hard as stone.

It may be mistaken for cholesteatoma, dried blood, a foreign body, cotton stained with secretion, etc. In some cases there is an excessive exfoliation of epidermis, which, becoming admixed with hairs and cerumen, lodges in the canal, thereby causing its occlusion. In these cases we have to deal with a pathological desquamation of epidermis rather than with a hypersecretion of cerumen.

Prognosis.—When sudden loss or diminution of hearing follows the introduction of water or other liquids into the meatus, the prognosis as to the hearing is good, as the disturbance is probably due to the swelling of the plug, which obstructs the canal. Cases complicated by either adhesive otitis or labyrinthine affections are not greatly relieved by the removal of the cerumen.

If we apply the tuning fork to the vertex, as in Weber's test, and the sound lateralizes to the obstructed ear, we gain no information, as the lateralization might be due to either middle-ear disease or to the plug. If, however, it lateralizes to the unobstructed ear, we may suspect labyrinthine involvement on the obstructed side.

Treatment.—The only form of treatment to be recommended is the removal of the cerumen by forcible injections of warm water with a syringe. If the plug has a moist appearance, or is soft to the probe, the injections may be made at once; whereas, if it is hard and lustreless, it should first be moistened by instilling a few drops of a solution of

bicarbonate of soda and glycerin in water; this should be repeated three or four times daily for about three days. The addition of the glycerin is advantageous on account of its hygroscopic property, which maintains the plug in a moist state between the instillations. When softened it may be removed with a syringe or with a cotton-wound probe.

In rare instances the use of a round-ended probe may become necessary on account of the firm adhesion of the cerumen to the meatus. Persistent injections will ordinarily remove all secretions. Dizziness, or even vomiting, is sometimes induced by the force of the stream, the intralabyrinthine pressure being disturbed by the inward movement of the foot plate of the stapes.

Keratosis Obturans, or Epithelial Plugs in the External Meatus.—In 1874 Wreden described this condition, calling it "keratosis obturans." It is caused by a chronic desquamative dermatitis, in which the epithelium is gradually thrown off and accumulated layer by layer in the fundus of the canal. More or less deafness results, according to the degree of occlusion and the proximity of the plug to the drumhead. It is often mistaken for cerumen, as its layers may be admixed with and its surface covered by it. A careful macroscopic or microscopic examination will clear the diagnosis. Mr. Richard Lake advances the theory that it is caused by a dry, scaly eczema, which is excited by a ceruminous plug, while Burnett suggests that it is due to an excoriation and slow exudation of dermoid cells, brought on by rough and clumsy attempts to clean the ear.

Pain in the meatus is the most constant symptom. In rare cases it radiates around the ear and over the temporal region.

After syringing the ear, the plug becomes whitish or grayish in color, on account of the removal of the outer layer of cerumen, which is readily soluble in water. It is firm and dense and more or less adherent to the walls of the meatus. After its removal, if placed in water, it does not soften and break up as cerumen does under like conditions. Its layers resemble sodden white parchment.

Treatment.—Before proceeding to remove the plug with the syringe, it should first be gently separated from the walls of the meatus with a flat applicator. This allows the stream of water to pass around and behind it, and facilitates its expulsion. If, however, it does not readily come away it should be removed piece by piece with a probe or forceps, one hour often being required for its accomplishment. Children do not calmly submit to the procedure, as it is somewhat painful; an anesthetic should, therefore, be given. Recurrences may be expected; hence, frequent examination and treatments may be necessary.

CHAPTER XXXVI

MALFORMATIONS AND NEOPLASMS OF THE AURICLE

MALFORMATIONS

Malformations of the auricle are of importance chiefly from a cosmetic point of view. The auricle plays such a small part in the function of audition that its entire absence does not materially influence the acuity of hearing. If, however, the auricle is so shaped as to occlude the meatus, it may materially interfere with the transmission of sound waves and thus impair hearing. In most cases, however, when there is a very marked defect there is also defective formation of the external auditory meatus, the middle ear, and the labyrinth; hence, diminution in hearing is usually due to other conditions than the changes in the auricle.

The malformations may be of a great variety of forms, ranging from a plurality of the auricle to its entire absence. Between these two extremes the auricle may be deformed to a slight degree, or it may be overdeveloped or misshapen in almost every conceivable way. It may be either arrested or overdeveloped. One part may be overdeveloped, while in another the development is arrested. It is not uncommon to see in a large company of people ears which project very markedly from the head, and which often give rise, especially among schoolchildren, to their possessors being called "yellow kids." The term "lop ear" is often applied to the same condition.

The defect may be either congenital or acquired. If congenital, it is due to a lack of closure of the branchial clefts and to a disproportionate development of one or more of the six tubercles or centres of development of the auricle. It may be unilateral or bilateral, usually the former. The bones of the face upon the side affected are usually also arrested in their development.

Stahl, in 1859, called attention to the fact that deformity of the auricular cartilage might be regarded as an indication of arrest of development of the skull, and that it bore a relationship to the development of the skull. Defective formation may consist of the entire absence of the auricular cartilage, although it is probable that in nearly every instance a careful examination will reveal a small cartilaginous growth beneath the skin. The arrest may take on the form of a simple shrivelling of the whole auricle, or a portion of it. On the other hand, it may consist of an excessive development of one part and a diminished development of another; or it may assume any irregular type of development, as a twisted shell, or it may be hooked, cone-shaped, fissured, or cauliflower-like in form.

Sometimes the upper portion of the auricle is turned downward from above and compressed against the middle portion, as is seen in the old statues of Pan (Politzer); or it may have deep indentations or horizontal fissures, and in rare instances it may be spindle-shaped. The tragus may be twisted inward, so as to close the meatus, or there may be an absence of the auricle with the exception of the lobule, which may be free or adherent to the adjacent skin. The meatus was present in a case of this kind reported by Schwartze. It opened beneath the lobule and led upward and inward to the drumhead.

The auricular appendages or supernumerary auricles, according to Virchow, consist of reticular cartilage, subcutaneous cellular tissue, and skin. They are usually located in front of the tragus, although they may be on the lobule, the side of the neck, or the shoulders. Saissy, in 1829, advanced the theory that malposition of the auricle from an improperly placed head-dress invariably led to arrest of development. He says: "Boys often wear their hats so low upon the head as either to push the ear outward and cause it to project from the head, or to compress it against the head and cause it to assume too close a position. The latter often occurs in females from confining the ears too closely with the head-dress. To remove the deformity, it is only necessary to correct the habit."

Maschziker, in 1864, in his text-book on *The Ear and its Diseases and Their Treatment*, states that ears are placed in malposition by too tightly drawn caps on children.

I have known mothers to bandage the ears of their little ones to bring them more closely to the head, even when their fathers had widely protruding auricles, and the children had evidently inherited the physical trait. Thus the scientific tradition still holds popular credence, and many a little child is made to suffer in consequence.

Saissy's views on the subject of imperforation of the external meatus were more nearly correct, as he regarded it as usually associated with a congenital and irremediable defect of the middle and the internal ears. The *etiology* of auricular deformity is to be found in the disordered development of the organ of hearing. There is insufficient closure of the upper two branchial clefts, which arrests or accelerates development of one or more of the six tubercles or centres of development, as shown by Minot, Talbot and others.

Classification. –Auricular deformities may be classed as follows:

(*a*) Entire absence of the auricle.

(*b*) Overdevelopment of the auricle (macrotia).

(*c*) Plurality of the auricle (polyotia, supernumerary).

(*d*) Arrested development of the auricle (microtia, shrivelled).

(*e*) Distortions of the auricle (cat-ears—as in the statues of Pan—shell-, scroll-, hook-, spindle-, cone-, fissure-, and cauliflower-like formations).

(*f*) *Fistula in auris congenita* is a remnant of the first branchial cleft, and was first described by Heysinger in 1870. It opens in front of the ear, either above or below the tragus, and is a blind canal filled with creamy secretion mixed with pus. When its mouth becomes closed

the secretion accumulates within the canal, which may be felt as hard nodules beneath the skin. Fistula auris congenita is of slight importance, and may be healed by laying it open with a knife and removing the epithelial lining and bringing the parts together again, after which they unite by first intention, and thus obliterate the canal. Mild caustic applications may be applied within the canal to excite inflammation and adhesions for the purpose of closing the canal.

Fig. 387 illustrates one of my cases of microtia. The drawing is from a plaster cast of the ear. The young man is healthy and has a normal ear upon the opposite side. The cartilages of the fragmentary auricle are not attached to the skull in any way except by the skin. There is an entire absence of the external auditory meatus, and bone conduction is nil upon this side. He came to me to have the ear "opened up," if I thought it advisable. As there was no bony meatus, and the autopsies on similar cases have shown the middle-ear apparatus and labyrinth to be absent or quite rudimentary, I advised him to leave the ear as it was.

Fig. 387

Author's case of microtia. The external auditory meatus, middle ear, and labyrinth are absent.

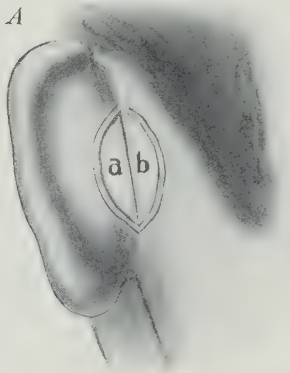
Treatment.—*Macrotia.*—Figs. 388 and 389 illustrate one of my cases of macrotia. The case was referred to me by F. G. Suker, for the reduction of the lop-ear. The boy was eleven years old, and presented numerous stigmata of degeneracy. His schoolmates called him the "yellow kid." It was, therefore, decided to overcome the defect by operating upon the auricles. This was done under general anesthesia.

The skin on the posterior surface of each auricle was incised with a knife, the line of incision extending in a curve from within one-fourth inch of the superior attachment of the auricle to within one-half inch of its inferior attachment. A second incision was begun at the upper point and extended backward and downward over the mastoid process one-half inch posterior to the attachment of the auricle, and made to join the inferior end of the auricular incision (Fig. 388). An ellipse or segment of skin not unlike a segment of orange peel was thus outlined. This was dissected from the auricle and the mastoid process. The second step of the operation consisted in cutting through the cartilage of the auricle, following the line of the auricular skin incision. The cartilage was then severed at the auriculomastoid junction, care being exercised to avoid cutting through the skin on the anterior surface of the auricle. The cartilage was next carefully separated from the anterior skin of the auricle (*a*).

The third step of the operation consisted in closing the wound (Fig. 389). This was done in such a way as to bring the auricle close to the

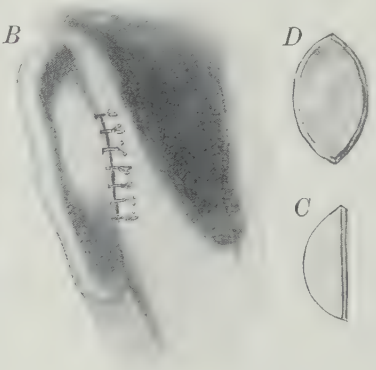
head, as the operation was done principally for this purpose. In order to do this, four deep stitches with silkworm gut were taken, so as to include the auricular skin, the auricular cartilage, the fibrous tissue over the mastoid, and the mastoid skin. These were drawn firmly together and secured. Ochsner's continuous horsehair suture was then used to bring the edges of the skin together.

FIG. 388



A, operation for macrotia, or lop-ear. An elliptical piece of skin (*a*, *b*) has been removed from the posterior wall of the auricle and mastoid process. *a*, the area of cartilage to be removed from the concha of the auricle.

FIG. 389



The operation for macrotia, or large projecting auricle: *B*, the sutured incision at the close of the operation; *C*, the cartilage removed from the concha of the auricle; *D*, the skin removed from the posterior aspect of the auricle and the mastoid process.

The superficial sutures were removed on the sixth day and the deep stitches on the ninth day.

The results of the operation were excellent. Before the operation the auricles at Darwin's tubercle were 3.5 cm. from the side of the head, and after the operation they were 1.5 cm. distant. Three months after the operation they were 1.25 cm. from the head.

NEOPLASMS OF THE EXTERNAL EAR

Othematoma.—**Definition.**—This is a disease of the auricle characterized by an effusion of blood between the perichondrium and the cartilage. It may occur spontaneously or from direct violence. When it occurs spontaneously it is probably due to degenerative changes in the bloodvessels of the fibrous bands which traverse the cartilage of the auricle. It is also probable that degenerative changes occur in the fibrous tissue.

Etiology.—Dementia seems to have a close relationship to the disease, as it is commonly found in the insane. Inhuman treatment of this class of patients has been so often charged, and it is more than probable that traumatism accounts for it among them to a large measure. This

is rendered more than probable by the fact that most of the cases have involvement of the left ear, because the blow from the right hand of the attendant would strike this ear. It must not be presumed, however, that this is the only cause, as the degenerative changes above referred to would be expected in this class of patients. The ex-champion pugilist, "Battling" Nelson, has othematoma, which was caused by numerous blows upon the ear in a series of boxing matches in remote places where he did not have the opportunity of applying hot water.

The condition is common among the wrestlers of Japan, traumatism being the probable cause.

Symptoms.—The tumor forms quickly, and this distinguishes it from perichondritis, angioma, and other neoplasms. The rapid development after an injury is quite characteristic. Its color is bluish, and it is rounded

FIG. 390

Othematoma with ossification following a history of dementia and traumatism. (Dr. G. McAuliff's case.)

and soft to the touch. It does not have the distinct fluctuation common to fluid sacs beneath the skin, but offers a doughy resistance. If it is due to traumatism it is usually quite large, and often involves the whole or the upper portion of the auricle; whereas if it is idiopathic it is often quite circumscribed, being limited to a nodule in the concha or other depression of the auricle. It is most common on the anterior or concave surface of the auricle (Fig. 390).

Pain is present in the traumatic variety, but is absent in the idiopathic. The tumor is opaque by transmitted light, whereas that of perichondritis is transparent. If the auditory meatus is occluded by the swelling, deafness and tinnitus are present. It should be borne in mind that the deafness may be due to the rupture of the eardrum from concussion. In the case of "Battling" Nelson, the hematoma became organized and caused permanent deformity.

Diagnosis.—The diagnosis is based upon the rapid development of the growth after an injury, the opaqueness by transmitted light, and the absence of febrile symptoms. In the spontaneous variety the rapid development of the tumor is quite characteristic.

Prognosis.—The traumatic variety ends by resolution more readily than the idiopathic variety, except when there is extensive damage to the cartilage. If there are no reactive symptoms and the swelling diminishes in size, the prognosis is favorable. Violent inflammatory symptoms, on the other hand, necessitate opening the tumor, thus rendering the prognosis more unfavorable. In some cases there is recovery without visible deformity, while in others recovery occurs with great shrinkage or other deformity of the cartilage.

Treatment.—The treatment should be symptomatic and modified to correspond with the peculiar pathology of the case. If, for example, the othematoma is due to degenerative changes in the bloodvessels and the connective tissue or the cartilage of the auricle, it would be wrong to apply massage to promote absorption, as such manipulation would probably provoke more hemorrhage. Such a procedure, if tried at all, should be deferred until regeneration has closed the interior wounds. Pressure bandages are also contraindicated for the same reason. The application of ice-bags or a Leiter coil may exert a favorable influence in preventing passive inflammatory swelling; and if it is already present, the cold reduces it somewhat. The application of heat is better treatment, as it promotes regeneration. Cooling lotions locally and cathartics may also be tried with some advantage. The inflammatory type should be incised and antiseptic dressing applied.

Politzer recommends the puncture of the tumor in the early stage of its development. If this is not followed by relief, it is better to open it thoroughly by free incision, after which the contents are removed and the cavity packed with iodoform gauze.

Angioma. Symptoms.—The bright red or lurid patches which are not elevated above the surface of the skin are not included in this group of tumors. The term "angioma," as used here, refers to the cavernous tumors, which are bluish red in color and are made up of a series of venous sinuses or cavities of various sizes and shapes. They are often separated from each other by perforated fibrous septa, which afford free intercommunication of their blood contents.

They may appear in the auricle, in the meatus, or in both. They may be either primary or secondary extensions from adjacent structures. They vary in size but rarely grow larger than a small hen's egg. They are irregular in shape. Pulsation is occasionally present. Angioma is sometimes congenital, while in other cases it develops after trauma or after the gradual dilatation of the bloodvessels of the simple angioma, the bright red or lurid patches referred to in the preceding paragraph. Cases are on record of angiomata which appeared after the auricle had been frozen.

The presence of pain depends chiefly upon the rapidity with which they grow. If of rapid development and large size, the pain is consider-

able. Troublesome pulsation is another characteristic of angioma of rapid growth.

Deafness is present in those cases in which the meatus is occluded. Reflex cough may also be present when the meatus is involved.

Diagnosis.—Othematoma is the only tumor which might be confounded with cavernous angioma. The former is of rapid growth, smooth in outline, and opaque by transmitted light; whereas angioma usually develops more slowly, is irregular in outline, and is transparent by transmitted light.

Treatment.—The treatment should be addressed to the reduction of the blood contents of the tumor, which interfere with its circulation This may be accomplished in several ways. Electrolysis is, perhaps, the best method in growths of small or medium size. The needles connected with the positive pole of the battery should be thrust through the growth, while the negative (sponge electrode) pole is placed on some remote portion of the body. The positive pole liberates oxygen and acids, which coagulate the blood and soft tissues, thus contracting and obstructing the cavernous sinuses of the tumor. Should the negative pole be applied as recommended by Hovell, the results would be less certain, as the negative pole liberates hydrogen gas, which tends to liquefy the solid tissues. The negative pole is better adapted to use in fibrous tumors, on account of its liquefying properties.

Multiple puncture of the surface with needle points and brushing the surface with nitric acid has been recommended in small growths. Both measures produce scar tissue, and thus cause contraction.

Politzer recommends the passage of several silk sutures through the tumor. He first renders them aseptic and then saturates them in a solution of the perchloride of iron. The iron coagulates the blood and the threads act as nuclei for the clot formations.

The Paquelin cautery has been used in larger growths. Such treatment is necessarily limited to exceptional cases.

Injections of styptic remedies, as carbolglycerin, iodine, and the perchloride of iron, are not safe procedures, as they may cause extensive sloughing and subsequent disfigurement from cicatricial contraction. Suppuration and perichondritis may also follow the injections, the auricle becoming shrivelled and reduced in size.

Fibroma.—*Fibroma* of the external ear consists of spindle cells and connective tissue. It is usually the result of local irritation, as from the wearing of ear-rings, and is often found in negresses, who are peculiarly subject to fibromata, not alone in the external ear, but in other parts of the body. They vary in size up to that of a large walnut, are rounded in form, and may be pedunculated or sessile. They are usually located in the lobule, as this is the portion in which ear-rings are worn. They may appear elsewhere on the auricle or even at the entrance to the auditory canal.

Treatment.—A small V-shaped incision, including the growth, may be made, and the cut surfaces brought together by skin stitches, thus causing very little disfigurement. If the growth is pedunculated it

is easily removed with scissors, and the base cauterized and dressed antiseptically. Large growths may be removed by excision, the parts being brought together as well as possible to avoid disfigurement. If necessary, a subsequent plastic operation may be performed to overcome the deformity.

Cysts.—Like cyst formations in other parts of the body, those of the ear are the result of the plastic union of parts which are normally open or separated, *i. e.*, the sebaceous glands of the auricle may become infected, their orifices closed, and the secretions retained in the dilated and inflamed glandular sacs. They are variable in size, are soft, and may remain stationary in their development for several years.

Treatment.—The treatment of cysts of the auricle consists in a free incision into the tumor, the evacuation of its contents, curettement, and the application of the tincture of iodine to the surface of the cavity. A suitable surgical dressing should then be applied, and repeated daily while repair is taking place.

Epithelioma.—The growth begins as a hard nodule, situated in the skin or the subcutaneous connective tissue; it grows slowly for a time, but later develops quite rapidly. It is in this stage that ulceration is likely to occur. The growth may be an extension from contiguous structures, or it may be primary in the auricle or the meatus. Of the sixty cases reported, nearly all occurred in patients more than forty years of age. Dr. J. S. Brown reports a case in a man, aged seventy-eight years. Epithelioma may begin as warty or fissured surfaces, which finally ulcerate and continue to spread by the formation of new tissue at the edge of the ulcer. This tissue rapidly undergoes disintegration, and the ulcerous process may spread until the entire auricle and meatus or even the neighboring structures are destroyed.

The nodular enlargements on the auricle may be present several months before enlargement of the glands in the neck appears. Pain may not be a symptom until ulceration takes place; hence, in the early stage, epithelioma may be mistaken for fibroma. As the ulceration and the deeper extension of the growth progress, the pain increases, often becoming excruciating in character. The facial nerve may become involved, and facial paralysis develop. The auditory nerve may be affected, or hemorrhages may occur, and glandular enlargements develop, which may result fatally. Death may be due to septicemia, exhaustion, meningitis, thrombosis of the lateral sinus, or cerebral abscess.

Treatment.—The treatment of epithelioma here, as elsewhere in the body, consists in the complete removal of the growth by excision. To accomplish this, it may be necessary to remove the auricle in part or entirely. The resulting disfigurement may be corrected by a subsequent plastic operation or the adjustment of an artificial auricle. While the wound is healing, a vulcanized or a silver tube should be worn in the meatus to prevent cicatricial contraction.

Sarcoma.—Sarcoma of the auricle is rare. When present, it may be of the round-cell variety, which develops rapidly and leads to an

early fatal issue, or it may be of the fibrosarcomatous type, which grows slowly. This type may exist for many years without giving rise to marked symptoms. The round-cell variety is painful, as its rapid growth stretches the sensory nerves, and it is also often attended with inflammation in the parotid and the mastoid regions.

The appearance of the tumor varies according to the variety and the rapidity of development. If it is of the fibrosarcoma type, it is smooth and covered with normal skin. If it is of the round-cell variety, the rapid growth causes the skin to become eroded and the seat of fungous granulations. The eroded surface secretes an unsightly suppurating material composed of debris, pus, epithelium, leukocytes, and blood corpuscles. The ulcerating surface often bleeds profusely.

The external meatus may be the seat of round-cell sarcoma and, in extremely rare instances, of osteosarcoma.

Diagnosis.—A portion of the growth should be subjected to microscopic examination. The round-cell sarcoma is pale on cross-section, and exudes a milky juice; it is composed almost entirely of round cells and thin-walled bloodvessels. The fibrosarcoma has a considerable quantity of intercellular cement substance, and the macroscopic appearance of the tumor is coarse-grained and firm.

Prognosis.—It is obvious that this will depend upon the type of the growth, the round-cell variety being comparatively more speedy and destructive. In this type, death may result from intracranial extension, hemorrhage, or exhaustion.

Treatment.—Early and complete removal of the growth is the best treatment. This may be done with the knife or the galvanocautery. If the growth cannot be completely removed, the parts continue to discharge offensive material.

The Röntgen-rays have been used with some apparent success in superficial sarcomata, but we are not ready to recommend this method of treatment until further trial has demonstrated its real value. It is unsafe to try it in the round-cell variety, as the early surgical removal offers the only hope in this type of sarcoma. While using the Röntgen-ray treatment, extensions may occur, thereby rendering operative treatment hopeless. The rays are of special value, however, after operation, as recurrences are less frequent or are delayed by their use.

CHAPTER XXXVII

DISEASES OF THE AURICLE AND EXTERNAL MEATUS

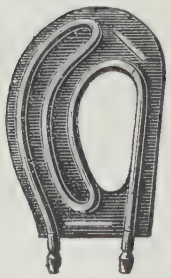
PERICHONDRITIS OF THE AURICLE

This is a rare affection and resembles othematoma. The upper portion of the auricle is usually involved, as the cartilage is chiefly found there. The lobule escapes, as it is free from cartilage.

Symptoms.—If the inflammation occurs as a complication of furunculosis of the meatus, the pain characteristic of that condition is present; whereas, if it begins in the auricle, the first symptom may be circumscribed redness and swelling, which gradually spreads and becomes more severe, until it finally involves the whole of the cartilaginous portion, including the concha, or it may include the meatus. If the meatus is wholly occluded by the swelling, the hearing is impaired. Fluctuation soon occurs, and is due to the inflammatory exudate of viscid serum beneath the perichondrium. The natural contour of the auricle is modified by the swollen tissue, and its surface is reddened. The perichondrium of the entire auricle may become detached and thus interfere with the nutrition of the cartilage. This is a serious complication, especially if the secretion becomes purulent. Under such circumstances the cartilaginous auricle is apt to shrink or slough, and leave marked deformity.

The greatest care should be exercised to prevent additional infection when there is an abrasion of the skin and when an incision is made to evacuate the fluid beneath the perichondrium. Should active infection be present, many weeks or months may be required to check the progress of the disease, and even then the auricle will be greatly deformed. Perichondritis occasionally follows the mastoid operation, especially when the plastic meatal flap includes the concha of the auricle.

Fig. 391



Leiter's coil.

The deformity following perichondritis may be so slight as to attract no attention, or it may be so marked as to disguise completely the anatomical characteristics of the auricle.

Treatment.—The early treatment should be antiphlogistic in nature, heat being the best agent. The Leiter coil (Fig. 391) should be applied over the auricle and hot water passed through it. A hot-water bag may also be used. A saline cathartic should be administered

and leeches used around the auricle in conjunction with the heat. If fluctuation is present, an incision should be made to evacuate the fluid. The auricle should be cleansed before making the incision, to prevent the possibility of additional infection. The cavity should be carefully but thoroughly scraped with a dull curette, and then cleansed with an antiseptic solution. If the infection is severe and granulations are present, the cavity should be swabbed with the tincture of iodine or the compound tincture of benzoin. Free drainage should be maintained by the insertion of a gauze wick, over which the usual dressing of gauze pads should be placed and held in position with a bandage. The dressings should be changed every twelve hours.

Subsequent operative measures may be undertaken to correct the deformity if it is sufficient to produce disfigurement.

HERPES OF THE AURICLE

The etiology is not always clear, although herpes is apparently caused by middle-ear disease. It is thought by some to be caused by malaria, and by others to be a neurosis. It is most common in adults.

Symptoms.—The vesicular eruption is sometimes preceded by a stinging or burning pain, especially if the meatus is involved. The eruption is generally on the outer or concave surface of the auricle, which is supplied by the auriculotemporal branch of the fifth nerve. This is of interest, as the distribution of the eruption usually follows the terminal branches of this nerve. It is more rarely on the posterior or convex surface of the auricle, as the auriculotemporal branch of the fifth nerve does not extend to this region.

The course and appearance of the eruption is about as follows:

At first there is a reddened area, which becomes papular, then vesicular. The vesicles may become confluent and form bullæ. The vesicles at first contain clear serum, which later becomes cloudy and purulent. The duration of the vesicular stage is limited to a few days, after which the vesicles dry up, leaving crusts and an occasional superficial ulcer.

If the meatus becomes involved, more or less deafness and tinnitus is present.

Treatment.—Tonics, purgatives, and outdoor exercise are indicated to improve the general health of the patient. Cool or cold morning baths, or at least sponging of the neck and chest, are indicated to improve the tone of the vasomotor nervous system.

The blisters should be protected by starch or boric powder and cotton-wool dressings. The fluid contents of the vesicles should be emptied, care being taken to avoid removing the elevated dermis, and exposing the underlying parts to the air. This accident is attended with considerable pain. Boric acid powder may be applied in suppurative cases. If the meatus is involved, boric acid should be blown into it.

HERPES ZOSTER OF THE AURICLE

This is a vesicular eruption which appears on a reddened surface, although the area of redness does not extend much beyond the base of the blisters. The vesicles are arranged in groups and are quite painful.

They most often appear upon the posterior surface of the auricle and the lobule, and more rarely upon the anterior or superior surface of the meatus. They still more rarely develop upon the anterior surface of the auricle.

It is an affection of either the trigeminus or the great auricular nerve. In some cases it seems to be of ganglionic origin.

The location of the eruption is determined by the distribution of the affected nerve.

In rare instances the drumhead is involved, although the hearing may be but slightly affected thereby. Within a few days after the formation of the vesicles they burst, emptying their contents, after which crusts form at the site of the eruption.

A few days later new epidermis forms, and unless there is a recurrence of the disease, complete recovery takes place.

Treatment.—Although herpes has been recognized as a distinct disease for a long time, the treatment of it has not developed beyond an attempt to relieve pain and to prevent excoriations after the bursting of the vesicles. The internal administration of arsenic is often recommended with the idea of correcting the nervous disorder which is the chief cause of the trouble. It is doubtful, however, if it has any specific effect as a remedy. Anodyne remedies, such as the 5 per cent. ointment of the hydrochlorate of cocaine, may be applied locally with a fair degree of confidence that it will afford relief. Calomel dusted over the eruptions, especially after they have discharged their contents, induces healthy and speedy epidermization of the denuded surfaces.

DERMATITIS OF THE AURICLE

Dermatitis may be due to traumatism, exposure to heat or cold, and to a parasitic infection (Politzer). The treatment should consist of the application of solutions of lead.

It occasionally happens that when there is an abrasion of the skin of the auricle or a loss of the integrity of the epidermis due to eczema, etc., erysipelatous infection may occur and lead to a much more severe type of inflammation.

Treatment.—The treatment should be antiphlogistic, and weak solutions of ichthyol (1 to 5 per cent.) should be applied locally.

Should the deeper tissues become involved and pus accumulate therein, free incision should be made and the parts treated according to aseptic surgical principles.

Dermatitis from Exposure to Cold.—**Symptoms.**—Frostbite; chilblain; dermatitis congelationis auricula.

Etiology.—Exposure to extreme cold or prolonged exposure to moderate temperature, as in the autumn of northern latitudes, also the extreme thinness of the skin and slight amount of subcutaneous tissue separating it from the cartilage of the auricle, predisposes to dermatitis.

The disease is characterized by the formation of nodules and excoriations, especially on the elevated portions of the auricle.

In the extreme north the dermatitis is usually acute in character, and is attended with simultaneous freezing of the nose. More or less necrosis and gangrene, and partial loss of the auricle follows.

The affection is most common in young chlorotic girls of northern climates, and always appears at the beginning of cold weather. It is more than probable that insufficient and improper food predisposes to its occurrence. These conditions, together with the unstable vasomotor system at the age of puberty, may be considered the chief etiological factors.

Symptoms.—Ordinary frostbite is characterized by moderate swelling, redness, and circumscribed dermatitis.

The nodules heal slowly or not at all, and become covered by bloody crusts. Even after the crusts disappear, the skin continues to exfoliate epidermis for a long time. In addition to these symptoms, which are apparent to the eye, there are lancinating pains, sense of heat, itching, etc., which cause the patient to scratch or rub the parts, thereby increasing the inflammation.

Treatment.—In those cases which are due to extreme cold, snow or ice-bags should be applied. In the subacute varieties, Goulard's extract is serviceable. The auricle may be painted with iodine collodion, or camphor ointments. For the relief of the intolerable itching the following mixture is of value:

℞.—Collodion . . . . . ʒj
Ol. ricini . . . . . ♏xx
Ol. terebinth. . . . . ʒj—M.
Sig.—Apply locally to relieve itching.

The frequent application of camphor ointment will relieve the itching.

FURUNCULOSIS OF THE EXTERNAL MEATUS

Synonyms.—Follicular inflammation of the external auditory canal; otitis externa; follicularis s. circumscripta.

Etiology.—Furunculosis of the external auditory canal is a circumscribed inflammation involving either the hair follicles or the sudoriferous glands. As these organs are limited to the cartilaginous or external portion of the canal, the furuncles are not found in the deeper or osseous portion. The boils may occur without known cause, or they may be a part of a general furunculosis. They may occur in the course

of suppurative otitis media and chronic eczema. Traumatism from attempts at cleaning the ears often causes them. Furunculosis most often appears in the spring and autumn, and is chiefly a disease of adult life, though I have seen cases in infants. General debilitating diseases or their sequelæ predispose to it.

Symptoms.—The hearing is but slightly affected in most cases, as the lumen of the canal is not completely obstructed. The pain is more or less intense, according to the depth of the inflammatory process. The furuncle does not always present the appearance of a boil, as the skin is tense and closely adherent to the cartilaginous meatus, thus preventing the usual elevated appearance.

The auricle is extremely sensitive to the touch, and the movements of the inferior maxilla in mastication cause pain. The tension of the skin becomes so great that the patient is often unable to sleep. The swelling in the external meatus is more or less diffused on account of the close adhesion of the skin to the cartilaginous meatus, and with the inexperienced may be mistaken for the redness and swelling in the postsuperior portion of the meatus in mastoid inflammation. It is easily differentiated from it, however, by remembering that the swelling due to mastoid disease is limited to the postsuperior wall of the osseous or deeper portion of the meatus, while that due to furunculosis is in the posterior and inferior wall of the outer or cartilaginous portion. The pain is often greater in furunculosis. In infants the differentiation is more difficult, as the meatus is very shallow and the swelling is near the membrana tympani, which it may obscure.

The temperature is irregularly elevated during the first few days. Deafness and tinnitus are present if the meatus is occluded, though they may be present without occlusion. When this is the case the inflammation has probably extended to the drumhead and the tympanum.

The more superficial the furuncle, the greater the redness and the more circumscribed its area. Pain may or may not be present. If the deep tissues are involved the redness and swelling are more diffused, while the pain is greater. In some cases the surrounding tissues become more or less swollen, as, for instance, when the anterior portion of the meatus is involved, the skin in front of the tragus is swollen and purple in color; whereas if the posterior portion is involved, the mastoid skin may be swollen and simulate mastoiditis. Glandular enlargement in the lateral region of the neck is not commonly present.

Course.—Furunculosis of the meatus is likely to go on to suppuration, which usually takes place in from six to eight days. The deeper the inflammation the more delayed the voluntary escape of pus. The pain and swelling subside immediately after the pus is liberated, especially if it is done by incision. Incision should be made early, as the progress of the disease is often thereby checked. The meatus should then be irrigated with warm boric acid solution, thoroughly dried and dusted with bismuth, and a gauze wick inserted for drainage. The dressing should be changed daily until the swelling and discharge have materially subsided. If the boil is allowed to rupture spontaneously granulations

may spring from its crater, and be mistaken for middle-ear polypus. Recurrences are to be expected in many cases.

Treatment.—Abortive treatment may be used before the formation of pus has taken place. The best remedy is a 12 per cent. solution of carbolic acid in glycerin. This should be instilled into the meatus, or applied with a cotton-wound applicator if the canal is open. Its early use is often followed by a complete disappearance of the process. The Leiter ice coil gives relief to the pain. Mixtures containing opium, morphine, cocaine, etc., are recommended, the carbol-glycerin mixture, however, is not only curative, but analgesic as well. Poultices have been recommended, but their use is irrational and obsolete. Antiseptic solutions are valuable adjuncts in the treatment of furunculosis, and the carbol-glycerin solution answers this purpose admirably, in addition to its anodyne and curative properties. Should it fail to give the desired relief, the meatus is at least prepared for operative measures.

In a large majority of cases the process has gone on to the suppurative stage before the physician is called in. When pus is present, the furuncuulous area should be freely incised with a narrow bistoury. Pus may not appear at once, but this should not deter the physician from incising each swollen and reddened area. If voluntary rupture has occurred and the flow of pus is obstructed by granulations, the area should be opened more freely.

After-treatment.—Immediately after incision, the exposed cavities should be cleansed with a 5 per cent. solution of carbolic acid to check the growth of the pus cocci. Frequent instillations of the peroxide of hydrogen should be used to keep the wound and the meatus free of pus.

The ceruminous secretion is often absent after an attack of furunculosis, or, if present, is of a dry, crumbling quality. Intolerable itching usually complicates furunculosis.

Various remedies for the relief of the itching have been recommended. The white precipitate ointment, boric acid 5 per cent. in lanolin, and the glycerin-carbolic acid solution are valuable for this purpose.

The entrance of plain water into the meatus often leads to a relapse, hence care should be exercised to prevent it.

DIFFUSED INFLAMMATION OF THE EXTERNAL MEATUS

Synonym.—Otitis externa diffusa.

Etiology.—The causes are (*a*) infections from without or from within the middle ear; (*b*) traumatism; (*c*) excoriation of the cutis of the meatus; and (*d*) the injection of irritating fluids into the meatus.

Symptoms.—Unlike the furuncuulous type, the symptoms are chiefly limited to the osseous meatus and the drumhead. The cutis is swollen and congested, and after a few days throws off a serogelatinous secretion, which is often so tenacious that it can be removed *en masse* (Politzer). It is charged with pathogenic organisms, thus showing its bacteriological origin.

Great *pain* in the region of the ear is usually present, and movements of the inferior maxilla aggravate it. Tinnitus and dizziness are occasionally present. The hearing may be impaired, especially if the drumhead is much swollen, or if there is a large accumulation of thick secretion.

The *duration* of the disease is three or more days. If it runs an uninterrupted course, an acute case may terminate on the third day. The hearing is usually normal after the inflammation ceases. In rare cases an excoriated or ulcerous surface is left, and becomes the seat of a granulation tumor, which, when removed, checks further secretion of pus.

Periostitis and *hyperostosis* may remain as sequelæ in rare cases.

Prognosis.—In the simple forms, complete recovery usually occurs, while in those cases which are complicated by excoriations, constriction of the meatus from periostitis, hyperostosis, and dermoid thickening are likely to affect the function of the ear.

Treatment.—It should be borne in mind that the disease is usually of bacteriological origin, and remedies should be applied accordingly. The carbol-glycerin mixture (12 per cent.) is, perhaps, one of the most reliable remedies. It should be instilled into the meatus two to three times daily and cotton-wool introduced into the cartilaginous canal. The Leiter coil, and leeches to the tragus and the mastoid region are of great value when there is swelling and pain. Antiseptic solutions of all kinds have been recommended, but it is doubtful if any of them are of special value. It may be said of aqueous solutions in general that their utility is questionable. Remove the secretions from the meatus with the peroxide of hydrogen and a cotton-wound applicator and then apply carbol-glycerin mixture.

If ulcers form and show no tendency to heal, they should be cauterized with a 90 per cent. solution of the nitrate of silver.

HEMORRHAGIC INFLAMMATION OF THE MEATUS

Synonym.—Otitis externa hæmorrhagica.

This is a form of hemorrhage beneath the superficial layer of the skin of the osseous meatus, and in most cases is probably a complication of influenza otitis media. The hemorrhagic areas appear as bluish swellings on the inferior or the posterior wall of the meatus. To the probe they are soft and often rupture upon very slight pressure. The vesicles may remain for several days, and when they disappear others may come to take their place. In from one to two weeks they disappear altogether, and complete recovery takes place. The hearing, if affected, returns to normal.

Treatment.—The hemorrhagic vesicles should be opened with a probe and gauze drainage applied to the meatus. The dressing should be removed daily. Politzer recommends dusting the meatus with boric acid powder in addition to the gauze drainage.

CROUPOUS INFLAMMATION OF THE MEATUS

Synonym.—Otitis externa crouposa.

This is a very rare condition, and usually occurs together with influenza otitis media or furunculosis of the meatus. The false membrane is limited to the osseous portion of the meatus and to the outer surface of the drumhead, and in this is similar to the diffuse inflammation of the meatus. It sometimes appears with a similar process on the tonsils (Gottstein). The membrane forms in from one to two days and is firmly attached; it may, however, be removed by forcible syringing. It may form a cast of the osseous meatus and the drumhead. The microscope shows it to be composed of a fibrous network infiltrated with round cells, nuclei, epithelium, Staphylococcus pyocyaneus, and Streptococcus pyogenes (Politzer).

The formation of the membrane is attended with some pain which is relieved when it is cast off. Recurrences are common.

Prognosis.—The prognosis is favorable. In rare cases the cartilage of the meatus becomes necrotic or gangrenous.

Treatment.—The treatment consists in removing the false membrane with forceps or by antiseptic solutions applied with a syringe, and drying the meatus and dusting it with an antiseptic powder.

EXOSTOSIS AND HYPEROSTOSIS OF THE MEATUS

These two terms are often used synonymously, although, according to strict pathological interpretation, they should be used to describe different lesions of the bony tissue.

An exostosis is a bony tumor growing from the wall of the meatus, which may be either sessile or pedunculated. Hyperostosis is a diffuse thickening of the bony tissue, or a true hyperplasia.

Etiology.—The cause of these pathological changes is often unknown, but in many instances they are due to conditions which may be easily recognized. Among them may be mentioned.

(*a*) Traumatic fracture of the walls of the meatus, whereby a circumscribed periostitis is excited, which finally results in the formation of a bony mass or tumor.

(*b*) They may be due to developmental causes, particularly in those cases wherein the middle and the inner section of the osseous meatus on each side is the seat of the growth. When due to faulty development, the growths are usually small. They may be either sessile or pedunculated.

(*c*) Chronic suppuration of the middle ear may excite a secondary inflammation of the membranous canal, and cause a fibrous or connective-tissue thickening, which, after a long period of time, may be metamorphosed into osseous tissue

(*d*) There are some cases in which heredity seems to be a factor in the production of the growths, as the same condition may be traced through a few generations.

(*e*) Syphilis is undoubtedly a cause of the growths, although not in a very large number of cases.

(*f*) Gout has been thought to be another cause, but it is doubtful if this condition leads directly to their formation.

It is more probable that the gouty diathesis causes an inflammatory process of the skin and the periosteum, which finally undergoes a retrograde change and becomes the seat of lime deposit.

Symptoms.—The symptoms are chiefly those recognizable by the aid of the eye and the probe, although in some cases in which the lumen of the ear is completely occluded, the function of hearing may be affected. If the growth is an exostosis, it appears as a rounded, elevated mass, with a tense, whitish covering of skin. The lumen of the meatus is reduced to a crescentic or slit-like opening. The swelling or growth is composed of very dense tissue. If it is sessile, it will be difficult to differentiate between it and a hyperostosis; but if it is pedunculated, the differential diagnosis may be more easily made, as this type of growth is more often an exostosis. A favorite seat for the growths is at the junction of the osseous and the cartilaginous portions of the meatus. They may, however, form in any portion of the canal. Deafness may be present, although it is not marked, unless there is concurrent disease of the middle ear or the labyrinth, except in those cases in which the growth completely obstructs the lumen of the canal. Secondary inflammation of the cutaneous meatus may be caused by the pressure of the growth against the opposing walls. In such cases there will be more or less secretion from the dermatitis thus excited. Cases have been reported in which the pressure of the growth was so great that necrosis of the surrounding bone tissue resulted, thereby complicating the case.

Treatment.—The treatment is necessarily limited chiefly to surgical procedures, except for the relief of those symptoms which are due to secondary inflammatory processes. If the growth is large enough to interfere in any way with the function of audition, it should be removed. In some cases this can be done through the external auditory meatus without lifting the auricle forward, as is done in the mastoid operation. The skin and periosteum over the growth should be excised and elevated, and the bony mass removed or reduced with a small chisel or gouge or with a trephine. If the growth is sessile or diffused, and involves the entire length of one wall of the meatus, it would, perhaps, be futile to attempt to remove it through the external auditory meatus. A better and much simpler procedure would be first to lift the auricle forward, as in the mastoid operation, thus exposing the entire canal to view and affording easy access with instruments. When this is done, the skin of the osseous portion of the meatus should be carefully elevated with a small periosteum elevator, so that the healing process may be more certain and rapid after the operation. The exposed tumor should then be removed with a very sharp gouge, or, perhaps better still, by the use of a

trephine. This method of procedure is also productive of better results in many of the pedunculated growths, as the base can thus be completely removed.

The *indications* for operative interference should be based upon the amount of deafness present and upon the concurrent middle-ear disease, if present. If, for example, there is chronic suppurative ear disease, with impairment of hearing, it is quite essential to the proper treatment of the case that the external auditory meatus be completely freed from the obstructive lesion, so as to afford better drainage and opportunities for treatment of the middle-ear cavity.

Another indication is the presence of dermatitis with secretions, while a still more urgent indication is secondary pressure necrosis of the contiguous tissue.

It seems irrational, in view of the present status of surgery, to resort to the use of laminaria tents for the dilatation of the canal, as the process must necessarily be a long and painful one. This method was formerly in vogue and is still recommended in some of the modern text-books on otology.

STRICTURE OF THE EXTERNAL MEATUS

Etiology.—Obstructive lesions of the external auditory canal are due to the inflammatory swelling of the skin which lines its walls, as described under dermatitis, furunculosis, perichondritis, eczema, etc. It may also be due to newgrowths, exostosis, and fibrous thickening of the deeper dermal tissue. It is to the last-named condition that permanent obstruction of the lumen of the canal is usually due.

Cicatricial rings or bands are produced by prolonged inflammation of the meatus in the course of chronic otorrhea. In rare instances they are due to syphilis, diphtheria, etc., or to the use of the cautery and acids in the meatus. Partial closure of the canal sometimes follows the mastoid operation, especially if the plastic meatal skin flap is not properly sutured and the wound is tightly packed with gauze. (See Mastoid Operation.) In the aged the cartilage which supports the skin of the meatus undergoes atrophic changes, which allows the walls to collapse and obstruct the meatus.

In some cases the obstructive lesion is ring-like, while in others it may be limited to one wall of the meatus. If it is due to an exostosis, there is a tumefaction on one side of the canal. The tumor is hard to the touch of the probe, and may either partially or wholly obstruct the meatus. Exostosis sometimes follows the exfoliation of necrosed bone, while in other cases it develops from the periosteum or from the bone beneath, as true hyperostosis.

Treatment.—As the origin of the obstruction is various, so should the treatment be varied. If inflammatory, suitable treatment should be instituted. If it is cicatricial in character, laminaria tents and the subsequent introduction of hard rubber tubes may be used. In this way the stricture is dilated and maintained in this condition by the rubber

tubes. Electrolysis may also be used with advantage; from five to six sittings are required to reduce the fibrous constriction. The needles connected with the negative pole of the galvanic battery should be inserted into the fibrous ring, while a large sponge electrode connected with the positive pole should be placed in contact with the body. The amount of current necessary to soften the tissue varies from 25 to 50 ma., and each seance should last from five to twenty minutes, according to the amount and density of the fibrous tissue.

Another method of treating fibrous strictures is to split the canal longitudinally in several parallel lines and introduce sponge tents.

After thorough dilatation the hard rubber tubes should be used to maintain the patency of the meatus.

Jansen resorts to a surgical procedure, which is probably the most successful mode of treatment, whether the stenosis is cicatricial or osseous in character. He detaches the auricle as in the mastoid operation, and then dissects away the fibrous ring, osteoma, or hyperostosis. To cover the bony wound, he makes a pedunculated flap from the skin over the mastoid process and inserts it through the line of incision made in detaching the auricle.

Should the stricture be of long standing and accompanied by suppuration of the middle ear, a radical mastoid operation should be done, during which the canal may be enlarged.

MYCOSIS OF THE EXTERNAL MEATUS

Synonyms.—Parasitic inflammation of the external auditory canal; otomycosis.

Etiology.—The source of the mycotic infection is often unknown. Living in damp surroundings or in the presence of yeast spores seems to favor it; hence, it is rather common among bakers. The habit of instilling warm oil into the ears to relieve earache favors the growth of the spores, as the oil is a good soil for their development. The spores which most commonly cause the disease are the Aspergillus niger, flavus, and fumigatus. Several other varieties are occasionally found.

It usually occurs in adults, and rarely in children or in the old. As the sanitary and hygienic conditions surrounding the poor are bad, it is common among them. The fungus growth may, in rare cases, extend to the middle-ear cavity or even to the mastoid cells.

Symptoms.—The manifestations of the infection depend largely upon whether the spores have attacked only the epidermis or also the deeper living structures of the skin or the drumhead. If only the epidermis is affected, there may be no symptoms, even when the drumhead is covered with the false membrane; on the contrary, if the true skin is involved, deafness and tinnitus are more or less severe as a result of the swelling and inflammation which has been excited. This type of inflammation is known as *otitis externa parasitica*, and is characterized by shooting pains, itching, tinnitus, and deafness.

The appearance of the mycotic membrane is black or grayish in color, velvety in texture, and distributed chiefly over the osseous portion of the canal, although the drumhead and the cartilaginous portion of the canal may also be covered by it. It can be removed by syringing. The underlying skin is red, slightly swollen, and largely denuded of epidermis.

The *course* of this type of inflammation, if not properly treated, may extend over several weeks. Under treatment its duration may be much shortened.

The pains and other subjective symptoms are usually greatly relieved immediately after the removal of the membrane.

Treatment.—Almost the entire list of antiseptic mixtures and powders have been used for the relief of this disease, but the remedy *par excellence* is alcohol, which should be instilled into the meatus once or twice daily; two to four days are usually sufficient time to effect a cure. The alcohol should be used at intervals of every two weeks for a few months to prevent a recurrence.

ACUTE ECZEMA OF THE EXTERNAL EAR

The superficial layers of skin are involved, and, in the beginning, there is marked redness and swelling of the skin; nests or colonies of fluid-filled vesicles soon make their appearance.

Etiology.—It is not always possible to ascribe a cause for the eruption, although it is usually due to one or more of the following factors: viz., neurosis, scrofula, rickets, discharge of pus from the middle ear, irritating remedies, cold douches, and exposure to heat. Other causes exist in selected cases. It may be a primary affection or it may be secondary to a similar process on some other part of the body.

Symptoms.—The onset of the disease is characterized by burning and itching, which is soon followed by pain. Deafness and tinnitus are present in those cases in which the meatus and the drumhead are involved, especially when the exfoliated epidermis and secretions obstruct the lumen of the canal. If the disease is limited to the auricle, the hearing is not affected. There is some elevation of the temperature, especially in children. The pain and the pyrexia give rise to restlessness and inability to sleep.

The disease may terminate in one of three ways, namely: (*a*) In the mild form the vesicles dry up and the epidermis peels off on the second or third day, leaving the natural cuticle. (*b*) In a large number of cases the blisters discharge their contents, and after a few days the surface becomes covered with yellow crusts. In time these disappear and recovery takes place. (*c*) The third and most disagreeable mode of termination is the persistence of serous or purulent secretion for several weeks, after which the parts become covered with epidermis.

In some cases the eczema may persist in isolated areas for many weeks and leave more or less scar tissue and contraction, or it persists and becomes typically chronic in character.

The *treatment* will be considered under Chronic Eczema.

CHRONIC ECZEMA OF THE EXTERNAL EAR

Symptoms.—Owing to the involvement of the deeper structures of the skin, there is greater thickening and rigidity of the auricle than in the acute type. The crusts usually form in the hollows of the auricle and in the posterior groove, while beneath them is secreted a serous or purulent matter. The meatus may be obstructed by the thickening of its integument. The whole auricle, and in some cases the meatus, is the seat of a desquamative process. The process of desquamation and crust formation varies in different cases, although the desquamation is usually predominant.

Exclusive of the appearance of the skin, the itching is the most severe symptom. The patient is overcome with an irresistible desire to rub or scratch the parts, and thus produce deeper lesions of the skin.

Tinnitus and deafness may result from desquamative plugs in the meatus and from secondary hyperemia of the mucous membrane of the middle ear. It is barely possible that in rare cases hyperemia of the labyrinth may be induced.

The course of chronic eczema is quite different in individual cases, some are cured under treatment in a few weeks, while others obstinately persist under any form of treatment. Boils in the meatus may complicate the condition.

Treatment.—The general treatment should be addressed to the correction of constitutional dyscrasias and neuropathic states, which so often underlie the condition. Iron, arsenic, strychnine, iodine, and the bitter tonics should be given in suitable combination for this purpose. The administration of saline cathartics and an occasional dose of calomel will often aid in overcoming the eczema.

Perhaps one of the best measures for its relief is negative in character, namely, the avoidance of the local application of water, which greatly aggravates the eczema. If it is desirable to use water for toilet purposes, the patient should be instructed to add boric acid or a teaspoonful of common table salt to the quart of water. The irritating qualities of the water are thus reduced.

The local treatment is somewhat different in the acute and the chronic forms, hence they will be considered separately.

Local Treatment of Acute or Subacute Eczema.—The remarks concerning the avoidance of plain water are especially applicable to this type of eczema. If proper care is exercised, some cases will be cured with no local or constitutional treatment whatever. Others will persist in spite of any mode of treatment, and gradually pass into the chronic form. A soothing ointment composed of one dram of the oxide of zinc to the ounce of lanolin or vaseline is very sedative, especially if the disease is due to an irritating discharge from the middle ear. The addition of one grain of the acetate of morphine will increase the sedative action of the ointment. Calomel dusted on the excoriated or fissured surfaces acts well in some cases. Lotions of liquor plumbi subacetatis and resorcin are indicated

when there are large vesicated surfaces. As their application excites pain, the parts should previously be painted with a 5 per cent. solution of cocaine. Ichthyol in aqueous solution (2 to 50 per cent.) has proved a valuable remedy. The parts should be painted once or twice daily. Cotton pads may be applied over the painted surface to prolong the therapeutic effect of the remedy and protect the diseased area from the air.

When the case is in the crust-forming stage proceed as follows:

(*a*) Remove the crusts by first softening them for twenty-four or forty-eight hours by local applications of oil, vaseline, lanolin, balsam of Peru, or a 10 per cent. solution of Burow's mixture. If the oily preparations are used, cotton should be saturated with them and applied over the scabs, and protected by another pad of gauze lightly held in position by a bandage. If Burow's mixture is used, the pads of cotton saturated with it should be covered with oiled silk or rubber cloth to prevent evaporation. Change every two hours.

(*b*) At the end of twenty-four hours, the crusts may be removed with a probe or forceps. Great care should be exercised to avoid inflicting injury to the underlying surface, as to do so causes a larger crust to form.

(*c*) The parts are now ready for the medicated ointments referred to above. They should be changed every day. The parts should be carefully cleansed each time by wiping them with cotton pads, water being carefully avoided. If the crust formation is obstinate, the parts should be painted with a 1 to 3 per cent. solution of the nitrate of silver before reapplying the salve.

(*d*) When epidermization is established, the new skin should be protected from mechanical or chemical (water) irritants by the use of simple ointments for several weeks. If this is not done, recurrences are likely to take place and the hyperemia which is present in this stage may be exaggerated.

Local Treatment of Chronic Eczema.—It is rather difficult to outline a definite procedure for the treatment of chronic squamous eczema, as so many remedies are recommended, none of which may be depended upon except in selected cases.

Those remedies which soften the scaly epidermis and reduce the hyperemia of the skin afford the best results.

To soften the scaly epidermis, vaseline, lanolin, or olive oil should be rubbed in once or twice daily; or a 10 per cent. solution of Burow's mixture may be applied as described above.

After thus softening and removing the horny layer, the parts should be painted with a 10 to 20 per cent. solution of the nitrate of silver. The author has used this method after the suggestion of Politzer, with the greatest satisfaction. An immediate cure should not be expected, as several weeks are often necessary to effect it.

Fissures or cracks at the external auditory orifice are best treated with solid nitrate of silver or salicylic acid ointment.

Nearly all the ointments in the *Pharmacopœia* have been used in eczema, but further mention of them need not be made here. If the treatment according to the above principles fails, the case is probably one which will resist all treatment. In the event of failure, special care should be observed to soften thoroughly the scaly epidermis and to remove it, and then the silver solution should again be used. Many of the failures are due to the non-observance of this procedure.

CHAPTER XXXVIII

MALFORMATIONS AND DISEASES OF THE MEMBRANA TYMPANI

In early life the upper portion of the membrana tympani may be absent, with no history or previous suppuration. This is explained by the fact that in the embryo this is the last portion of the membrane to form, and, the process not being complete, a perforation or opening persists. Von Tröltsch suggested that some of the perforations just above or behind the processus brevis mallei, such as are seen in otorrhea, are congenital, but have become enlarged by a subsequent suppuration within the tympanum. This observation may be questioned in certain particulars, in view of the fact that the location of the perforation is usually indicative of the character and seat of the middle-ear involvement. For instance, a perforation in the region of the processus brevis mallei usually indicates a necrosis of the malleus, and possibly, also, of the tegmen tympani. We find that the perforation appears as readily in other portions of the membrana tympani if the focus of the middle-ear lesion is in other locations. Nevertheless, it may be said that a certain number of perforations in the region of the short process of the malleus may be of congenital origin, and that this portion of the membrana tympani is thereby rendered more vulnerable.

INJURIES OF THE MEMBRANA TYMPANI

While injuries to the membrana tympani are comparatively rare, nevertheless, when they do occur it is important to know the proper method of procedure. They may be due to either direct or indirect violence.

Etiology.—*Injuries by direct violence may* be due to (*a*) attempts to remove the cerumen from the meatus with a pin, a hairpin, a toothpick, an earspoon, etc.; (*b*) the accidental thrust of any long slender instrument, tool, or splinter of wood; (*c*) the introduction of a caustic or a hot fluid into the meatus; (*d*) the fracture of the bone which supports the membrana tympani; (*e*) and finally, sneezing, inflation of the ear, etc., may also rupture the membrana tympani.

Injuries by indirect violence may be due to (*a*) the violent and sudden compression of air in the meatus by a blow on the ear with the palm of the hand, or it may be due to (*b*) the concussion of the atmosphere during a violent explosion or discharge from a large cannon. In view of the more or less familiar occurrence of windows being blown outward at the time of an explosion, it may be readily appreciated how the membrana tympani may be ruptured by such an atmospheric disturbance.

Symptoms.—Pain is a prominent symptom in those cases in which there is severe reactionary inflammation, while it may be absent if little or no inflammation follows the injury. In some cases the pain is only present at the time of injury. Hemorrhage, more or less severe, may immediately follow the injury, or in rare cases it may continue for an indefinite period. Faintness, giddiness, nystagmus, staggering gait, convulsions, and nausea characterize those cases in which the foot plate of the stirrup is forced inward, and in which the trauma irritates or otherwise injures the labyrinth. The loss of hearing may be partial or complete and temporary or permanent. The tinnitus at first comes on as a loud noise, and then subsides until it is only moderate in severity or entirely ceases. The effects upon the hearing are various. Deafness may be so great that the watch can only be heard by contact, or, on the contrary, the patient may suffer from hyperesthesia acoustica. When the labyrinth is injured, the deafness may be great or absolute. If the injury involves the semicircular canals, the equilibrium may be disturbed for a few days or weeks.

If the injury occurs in an ear in which the drumhead is adherent to the promontory, it may overcome the adhesions and thus affect the hearing favorably. In some cases the orientation for sounds is lost, while in others there is simply a sense of fulness in the ears.

The rupture is usually located in the postinferior quadrant of the membrana tympani, the periphery not usually being involved, as the membrane is thicker and firmer near its border. The appearance of the rupture is usually a mere slit (dark line), which varies in extent and shape. In other cases, it may appear as a round perforation with ecchymotic spots scattered over the membrane. If the injury were inflicted by a blunt instrument, the perforation is irregular or ragged in outline.

Cases have been reported in which there was an escape of cerebrospinal fluid from the ear, a foreign body having entered the labyrinth. The fluid may also escape into the middle ear when there is a fracture through the petrous portion of the temporal bone.

The ossicles of the middle ear, more particularly the malleus, are sometimes fractured. While the fractured parts reunite, they do not usually do so in their normal position. The author once saw a case in which the handle of the malleus was fractured about 1 mm. below in short process and the parts reunited in nearly or quite their normal position.

Prognosis.—The prognosis is usually good, as the injury ordinarily consists of a simple laceration or perforation of the membrane. In those cases in which the labyrinth is involved the prognosis should be guarded. If the injury to the labyrinth consists of a perforation of its outer wall, good result may be expected after the lapse of a few weeks. The giddiness and nausea may persist for one or more weeks. If the osseous walls of the middle ear are fractured, or if the ossicles are injured, the hearing may be permanently impaired. Should purulent inflammation complicate the case, the prognosis becomes more grave. The functional tests of hearing should be used in all cases of fracture or injury, as by them the

surgeon is enabled to draw conclusions as to the extent and location of the injury and as to the probable outcome of the case.

Treatment.—In nearly all cases no treatment should be used other than the introduction of a cotton or gauze tampon into the meatus to prevent the entrance of infectious matter through the wound. If, in spite of this simple precaution, marked inflammatory symptoms develop, leeches should be freely applied over the mastoid region and in front of the tragus, to promote the reaction of inflammation and thus aid in destroying the bacteria. Great care should be exercised in the treatment of these cases lest infection be carried into the wound and the case become complicated by suppurative inflammation of the middle ear and mastoid cells; hence, meddlesome treatment is to be condemned.

MYRINGITIS; INFLAMMATION OF THE MEMBRANA TYMPANI

Etiology.—Myringitis may be primary or secondary. The primary form is rare, and when present it is usually due to an injury by a foreign body, instrumentation, or the introduction of caustic fluids into the meatus. Secondary inflammation of the membrana tympani is more common, and is due to an extension of an inflammatory process from the auditory meatus or the cavum tympani. Thus, in the various forms of dermatitis and acute otitis media catarrhalis it is often present.

Symptoms.—The chief symptoms are pain, more or less severe in character, with a slight rise in temperature. Deafness and tinnitus are present in proportion to the local injury, the swelling of the membrana tympani, and the nature of the associated middle-ear disease.

Objective Symptoms.—The membrana tympani is usually most affected in its upper portion and especially along the line of the handle of the malleus. In this region it is yellowish red in color, from the congestion present. In a few days or hours, the handle is lost to view, owing to the intense congestion and infiltration of the membrane, the upper portion of which bulges outward into the canal. The epidermic layer may become separated from the fibrous or middle layer of the ear drum by the serous or seropurulent fluid which accumulates between them. Blisters or blebs sometimes form. The inflammatory process may involve the osseous portion of the canal and thus obliterate the line of demarcation between the eardrum and the canal.

The *mode of termination* is by slow resolution, and the signs of inflammation often persist for many weeks. In some cases fatty degeneration or even calcareous deposits may remain after the disease is cured.

Abscess of the membrana tympani may occur in the course of acute otitis media. The process is confined chiefly to the fibrous and the mucous membrane layers, in contradistinction to the blisters which form under the dermic or outer layer.

Vesicular or *herpetic eruptions* sometimes complicate myringitis, as referred to above.

Hemorrhagic eruptions similar to those described under Otitis Externa Hæmorrhagica are occasionally present.

Diagnosis.—The chief diagnostic point to be found is the *slight disturbance of hearing.* The ear appears to be extensively and seriously involved, while the hearing is but slightly impaired. The appearance is much like that of acute suppurative otitis media, but the loss of hearing is slight as compared with that which occurs in the latter disease.

Prognosis.—The prognosis must be based upon a knowledge of the etiology of each case and upon the destructive or degenerative changes which occur in the membrana tympani. If the myringitis is due to a severe injury, or if fatty degeneration and calcareous deposits are in the substances of the membrana tympani, the prognosis is less favorable than when the case is simple in origin and of slight severity. On the other hand, if perforation takes place and chronic suppurative otitis media supervenes, the prognosis is still more unfavorable.

Treatment.—The treatment is (*a*) general, (*b*) local, and (*c*) surgical. The general treatment should consist in the administration of tonics, the iodides, and cod-liver oil if the patient is subject to any dyscrasia; saline cathartics should also be administered. The local treatment should consist of the application of natural or artificial leeches to the mastoid process, to increase the hyperemia and leukocytosis, *i. e.*, promote the reaction of inflammation. The instillation of solutions of cocaine are advised, but are of doubtful utility unless used in the following combination:

℞.—Cocaine hydrochloratis,
Menthol crystals,
Carbolic acid crystals . . . . . āā ℥j—M.
Sig.—One or two drops in the fundus of the auditory meatus will relieve the pain in from five to fifteen minutes.

The parts are at the same time anesthetized and prepared for the opening of the abscess in the membrana tympani if it is present. The remedy should be used with some caution, as it is likely to be absorbed in sufficient quantity to cause toxic symptoms. The instillation of alcohol into the meatus dilutes the solution and facilitates its removal if it should become necessary.

The *surgical treatment should* consist in the incision of the outer or dermic layer of the membrana tympani. In cases which are complicated by abscess, care should be exercised to avoid perforating the inner layer, as infection might thus be carried to the middle ear. Gruber recommends making incisions in the osseous portions of the auditory meatus near the membrana tympani. The incisions should be about $\frac{1}{8}$ inch long and parallel with the circumference of the drumhead, so as to incise the arterial branches at its circumference. The incisions promote the reaction of inflammation and favor resolution.

After the abatement of the acute stage a serous discharge is given off from the membrana tympani and the painful symptoms subside. The ear should now be irrigated with a warm boric acid solution, dried, and the meatus closed with absorbent cotton.

The cavum tympani (middle ear) may be inflated by the Politzer

method; the diagnostic tube should be used to determine if a perforation is present. The membrana tympani should also be inspected for the same purpose. If a perforation is present, the diagnostic tube conveys to the examiner's ear the whistling sound characteristic of a perforation. The membrana tympani may be so swollen that the perforation cannot be seen. The discharge of pus into the meatus is another indication of the presence of a perforation. This is rendered all the more probable if the discharge contains strings of mucus. The presence of a perforation and chronic otitis media render the prognosis more serious.

PERFORATION OF THE MEMBRANA TYMPANI; ULCERATION OF THE DERMIC LAYER; CHRONIC MYRINGITIS; CHRONIC INFLAMMATION OF THE DRUMHEAD

Etiology.—The causes leading to perforation of the membrana tympani may be either *external* or *internal*. One of the *external* causes is acute myringitis, with local fatty degeneration and subsequent sloughing of the substance of the drumhead, the degenerative process beginning with the outer layer and extending inward. Another external source is acute dermatitis of the external meatus. This may extend to the drumhead and result in the same degenerative and perforative processes. In many instances the fatty degeneration is not followed by perforation, but calcareous changes occur instead.

In some cases the destructive process is limited to a simple ulceration of the dermic layer, which may appear as a simple circumscribed roughness of the surface or as a reddened area where the epidermis is removed.

The *internal* causes of perforation or chronic inflammation are either the acute catarrhal or the acute suppurative forms of otitis media. The mucous layer of the drumhead first undergoes the ulcerative process, and the fibrous and dermic layers are involved at subsequent periods. The membrana tympani may long remain the seat of chronic inflammation, because the bloodvessels are injected and radiate from the margins of the ulceration or perforation.

Symptoms.—If the lesion is simple—a superficial dermic ulcer—the symptoms are slight, and tinnitus and a moderate disturbance of hearing are present. If the ulcer is phlegmonous in type, pain and increased deafness result. The secretions and the exfoliation of epidermis form crusts on the surface of the membrana tympani, which obscure the real lesion. Granulations may spring from the bottom of the ulcer.

In those cases in which there is perforation, the tinnitus and the deafness are great. If the middle-ear cavity is not primarily infected, it becomes so through the perforation. Pus is discharged through the opening into the external auditory meatus. If the ear is inflated by the Valsalva, the Politzer, or the catheter method, a whistling noise may be heard through the diagnostic tube. Inspection, after removal of the debris from the auditory meatus, usually reveals the perforation. It is often oval, though it may be round, pear- or kidney-shaped. Its

location generally indicates the focal centre involved within the middle ear or the accessory mastoid cavities.

Course.—The duration of chronic inflammation of the membrana tympani, with or without perforation, is usually quite prolonged. The dermic layer often undergoes repeated or continuous desquamation, or there may be foci of fatty degeneration with calcareous deposits. In some cases there is an atrophic process which renders the membrane thin and parchment-like, and its function is thereby impaired. In still other cases of external origin perforation occurs, and is followed by infection and suppuration within the middle ear. This may continue indefinitely, or until ulceration and necrosis of the bony walls of the middle ear and the pneumatic spaces of the mastoid process occur.

Treatment.—In those cases in which there is an active desquamation or dermic ulceration, the crusts should be softened with a warm solution of bicarbonate of soda, and then removed by syringing with a warm solution of boric acid. The author's experience has justified the local application of 10 gr. solution of the nitrate of silver or of the compound tincture of benzoin. The nitrate of silver stimulates healthy granulation and regeneration, and the compound tincture of benzoin is astringent and stimulates the process of repair.

If perforation has taken place and the cavum tympani is not yet infected, an endeavor should be made to bring about regeneration of the membrana tympani, and thus close the perforation. This may be done by maintaining the external auditory meatus and the membrana tympani in an aseptic condition, and by making stimulating applications to the margins of the perforations, with the view of promoting granulation until the opening is completely filled in. Various drugs and procedures have been employed for this purpose, the best one being the local application of a 20 per cent. solution of trichloracetic acid.

For the treatment of the middle ear complications see Suppurative Diseases of the Middle Ear.

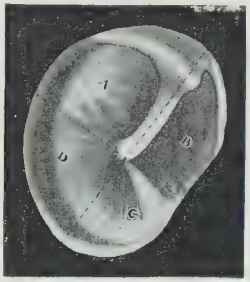
INCISION OF THE MEMBRANA TYMPANI

This method of treatment is coming into vogue more than formerly, as clinical experience has demonstrated that when it is done at the proper time an acute inflammation of the middle ear is aborted. Its effects are due to the promotion of the reaction of inflammation and the facility with which the drainage of the tympanic cavity is accomplished. The presence of the inflammatory exudate within the cavum tympani is a source of irritation because of its chemical composition and on account of the pressure it exerts upon the swollen and inflamed mucous membrane. It is, therefore, important that free drainage be established at a very early period in the course of the disease. Formerly, it was recommended that simple puncture of the drumhead be made for this purpose. Hovell advocates this procedure. The author's experience, however, has shown that such an incision is too small and that a free incision is attended by

immediate and better results. No harm comes from free incision of the membrana tympani, as union often takes place before it is desirable. Even when union does not occur early, only a very slight amount of scar tissue is left behind.

The operation should not be delayed until there is bulging of the membrana tympani, but should be undertaken as soon as there is marked redness and thickening. If the incision is delayed, the membrana tympani may be so swollen and red that the outline of the malleus cannot be distinguished, and bulging of the drumhead may occur, resulting in serious and extensive pathological changes. If it is done early, the progress of the disease is checked and the process of resolution is established. The incision increases the hyperemia and leukocytosis, and thus raises the resistance of the tissue and destroys the microörganisms.

FIG. 392

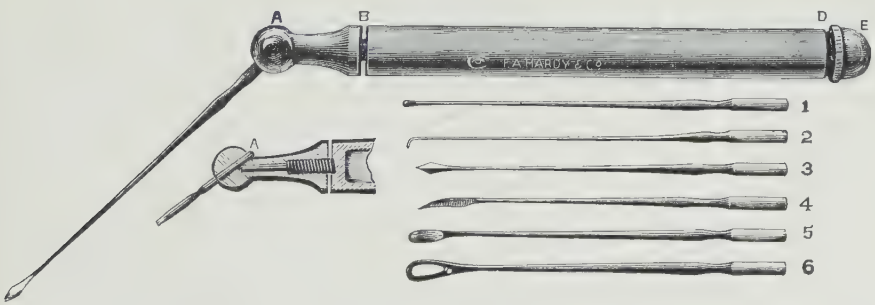


Right membrana tympani, showing the division into *A*, postsuperior quadrant; *B*, anterosuperior quadrant; *C*, antero-inferior quadrant; *D*, postinferior quadrant.

The *most suitable place for the incision* is in the posterior inferior quadrant (Fig. 392), as this is generally the most accessible, owing to the curvature of the anterior wall of the external auditory meatus, which obstructs the view of the anterior portion of the membrana tympani.

The best instrument for the purpose is a curved bistoury (Fig. 393). The lance-shaped or the pear-shaped knives are not well adapted, as they are made for simple paracentesis. The point of the knife should be introduced only far enough to penetrate the thickness of the membrana

FIG. 393



Ear instruments.

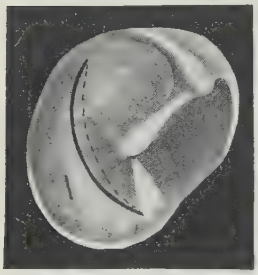
tympani, as to pass it deeper might subject the inner wall of the cavum tympani to injury. It should be remembered that the distance from the outer to the inner wall is only about $\frac{1}{12}$ to $\frac{1}{6}$ inch. The incision should be curved or V-shaped (Fig. 394), to allow a wider opening between the lips of the incision, and should be from $\frac{1}{4}$ to $\frac{3}{8}$ inch in length. In this way free drainage is established.

Immediately after the incision a bead of viscid mucus may be seen

protruding through it. The contents of the tympanic cavity are not discharged at once unless they are of a fluid nature, and to hasten this discharge, a solution of boric acid or bicarbonate of soda may be dropped into the meatus to liquefy it.

Previous to the incision the external auditory meatus should be cleansed with a 1 to 4000 solution of bichloride of mercury to render the membrana tympani and the auditory meatus sterile. Anesthesia of the membrana tympani may be obtained by dropping a small quantity of a solution composed of equal parts by hydrochlorate of cocaine, menthol, and carbolic acid into the auditory meatus. In from five to fifteen minutes complete anesthesia is produced, and the incision may be made with comparatively little or no pain. Complete absence of pain is not always obtained, however, as it should be remembered that the parts contiguous to the membrana tympani are often inflamed and sensitive.

FIG. 394



Showing a long, curved incision through the membrana tympani for the evacuation of inflammatory secretions. With such an incision the anterior flap is forced aside by the secretions as indicated by the dotted line, thus providing free space for drainage. A simple puncture or paracentesis as shown by the short line is inadequate and should not be practised.

Immediately after the incision the auditory meatus should be dried with a cotton-wound applicator and then loosely packed with sterilized gauze. The end of the strip of gauze should be made to touch the incised portion of the drumhead, while the rest is placed loosely in the meatus. It should be left in place until it becomes saturated with the secretions, when it should be removed and a fresh one introduced. During the first two or three days it may be necessary to pack the meatus two or more times a day. The patient should be kept in bed during this time, as much more favorable and rapid progress may be made under such conditions. After the first few days it is not necessary to dress the meatus so often, once a day being quite sufficient. A little later every other day will be all that is necessary. The dressings should be discontinued when the discharge through the incision ceases.

After the incision is made, all applications of solutions by means of the syringe are to be stopped, as infection may thereby be conveyed through the opening into the tympanic cavity. When the acute inflammation has somewhat subsided, inflation by the Politzer method through the Eustachian catheter should be performed to facilitate drainage.

Spontaneous perforation of the drumhead may take place in the course of the disease from softening of the tissues by maceration or from pressure necrosis. As already stated, this should be anticipated, if possible, either by instrumental perforation of the drumhead or by one or more of the remedies which have been recommended. Should spontaneous perforation occur the treatment should be similar to that recommended after incision of the membrana tympani.

Paracentesis is an almost obsolete form of incision, and is not given as synonymous with incision. The latter means a larger and more extensive opening in the drumhead than is implied by the former. By paracentesis is meant a mere puncture through the membrane with a double-edged or spear-pointed knife. What follows, therefore, refers to some form of incision and not to a mere puncture of the drumhead.

The *general purposes* of incision of the membrana tympani are: (*a*) To relieve pain; (*b*) to establish drainage for excessive secretions (catarrhal and suppurative); (*c*) to open the middle ear for certain operations; (*d*) to relieve intralabyrinthine pressure; (*e*) to allow sound waves to reach the oval and round windows; and (*f*) to promote the reaction of inflammation.

The indications for incision, as briefly outlined in the preceding paragraphs, may be amplified as follows:

1. In otitis media with excessive secretion, it may become necessary to make a free incision to prevent pressure necrosis of the drumhead and the tympanic mucosa. The secretion is often so thick and tenacious that it will not discharge through the Eustachian tube. Retention also causes pain, and there is danger of decomposition and infection. The incision also promotes the reaction of inflammation, and thus favors speedy resolution.

The operation should not be delayed until pronounced pain develops, bulging of the membrane being ample justification for the procedure. Should pain persist without bulging, the incision should be made, as it promotes the reaction of inflammation and thus favors resolution.

2. In acute myringitis abscess formations may occur between the layers of the membrana tympani. They should be opened, care being taken not to cut the inner or mucous layer which would expose the middle ear to the dangers of infection from the abscess.

Pearly gray blisters sometimes appear on the membrana tympani. These should be pricked, for if left to discharge spontaneously they prolong the danger of infection.

Inflammation of the deeper layers with bulging and purplish swelling should be scarified to relieve the pain and tension. Incisions through the entire thickness should not be attempted, for the reasons already stated.

3. Tenotomy of the tensor tympani muscle is sometimes performed to relieve deafness and tinnitus. (See Tenotomy of the Tensor Tympani Muscle.) The preliminary step in the operation is an incision of the membrana tympani.

4. *In chronic catarrhal otitis media*, a thickened membrana tympani from hyperplasia with obstruction of the Eustachian tube is often present. The rarefaction of the air within the tympanum causes the retraction of the membrana tympani and pressure upon the labyrinthine fluid by the foot plate of the stapes. The drumhead may be incised as a temporary measure, or a portion of the drumhead may be removed with a knife or cautery to admit air into the middle ear when the Eustachian tube is obstructed. All such measures have met with but partial or temporary success, the opening usually closing within a few days.

The relief is often pronounced while the perforation remains open, but quickly disappears after it closes.

Malherbe has written extensively upon what he terms "Evidement of the Mastoid," whereby a channel of communication between the tympanic antrum and the external acoustic meatus is established, as in the meatomastoid operation, which permanently overcomes the disturbance due to the closure of the Eustachian tube.

5. *In acute catarrhal otitis media*, attended with pain, bulging, and marked inflammatory infiltration, incision or scarification is often indicated to promote the reaction of inflammation and to establish drainage. If there is persistent pain, with or without bulging of the membrana tympani, incision is indicated. The relief which follows may be due to the hemorrhage, for in many cases there is no discharge of secretions for several hours, though it is more probably due to the promotion of the reaction of inflammation.

When there is a livid, boggy appearance of the membrane, it should be freely scarified, limiting the incisions to the outer layer. Circumscribed red spots sometimes appear in the course of the disease, which should be opened to hasten the process of resolution.

The most bulging portion of the membrana tympani may appear yellowish green in color, even though there is little pus in the secretion. Free incision should be made to establish drainage and to relieve the pressure necrosis which is beginning on the inner surface of the membrana tympani.

6. *Acute suppurative otitis media* affords the most common opportunity for incision of the membrana tympani, although it is often postponed until voluntary rupture occurs. The presence of pus within the middle-ear cavity when the drumhead is still intact is an imperative indication for incision. It is not necessary to wait for pain and bulging of the membrana tympani; in fact, it is *culpable negligence* to do so, as every hour adds to the destruction of tissue. *Incise the membrana tympani at once when the presence of pus is suspected in the middle ear*, as it is of the greatest importance to promote the reaction of inflammation to combat the bacteria and their toxins.

The perforation in acute suppuration is usually small, hence it should often be enlarged by radiating incisions toward the periphery (Fig. 395).

Persistent pain without bulging or profuse discharge of pus is an indication of retained pus within the antrum and mastoid cells. The incision in these cases should include the pars flaccida (Shrapnell's membrane), to afford a direct outlet from the attic and to increase the reaction of inflammation.

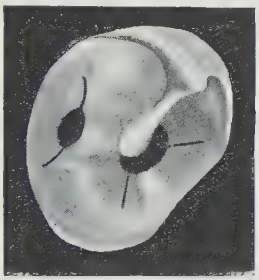
7. *Adhesive processes in the middle ear* sometimes gives rise to conditions which can be more or less relieved by incising the membrana tympani. The adhesive process may interfere with the vibratory action of the ossicles without the foot plate of the stapes being ankylosed. The opening in the drumhead admits sound waves into the tympanum where they strike the foot plate of the stapes, and fairly good hearing results. The tinnitus which is associated with the deafness is also relieved to some

extent. As it is not practicable to maintain the opening for any considerable length of time, the procedure has almost fallen into disuse.

Calcareous deposits in the membrana tympani are often found associated with adhesive processes. They act as foreign bodies and impair the vibratory function of the membrana tympani, and an opening, as above stated, admits sound waves directly to the oval window. Moreover, the equilibrium of air pressure is thereby established and the pressure on the labyrinth by the ossicles is somewhat lessened.

Through the opening it is sometimes possible to sever adhesive bands which extend from the malleus and incus to the walls of the tympanum. While the beneficial effects thus obtained are not permanent, temporary relief is marked and extremely gratifying to the patient. They are much depressed in spirits, and the temporary respite adds to their happiness. It should be frankly explained that the beneficial result will in all probability not be permanent.

FIG. 395



Showing two perforations of the membrana tympani and the incisions for facilitating drainage through them. The incisions should extend at an angle to the axis of the perforation so as to form movable flaps which may be pushed aside by the secretions.

8. Atrophy and relaxation of the membrana tympani from too frequent inflation or other causes may be improved by light scarification with a sharp-pointed bistoury. Only the outer and the middle layer should be cut through. In this way the scar tissue and blood supply will be increased, and the tension and tone of the membrane raised, with benefit to the hearing.

9. Exploration of the middle ear and the attic sometimes becomes necessary in chronic suppuration. This is best done when the opening in the membrana tympani is high, as the roof or tegmen is usually necrosed. If, therefore, the perforation is small or in the lower portion of the drumhead, it may be necessary to extend it by incision in an upward direction. This operation allows a small curved ear probe to be introduced into the attic for exploratory purposes.

Preliminary examination of the function of hearing should be made before incising or removing a portion of the drumhead to improve hearing in adhesive processes of the middle ear. Unless bone conduction for the watch and the c_2, 512 v., fork is good, but slight improvement will follow the operation. Lowered bone conduction is usually significant of

labyrinthine disease, hence incision of the membrana tympani will be of no value.

The middle and the lower portion of the posterior half of the membrana tympani is less sensitive than the upper portion, the sensitiveness gradually increasing as the upper limit is approached. Blake takes advantage of this fact and punctures the membrane in its least sensitive area, then applies cocaine to the cut surfaces, waits a few minutes, and extends the incision slightly upward, applies more cocaine, and so continues until the incision is extended the desired length.

He also recommends the injection of a 2 per cent. solution of cocaine through the Eustachian catheter into the middle ear, as a means of producing anesthesia of the membrana tympani in middle-ear operations.

Dupuy recommends the following mixture as a reliable local anesthetic in eardrum and middle-ear operations:

℞.—Aniline oil,
Alcohol . . . . . āā ʒj
Cocaine hydrochlorate . . . . . gr. vj—M.
Sig.—Drop into the meatus and middle ear.

This mixture does not always produce local anesthesia. In a number of the author's cases it has failed, notably in aural polypi.

More or less cyanosis occasionally attends its use, hence it should be applied with caution.

The following mixture is more reliable and less dangerous:

℞.—Cocaine hydrochlorate,
Menthol crystals,
Carbolic acid crystals . . . . . āā ʒj—M.
Sig.—Drop into the meatus or middle ear, and in twenty minutes anesthesia is complete.

The absorption is greatly facilitated by first macerating the drumhead with the peroxide of hydrogen.

Methods of Operating.—The electrocautery may be used in adhesive non-inflammatory cases. The opening thus made remains longer than one made with a knife. The points to be observed are the following:

(*a*) Preliminary local anesthesia should be produced by the injection of the above formula or a 2 per cent. solution of cocaine into the middle ear through a Eustachian catheter.

(*b*) The electrode should be a simple straight, pointed one with the shank so bent that the electrode handle and the hand of the operator do not obstruct the view.

(*c*) The current should be sufficient to instantly raise the point to a bright-red heat. If the platinum point heats too slowly the adjacent parts may be injured by the radiation of heat. The pressure exerted by the electrode should be slight to avoid the danger of injuring the mucous membrane of the inner tympanic wall.

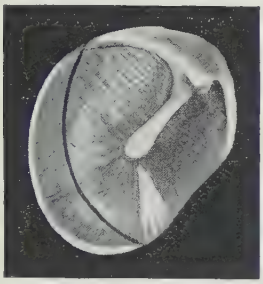
(*d*) Contact should be made with the drumhead before the electric current is turned on.

(*e*) Usual time of heat contact, one second.

Incision with a Lancet.—Preference should be given to Hartman's curved lancet, the spear-pointed instruments formerly used being of little value except for simple puncture.

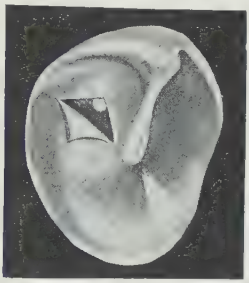
The most favorable or available location for incision in adults is the posterior half of the drumhead (Fig. 392). In children the external meatus is shallow and straight, so that all portions of the drumhead are accessible.

FIG. 396



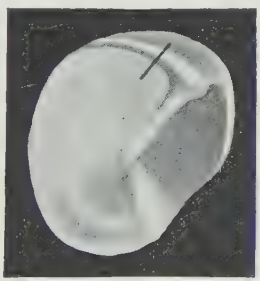
Showing a long, curved incision of the membrana tympani extending into the superior wall of the meatus (white line). As there is a plexus of bloodvessels around the margin of the membrana tympani, greater reaction of inflammation is produced by extending the incision through it, hence the improvement of the inflammation is more prompt than in simple incision of the membrane. (See Reaction of Inflammation.)

FIG. 397



Incision for stapedectomy, showing the incudostapedial articulation. The stapedius muscle should be severed to prevent the dislocation of the stapes, the incudostapedial articulation broken, and the stapes removed from the oval window. This operation is rarely justifiable.

FIG. 398



Showing an incision through the posterior fold of the membrana tympani to relieve the tension of the membrane in adhesive processes.

Other things being equal, the most bulging portion (fluid being present) should be incised, because it is the point of least resistance and because the parts are not so sensitive in this area. If the bulging is pronounced, the incision can often be made without the use of a local anesthetic.

The length, direction, and character of the incision should depend upon the purpose for which it is made. If it is done to establish *free drainage*, it should be long and curved, or angular (Fig. 396). If it is to expose the contents of the middle ear, as for operations upon adhesive bands and upon the stapes, the V-incision recommended by Blake (Fig. 397) should

be made. If it is for the purpose of admitting air to the middle ear, a round or triangular opening may be made. The cautery is well adapted for this purpose. If it is done preliminary to tenotomy of the tendon of the tensor tympani, or for plicotomy, a short, straight incision (Fig. 398) is all that is necessary.

Postoperative Considerations.—(*a*) When the *incision* is made to evacuate mucus or mucopus, a pulsation synchronous with swallowing and articulation will occur at the point of incision. Pus and mucus rarely appear immediately after the incision. This is quite disconcerting to the inexperienced aurist, as he may have unwittingly promised an immediate evacuation of the secretions. A little experience, however, will teach him that on account of the thick and adhesive character of the secretions, they will usually require several hours to appear. The expulsion of the secretions can be hastened by instilling a warm solution of bicarbonate of soda into the middle ear. The soda overcomes the adhesive property of the mucus and facilitates its discharge. Sometimes the mucus is so thick and tenacious that it can be seized with forceps and removed. It may also be removed by suction with the Delstanche masseur.

(*b*) Closure of the incision in non-suppurative cases usually occurs in from one to three days. In suppurative cases it may remain open a few days or indefinitely.

(*c*) The *dressing* should consist of a strip of sterilized gauze placed loosely in the meatus, but touching the drumhead. If the discharge is profuse, a pad of gauze may be placed over the auricle and held in position by a bandage. The meatus and the auricle should first be cleansed with a 1 to 3000 bichloride solution before introducing the gauze dressings.

CHAPTER XXXIX

DISEASES OF THE EUSTACHIAN TUBES

THE RELATIONSHIP OF THE EUSTACHIAN TUBES TO HEARING AND MIDDLE-EAR DISEASES

The Eustachian tube is the chief source of communication between the epipharynx and the middle ear. Through it the tympanic cavity is ventilated and the normal tension of the drumhead and the ossicular chain is maintained, thereby facilitating the transmission of sound waves to the internal ear. The pharyngeal end of the tube is supported by cartilage, while the tympanic end has an osseous framework. At the union of the cartilaginous and the osseous portions the tube becomes narrow, forming what is known as the isthmus. The throat end is subject to the diseased processes peculiar to the epipharynx, while the tympanic end is affected by the changes peculiar to the tympanic cavity. In other words, the throat end is subject to pronounced catarrhal and suppurative inflammations and to hypertrophy of the lymphoid tissue in its mucous membrane, and the tympanic end to catarrhal and adhesive changes in addition to the suppurative process. The adhesive process is, therefore, chiefly found in the less accessible portion of the tube – namely, beyond the isthmus, and consequently difficult to reach with electrolytic bougies, or those used for the purposes of simple dilatation.

The relationship of the Eustachian tube to the diseases of the tympanic cavity is twofold, namely: (*a*) Obstruction of its lumen by catarrhal congestion, hypertrophy, cicatricial contraction, and mucous plugs; and (*b*) as an avenue through which infective material may gain entrance to the middle ear. The obstructive lesions or accumulations prevent the proper ventilation of the tympanic cavity, and the contained air becomes rarefied through the gradual absorption of the oxygen, thus causing retraction of the drum membrane and engorgement of the bloodvessels of the mucous membrane.

The retraction of the drumhead increases the tension of the ossicular chain, and interferes with the normal transmission of sound waves to the labyrinth. Tinnitus and deafness thus result. The obstruction to drainage lowers the resistance of the tissues and predisposes to infection and inflammation.

Infectious material may gain entrance into the middle ear during acts of yawning, coughing, sneezing, or swallowing. The tube is lined with ciliated columnar epithelium, having a wave-like motion toward the pharyngeal orifice. In the healthy state bacteria rarely travel toward

the middle ear on the mucosa. If, however, the catarrhal inflammation of the lining membrane of the tube is severe or prolonged, the epithelium may lose its cilia, and allow germs to reach the middle ear without the tube being opened by the acts of coughing and sneezing.

Tubal tonsils, or *hypertrophy* of the lymphoid tissue in the mucous membrane of the cartilaginous portion of the tube, is another possible source of obstruction. A study of the histology of this structure shows lymphoid tissue to be present in considerable quantity, and it is more than probable that hypertrophy of this tissue is often responsible for tubal and middle-ear disturbances heretofore ascribed to catarrhal or other diseases.

TUBAL CATARRH; CATARRHAL INFLAMMATION OF THE EUSTACHIAN TUBE; SALPINGITIS

Etiology.—Owing to the intimate anatomical connection of the mucous membrane of the Eustachian tubes with that of the epipharynx, it is easy to understand why they are usually involved in the course of an attack of epipharyngeal inflammation. If the epipharyngitis is chronic in character, the tubal disease is likewise chronic. While tubal catarrh is usually secondary to a like process in the epipharynx, it is not always so, especially in children. In young children the pharyngeal orifice is narrow and is easily obstructed by the secretion and foreign matter. For this reason local inflammation may occur in the tubes independent of the epipharynx.

Adenoid growths are often associated with a chronic epipharyngitis, which extends by continuity to tissue of the tubes. The adenoids do not often afford a mechanical obstruction to the patency of the tubes, as they grow from the posterior and superior walls of the epipharynx, and, therefore, do not involve the regions of the Eustachian orifices on the lateral walls. In some instances, however, they overlap the mouths of the tubes and thus obstruct them. Tuberculosis may be associated with adenoid growths and predispose to tubal inflammation.

Thomas H. Brunk first, and later W. S. Bryant, called attention to the presence of granulation tissue and adhesive bands in Rosenmüller's fossæ, claiming that their removal with the finger introduced through the mouth, or with a straight curette through the nose, relieved tubal catarrh and deafness. Indeed, this opinion is attracting considerable attention, as the removal of these bands have in numerous cases been followed by great improvement. The adhesive bands are frequently present and should be searched for more frequently than has been customary.

Pathology.—Congestion and round-cell infiltration characterize the early and acute stages of the disease. At a later period the epithelial covering becomes thickened, and fibrous tissue is deposited in the subepithelial layers. Hypertrophy of the mucous membrane occurs when the inflammation continues for a long time. If the inflammation

is severe or prolonged, the cilia are exfoliated, leaving the membrane denuded in places. The catarrhal inflammation may extend to the middle ear, although it has a tendency to limit itself to the pharyngeal or cartilaginous portion of the tube.

Symptoms.—The subjective symptoms are a feeling of fulness in the ears, which may be constant or intermittent, accompanied by subjective noises and deafness. Pain is not usually severe, although it may be if the inflammation is pronounced. If there is marked retraction of the drumhead, giddiness and nausea may be complained of. The sense of deafness is often out of proportion to the actual deafness. The patients apply for relief with the statement that the external canal is filled with cerumen. During mastication and swallowing, they often experience marked, though brief, relief from the symptoms. This is explained by the incidental, but incomplete, ventilation of the tympanum during the act of swallowing. Upon *posterior rhinoscopy*, the mucous membrane of the epipharynx and the Eustachian orifices appears reddened, swollen, and covered with a thick mucous secretion. The mouths of the tubes are contracted by the swollen membrane, and may contain a thick, tenacious mass of mucus. If adenoids are present, the furrows between the lobules are more or less filled with a slimy secretion admixed with pus. The ethmoidal and sphenoidal sinuses may also be the seat of inflammation. With good illumination it is possible to see the enlarged and tortuous bloodvessels in the inflamed area.

The drumhead is more or less changed in its position and appearance by the rarefaction of the air in the tympanic cavity. It is more cupped, the handle of the malleus is foreshortened, and the short process and the posterior fold extending from it are more prominent. The angle formed by the handle of the malleus and the posterior fold becomes more acute with the increased retraction. The cone of light is diminished, broken, or altogether wanting. If the drumhead is extremely retracted, the promontory and the long process of the incus become visible through it.

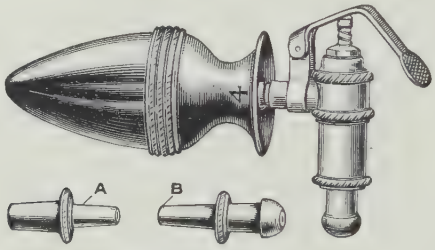
Prognosis.—The prognosis is good in those cases in which adenoid growths are removed, especially in children. It is also good in the early, or congested stage of the simple catarrhal type in adults. In the hypertrophic stage it is not good, as the obstruction is more permanent in character. If the obstruction is due to lymphoid hypertrophy in the pharyngeal end of the tube, the prognosis is not good, although the removal of the adenoids reduces the congestion and improves the deafness. If the obstruction is due to adhesive bands in Rosenmüller's fossa the prognosis is good if the bands are removed.

Treatment.—The treatment of tubal catarrh should be largely addressed to the antecedent nasal and epipharyngeal conditions. If there is pronounced nasal catarrh, sinuitis, nasal obstruction, or epipharyngitis, appropriate treatment should be undertaken, and the adenoids should be removed. Removal of the adenoids is usually followed by pronounced and immediate relief. Having corrected the nasal and the epipharyngeal disorders, the tubal inflammation often subsides without further treatment. Such a favorable result does not always

follow, however, especially if the mucosa has become hypertrophic or hyperplastic in character. In many cases there is a mixture of turgescence and hypertrophy, when local medical applications are only capable of removing the congestion and limiting the further development of the hypertrophic process.

Perhaps the most useful method of applying remedies to the vault of the pharynx and the Eustachian orifices is by gargling after the von Tröltsch method. The patient should lie on his back while gargling, to allow the fluid to enter the epipharynx. This is not difficult, as the head can be turned to one side in taking the fluid into and in ejecting it from the mouth. By following this method, the whole of the epipharynx, including the Eustachian orifices and the nasal chambers, may be reached by astringent and antiseptic remedies, with very favorable results. The deafness and tinnitus are often thereby relieved.

Fig. 399



Buttles-Pynchon inhaler.

The injection of from 1 to 4 minims of weak astringent solutions into each of the Eustachian tubes through a catheter is recommended. Care should be taken to avoid injecting it into the middle ear, as reactionary inflammation might follow. The syringe should be so gauged as to fill the catheter and leave a surplus of from five to ten minims. The extra solution is to allow for the inevitable escape of fluid into the epipharynx. The nose and the epipharynx should be sprayed with a 2 per cent. solution of cocaine to reduce the sensibility of the parts before introducing the catheter. The solutions most often used are: (*a*) The iodide of potassium, 10 gr. to the ounce; (*b*) the bicarbonate of soda, 3 to 5 gr. to the ounce; (*c*) the sulphate of zinc, 1 gr. to the ounce; and (*d*) the nitrate of silver, 2 to 5 gr. to the ounce.

Various vapors of iodine, ammonia, menthol, camphor, eucalyptol, etc., have been recommended. Iodine and ammonia are readily volatile, and the fumes therefrom may be sufficiently generated in a Buttles-Pynchon inhaler, shown in Fig. 399. A piece of sponge or cotton should be moistened with the desired solution and placed in the chamber of the inhaler. The inhaler should be connected with the catheter and air forced through it into the Eustachian tube. Another way of using the vapors of the foregoing drugs, either singly or in combination, is with a nebulizer. Either the nebulizer may be attached to the Eustachian

catheter, or the vapors may be driven into the middle ear by the modified Politzer method, in which the nebulizing device takes the place of the rubber bag used by Politzer. In other respects, proceed according to the directions given under the Politzer method. The author has often put a few drops of the desired volatile solution into the Politzer bag and then practised inflation in the usual manner.

The value of the foregoing topical remedies does not consist alone in the medicinal properties of the drugs, but includes also the mechanical effects of inflation. The current of compressed air directed into the orifice of the Eustachian tube removes the secretions and temporarily unloads the congested vessels and establishes normal glandular activity.

If adhesive bands are present in Rosenmüller's fossa, the index finger of the right hand should be introduced through the mouth and the right fossa thoroughly curetted with the nail. The left index finger should be used to curette the left fossa.

The principles to be observed in the treatment of tubal catarrh may be summarized as follows:

(*a*) The correction of obstructive nasal lesions, and of inflammatory diseases of the nose and accessory sinuses.

(*b*) The removal of neoplasms, adhesive bands, and other inflammatory conditions in the epipharynx.

(*c*) The topical application of antiseptic, astringent, and stimulating remedies to the mucosa of the Eustachian tubes.

(*d*) The mechanical effects of inflation.

(*e*) The administration of remedies to give tone and vigor to the general system.

It should be said, in reference to the latter principle, that in many cases of deafness from tubal catarrh, the administration of tonics and other constructive remedies is often followed by an improvement in hearing. This is especially true in those cases in which there is no pronounced nasal or epipharyngeal disease. It is usually best to begin the treatment with a 2 to 3 gr. dose of calomel at bedtime, followed by a saline cathartic the following morning. After this, laxative doses of cascara may be given twice daily. The patient's alimentary tract is thus kept in a condition to care for and distribute the constructive remedies. These remarks are equally applicable to all catarrhal affections of the upper respiratory tract.

The Relation of the Eustachian Tube to Mastoiditis.—The Eustachian tube is adequate to drain all secretions from the middle ear, but it is often inadequate to drain the combined secretions of the middle ear, mastoid antrum, and cells, resulting in retention, pressure necrosis, and all the phenomena peculiar to mastoiditis. If the secretions from the antrum and mastoid cells are diverted from the middle ear, the Eustachian tube effectually drains it, and the diseased process rapidly improves. (See Author's Modified Radical Operation.)

OBSTRUCTION OF THE EUSTACHIAN TUBE

Partial Obstruction.—Etiology.—Obstruction of the Eustachian tube may be due to a variety of conditions, namely: (*a*) Hypertrophy of the mucous membrane, especially in the pharyngeal or cartilaginous portion, the hypertrophy being an extension of the same process from the nose and the epipharynx. (*b*) Repeated inflammations, giving rise to a hyperplastic thickening and consequent obstruction. (*c*) Adhesive bands or constrictions forming in either the tympanic or the pharyngeal end of the tube, especially if the same pathological process is going on in the tympanic cavity. (*d*) Syphilis, tuberculosis, and diphtheria at the pharyngeal orifice, causing cicatricial contractions which more or less obstruct the opening. (*e*) Adenoids, while they do not grow from the Eustachian orifice, may be so large as to overlap and thus close it. (*f*) Paralysis of the palatal muscles from diphtheria and mixed infection, or from degenerative changes of the muscular fibers from repeated inflammations coincident with tonsillar inflammation, giving rise to collapse of the muscular and other soft tissue at the pharyngeal orifice and thus causing its occlusion.. (*g*) Adhesions of the posterior pillars to the tonsils interfere with the muscular movements and contribute to the collapse of the Eustachian orifices. (*h*) Degeneration of the palatal muscles as a result of severe or repeated inflammation of the tonsils and contiguous structures. (*i*) Certain anatomical features, as exostoses and hyperostoses of the walls of the tubes, give rise to obstruction; there may be a sudden bend in the direction of the tube, or the carotid canal may encroach upon it and thus obstruct it. (*j*) Adhesive bands in Rosenmüller's fossa as described by Brunk.

Diagnosis.—The diagnosis may be made by observing the characteristic retraction of the drumhead, foreshortening of the handle of the malleus, and the prominence of the short process and the posterior fold of the tympanic membrane. Postrhinoscopic examination may show either cicatricial contraction, overlapping adenoids, or collapse of the Eustachian orifice. The pillars (glossopalatine and pharyngopalatine arches) of the fauces may be adherent to the tonsils, and cause more or less atony of the palatal muscles. The diagnostic tube used during inflation gives the strident or rough murmur characteristic of tubal obstruction. If the Eustachian tube is normally patent, the tubal sound is soft and blowing in character.

Complete Obstruction.—This condition may be due to one or more of the causes given under Partial Obstruction, although it is usually due to syphilitic, tuberculous, or diphtheritic cicatricial contraction at the mouth of the tube. The symptoms are the same as in partial obstruction, excepting that tympanic inflation gives no rale or murmur through the diagnostic tube.

Undue Patency of the Eustachian Tubes.—This condition is nearly always associated with atrophic changes in the entire mucosa of the

upper respiratory tract, especially of the nose, epipharynx, and oropharynx. The process may not involve the entire Eustachian tube, but may be limited to the pharyngeal orifice. Urbantschitsch reports a case of this kind in which the end of the little finger could be inserted into the orifice.

The characteristic symptoms are the inward and outward movements of the drumhead synchronous with the respiratory movements, and the soft, blowing murmur heard through the diagnostic tube, even without inflation. There may be autophony or the ringing of the patient's voice in his own ears. The voices of others sometimes give rise to the same disagreeable sensation. The symptom is somewhat different from hyperesthesia acoustica, in which there is a painful distinctness of hearing; whereas in autophony the patient's own voice seems to ring or roar in his head.

Treatment of Obstruction and Undue Patency.—The treatment of partial obstruction varies with the lesion causing it. If there is catarrhal congestion of the mucous membrane at the pharyngeal orifice, relief may be afforded by the judicious use of antiseptic and astringent sprays in the nose and epipharynx. If, however, the hyperemia is due to anterior nasal obstruction, this should be corrected. The removal of adenoids is indicated to relieve the epipharyngitis and the resulting tubal catarrh, as well as to overcome the mechanical obstruction they may form at the mouth of the tube.

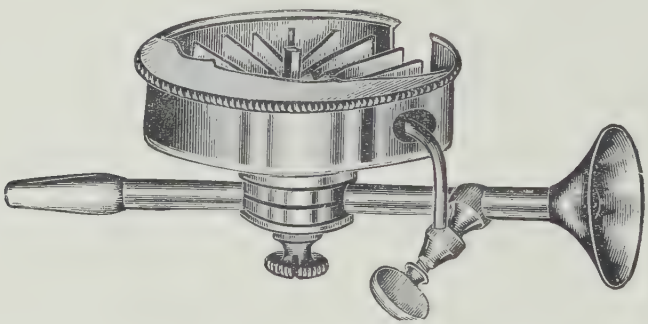
It is difficult to overcome cicatricial contractions, especially if it is due to syphilis. If due to diphtheria and tuberculosis, electrolysis may be of value. An olive-tipped electrode, with the curvature of a Eustachian catheter, should be introduced through a catheter. The tip should enter the Eustachian orifice to the isthmus of the tube. The shaft of the electrode should be covered with some insulating substance and the strength of the current should vary from 5 to 30 ma., according to the density and resistance of the tissue. Seances should last for from five to twenty minutes. The negative pole of the battery should be connected with the Eustachian electrode, as the tissue to be reduced is dense and fibrous. If it is a simple hypertrophy, the positive pole should be used. If the lumen of the tube is constricted higher up by adhesive bands, a small, gold-tipped electrode should be introduced through the Eustachian catheter until it comes in contact with the constriction, as recommended by A. B. Duel. It is claimed for electrolysis in these cases that the obstruction disappears and the hearing and tinnitus are improved. Others have found it of no practical value. The status of electrolysis at best is open to criticism. The benefits derived from it within the Eustachian tube may well be attributed to the dilatation and inflation which are incidental to the procedure. Theoretically electrolysis is an ideal treatment for fibrous constriction, while practically it has been disappointing in the hands of most otologists. In obstinate cases it should, however, be given a trial, and will in some cases be attended with astonishingly good results.

The use of bougies in reducing tubal stenosis has long been recognized

as of considerable value in those cases in which the stricture is not composed of connective tissue. If it is due to turgescence or simple hypertrophy, the results are often good. The bougies may be made of silkworm gut, whalebone, or celluloid. Those made of silkworm gut may be impregnated with astringent remedies, as silver nitrate, sulphate of zinc, etc., which often adds to the therapeutic effect. The whalebone bougie is easier to introduce on account of its polished surface. Celluloid bougies are also smooth and easy to introduce, but are more liable to break.

Suarez di Mendoza has devised a metal catheter which may be removed, leaving the bougie in the Eustachian tube. The catheter is divided longitudinally into two parts, and it can be separated and removed, leaving the bougie in position. It is then cut off even with the nose and left in position for twenty-four hours. By this method, speedy dilatation is obtained.

Fig. 400



Weaver's intratympanic masseur.

Caution.—The introduction of bougies into the Eustachian tube may injure the mucosa, hence emphysema of the submucous tissue may occur if inflation is practised immediately afterward. It should rather be done when the patient returns two days later for another treatment. The introduction of bougies may be practised two or three times a week. In favorable cases, the rough strident murmur heard through the diagnostic tube during inflation will have been replaced, after a few treatments, by a soft, full, blowing murmur.

In some cases great difficulty is experienced in passing the bougie beyond the pharyngeal orifice, as it bends and returns with a sharp tingling or smarting sensation in the lateral walls of the pharynx. The Eustachian catheter should be given a larger and sharper curve, so as to direct the tip of the bougie more in the direction of the lumen of the tube.

The bougie should be pressed firmly against the constriction until it passes it, or until the hope of doing so is abandoned. When it is found impossible to pass the bougie, electrolysis should be tried. Larger bougies may be successively introduced until inflation gives

a free, full, blowing murmur. After this they should be passed at longer intervals for several weeks or months.

Massage of the Eustachian tube may be accomplished by the Weaver masseur (Fig. 400). The masseur is attached to the catheter and the current of air from the compressed-air tank turned on, the turbine wheel interrupting the current of air. The mucous membrane of the tube and middle ear is thus rapidly and intermittently compressed. The bloodvessels and lymphatics are unloaded, and the glandular elements are stimulated to greater activity. The tympanic cavity is inflated and the air tension restored. In turgescence and hyperemia of the tubal membrane, this method of treatment is highly commended.

It should be said in conclusion that no one method of treatment is applicable to all cases. Each should be carefully studied and all the facts considered before determining the line of treatment. The nasal and epipharyngeal condition, as well as the general health, should be regarded as essential factors in determining the course of treatment in each individual case.

CHAPTER XL

THE PRINCIPLES AND METHODS OF TYMPANIC INFLATION

The data of an anatomical, physiological, and clinical character, upon which the principles of tympanic inflation should rest, are as follows:

(*a*) The Eustachian tube extends from the lateral wall of the epipharynx to the cavity of the middle ear in an upward, outward, and backward direction. If the head is rotated to the right and then inclined forward, the right Eustachian tube will stand perpendicular to the plane of the earth, thus favoring the drainage of the right middle ear.

(*b*) The pharyngeal orifice of the Eustachian tube is trumpet-shaped; hence, when a current of air is forcibly thrown into it, the contained secretions are "dished" out and carried into the epipharynx, while the residual air passes on through the tube into the middle ear.

(*c*) The walls of the Eustachian tube are covered with ciliated epithelium, the cilia creating a current toward the pharyngeal orifice. If the secretions are thick and become dried in the orifice, the sudden impact of air during inflation dislodges the mass and clears the way for the successful inflation of the middle ear.

(*d*) The walls of the tubes are approximated when in the normal state of rest, and are only opened during inflation of physiological or artificial origin.

(*e*) The drumhead, being the only yielding wall of the tympanic cavity, is pushed outward toward the external meatus during inflation.

(*f*) The handle of the malleus is also carried outward, as it is in intimate relationship with the drumhead.

(*g*) The incus and the stapes follow the outward movement of the malleus only to a limited extent, as the articulations are such as to permit the malleus to swing in this direction without marked movement of the other ossicles. The inward movement of the handle of the malleus is, however, accompanied by a corresponding, though more limited, movement of the incus and the stapes in the same direction.

It is obvious, therefore, that in adhesive processes affecting the motion of the malleus, inflation exerts more or less influence in breaking them down; whereas if the adhesions affect the incus and the stapes, but slight influence is exerted.

(*h*) The mucosa of the tympanic cavity is supplied by numerous bloodvessels, capillaries, and lymph channels, which upon inflation (in catarrhal inflammation) become less engorged and return to their normal state of fulness. In other words, inflation is followed by an active hyperemia and an approach toward normal physiological activity of

the tissues composing the mucous membrane. The secretions become thinner in character and approach the normal. They are, therefore, more easily carried toward the Eustachian tube by the wave-like motions of the ciliated epithelium.

(*i*) The oxygen is gradually absorbed from the air within the tympanic cavity, hence, after several hours, rarefaction takes place, thereby again causing the drumhead to retract. This does not occur in normal conditions, as air is admitted to the middle ear during each act of deglutition and yawning.

(*j*) The palatal muscles have more or less control over the patency of the tubes, hence it is important that they be free to act to their full capacity. Repeated inflammations of the tonsils and fauces give rise to adhesions to the pillars of the fauces (glosso- and pharyngopalatine arches) and to degenerative changes in the muscular tissue. The action of the palatal muscles is thereby interfered with and the regulation of the patency of the tubes is impaired. The ventilation of the tympanic cavity cannot be fully accomplished, hence more or less deafness and tinnitus follow.

(*k*) Passive congestion of the mucosa also results from the rarefaction of the air in the middle ear, and leads to abnormal activity of the mucous glands, as well as to a change in the character of the secretion. A true catarrhal state is thus induced. Repeated inflations, together with other appropriate treatment of the nose and throat, will, in many cases, be followed by a lessened congestion, a restoration of the glandular activity, and a return to the physiological ventilation of the tympanum.

(*l*) Thick, tenacious secretion is not easily forced from the middle ear through the Eustachian tube by inflation. The circulation and the glandular elements of the mucous membrane become impaired. Nevertheless, the thick tenacious secretion is gradually absorbed or discharged.

(*m*) The transmission of sound waves through the ossicular chain to the labyrinth is only perfectly performed when the tension existing between the drumhead, the ossicles, and the intralabyrinthine fluid is normal. If the tension is disturbed, more or less impairment of the hearing results. Tympanic inflation restores the normal tension, unless adhesive bands prevent the drumhead springing into position.

(*n*) When the drumhead is perforated, the secretion flows from the middle ear into the external auditory meatus.

The foregoing data show that the objects of intratympanic inflation are as follows:

1. To restore the normal tension between the drumhead, the ossicles, and the labyrinth.
2. To restore the normal circulation in the bloodvessels and the lymph spaces.
3. To render the secretions more nearly normal.
4. To remove the morbid secretions from the Eustachian tube and the tympanic cavity.
5. To break down newly formed adhesions.

By establishing the foregoing conditions, tinnitus is relieved, hearing

improved, catarrhal inflammation checked, and the suppurative processes ameliorated.

Methods of Inflation.—Valsalva's Method of Inflation.—While this method is not of such general utility as either Politzerization or catheterization, nevertheless it has a place in otological practice which is not filled by either of the others. Although its therapeutic effects are rather limited, it is of diagnostic value.

The method consists in forcing the air into the middle ear by a forcible expiratory effort while the mouth and the nose are closed. The success of the effort is in proportion to the dynamic power of the muscles of the individual and the character and degree of the obstruction in the Eustachian tube. The muscular power in children and women is less than in adult males, hence it is proportionately less successful in the former.

The *hindrances* to the successful performance of inflation are: (*a*) Thick, tenacious secretions in the mouth and the lumen of the tube, as well as in (*b*) the tympanic cavity. (*c*) When the tympanic cavity is in a state of partial vacuum from the absorption of the oxygen from the contained air, which causes the tympanic end of the tube to collapse by the suction thus created. (*d*) Fibrous adhesive bands resulting from chronic inflammation of the tubal membrane stretching across the lumen of the tube and obstructing it. (*e*) When the mucous membrane in a state of catarrhal inflammation is congested or even hypertrophied, thus interfering with tympanic inflation. (*f*) When the mucous membrane of the Eustachian tube is supplied with lymphoid tissue, which underfavorable conditions undergoes an hypertrophy akin to the same process in adenoids and tonsils, thereby diminishing the lumen of the tube. (*g*) Thick, tenacious secretions in the middle-ear cavity offering resistance to tympanic inflation. (*h*) The fact that there is no exit other than the Eustachian canal for the air entering the middle ear, a factor of some importance. It does not seem to the author, however, that it plays the major role assigned to it by some authors, notably Politzer, who thinks the drumhead offers considerable resistance. In such cases it is only necessary to open the Eustachian tube, when the air will rush in from the epipharynx to equalize the pressure on the two sides of the drumhead. This is the result of physical laws, and requires no force or artificial intervention other than a patent Eustachian tube. After this is accomplished, the air in the middle-ear cavity may be compressed even beyond the line of equilibrium, in order to stretch or break down adhesive bands, or to expel the secretions.

The *diagnostic value* of this method is inferior to the others, inasmuch as it is less sure of being successful. In normal cases, when the desired result is obtained, a soft blowing sound is heard, which Politzer ascribes to the outward bulging movement of the drumhead. The author is inclined to take the view that it is due to the friction of the current of air in its passage through the collapsed Eustachian tube. If the tube is filled with secretions, as in moist tubal catarrh, the sound is changed to a moist bubbling murmur.

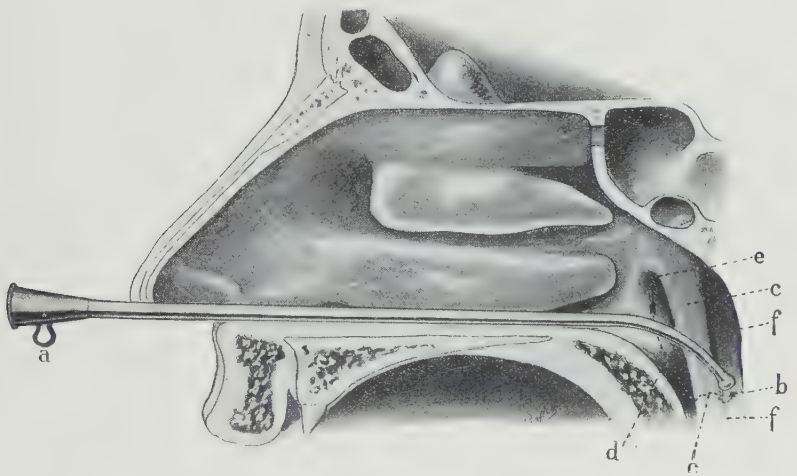
The *prognostic value* of the method is considerable, in view of the fact that in those cases of catarrhal otitis media in which it can be successfully performed the prospects of cure or relief are good.

FIG. 401

Bulbous-tipped silver Eustachian catheter.

Caution.—A word of *caution* should be given in regard to the evils attending Valsalva's method of inflation as a therapeutic measure. If the tinnitus and the "stuffed-up" feeling in the ears are relieved by this method, the patient is tempted to resort to its use so frequently and for so long a period of time that there is great danger of overstretching the membrana tympani, thereby rendering it atrophic. The author never recommends the method for therapeutic purposes, but, on the contrary, often discourages its use by those who have already adopted it.

FIG. 402



Showing a method of catheterization: *a*, the ring indicating the direction of the tip of the catheter; *b*, the posterior wall of the pharynx; *c*, *c*, the ridge forming the posterior lip of the mouth of the Eustachian tube; *f*, *f*, Rosenmüller's fossa; *b*, *d*, *e*, the route traversed by the tip of the catheter to enter the mouth of the Eustachian tube.

Catheterization.—Catheterization was first brought to the attention of the Paris Academy in 1724 by a postmaster named Guyot, but its therapeutic value was not clearly stated until a century later by Saissy, in his treatise on the *Diseases of the Internal Ear*, 1819.

The Binnafont or Kramer method consists in introducing the catheter (Fig. 401) through the inferior meatus of the nose into the epipharynx, where it is turned outward and upward into the mouth of the Eustachian

tube. The curved tip of the catheter should be kept on the floor of the nose at the junction of the floor and the septum. When the tip touches the posterior wall of the pharynx, it should be rotated outward into Rosenmüller's fossa, then rather quickly drawn forward over the bulging posterior lip (plica salpingopharyngeus) of the Eustachian orifice into the pharyngeal mouth of the tube. The eyelet of the catheter indicates the direction of the curved tip, which, when in the mouth of the tube, is generally turned in an upward and outward direction, toward the outer canthus of the eye. In some cases, however, the tip enters the orifice when directed horizontally outward (Fig. 402).

It may be necessary to change the angle of the curvature of the tip to suit individual cases. Saissy recommended an angle of 130 degrees, while Politzer advises 145 degrees.

The best instruments are made of pure silver, as they can be easily changed in shape and may be sterilized in boiling water, eliminating the liability to infection. Before the days of sterile surgery, hard rubber catheters were largely used, and they are still recommended by some authors. Saissy, however, nearly one hundred years ago, recommended silver, which is today preëminently the best material for the purpose.

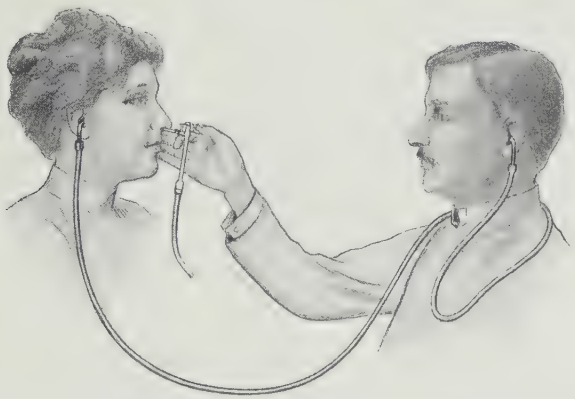
The Lowenberg Method.—The Lowenberg method consists in turning the tip of the catheter, after it has entered the epipharynx, toward the median line until the metal ring on the outer extremity assumes the horizontal position, and then drawing it forward until it touches the posterior extremity of the septum. In making the forward movement the outer extremity should be slightly removed from the septum, so as to bring the curved tip beyond the median line, thereby making sure that it catches on the septum. The outer end of the catheter should then be moved toward the nasal septum, and held near the tip with the fingers of the left hand. The tip should then be rotated downward and outward more than 180 degrees, or through more than half a circle, into the pharyngeal orifice of the Eustachian tube. If there is no malformation and the velum palati is not so tense as to displace the tip backward, it will enter the orifice, where it should be held during inflation.

The fixation of the catheter, after it has been properly introduced into the pharyngeal orifice of the Eustachian tube, is most easily accomplished by grasping the free end between the thumb and the forefinger, while the other fingers rest across the bridge of the nose.

The auscultation or diagnostic tube (Fig. 403) should be used to determine whether the catheter is in place. The statements of the patient on this point are not trustworthy, as the sensation produced by inflation often gives rise to a feeling of fulness in the ears when the auscultation tube does not confirm the patient's statement. The physician should make a common practice of using the auscultation tube when inflating the ears, not alone to judge whether the procedure is successful, but to enable him to determine the condition of the Eustachian tube and the middle ear. If there is a soft, blowing murmur, the tube is normally open, although it may be normally inflated and the murmur not heard. This is exceptional, however, and the fact of inflation can be demonstrated

by using the manometer tightly fitted into the external auditory meatus. The U-shaped tube of the manometer should contain a few drops of colored fluid, which will rise in the outer arm of the manometer tube during inflation. If the Eustachian tube is obstructed by catarrhal swelling or hypertrophy of the mucous membrane, the character of the sound during inflation becomes sibilant and rough. The presence of mucus in the tube is indicated by moist bubbling rales. It occasionally happens that at the beginning of inflation there are signs of obstruction, which after a few moments suddenly disappear. In these cases it is probable that a thick plug of mucus obstructed the tube and was dislodged by the operation. In atrophic otitis media the Eustachian tube is correspondingly open, and inflation gives a very soft, blowing murmur.

FIG. 403



Inflation of the middle ear through a catheter attached to a compressed air apparatus, the American method. The catheter is held in position with the left hand, though not thus shown in the illustration.

Other Methods of Catheterization.—There are several other methods of catheterizing the Eustachian tubes, not commonly used, that in exceptional cases may be resorted to.

(*a*) Catheterization from the opposite nasal cavity may be done with the ordinary catheter in those cases in which there is a narrow pharyngeal vault, by introducing the catheter along the floor of the nose in the usual way until it reaches the posterior wall of the pharynx, then rotating the curved tip toward the opposite Eustachian orifice until the ring on the outer end of the catheter stands horizontally toward the median line. The outer end of the catheter should then be removed from the septum, thus bringing the tip in approximation with the pharyngeal opening of the tube. Gentle pressure in a backward direction will bring it well into the opening. Inflation should then be practised in the usual manner.

This method may be used when there is an obstructive lesion in the nose upon the side to be catheterized and in those cases in which there is congenital occlusion of the posterior nares on that side.

(*b*) Catheterization through the mouth may be done by using an instrument with a longer curve than is ordinarily used through the nose. The postrhinoscopic mirror will be found very useful in placing the tip in the mouth of the tube. When there is cleavage of the palate the ordinary catheter may be used, as the soft palate is out of the way, thereby enabling the operator to reach the mouth of the tube with the shorter curved tip. In many of these cases the operation may be accomplished without the use of the postrhinoscopic mirror, as the pharyngeal openings may be seen with the unaided eye.

The Diagnostic and Therapeutic Value of Catheterization.—There are various methods of forcing air through the catheter into the middle ear, all of which are of value, the choice of method depending largely upon the mechanism afforded by the local instrument dealers rather than upon the peculiar merits of any individual method.

(*a*) The Politzer bag, shown in Fig. 405, is connected directly with the Eustachian catheter, and is, perhaps, the most familiar apparatus for this purpose, owing to the reputation of its distinguished inventor. It is admirably adapted to the use of general practitioners on account of its simplicity and the slight expense.

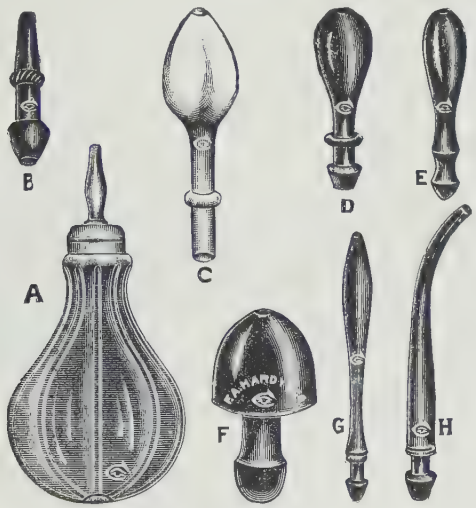
(*b*) The equipment of a modern American otologist, however, usually affords appliances which are even more convenient, and perhaps more scientific in their application in office practice than the Politzer bag. Many offices in the large cities now have compressed air piped through the building, and with a gauge the desired pressure can be obtained for each individual case. An equipment of this character is admirably adapted to the purposes of the otologist, and renders the work of inflation more exact and scientific in its application. The shut-off should be applied to the expanded end of the catheter after it is properly adjusted, and inflation accomplished by liberating the air by means of the lever, as is done in spraying the nose and throat (Fig. 403). The exact amount of air pressure can be accurately estimated by the pressure gauge. The author uses the regulator attached to the compressed-air tank devised by Edwin Pynchon. It is so arranged that the amount of air pressure can be quickly adjusted to the needs of the case. A pressure of from seven to twenty-five pounds is all that is ordinarily required for the inflation of the middle ear. In some cases a pressure as low as five pounds is quite adequate for the purpose.

(*c*) The nebulizing inflator is an instrument whereby inflation can be performed through the catheter in a very simple and easy manner. The tip of the nebulizer is made to fit into the expanded end of the catheter, and the medicated nebula is driven through the catheter into the middle ear. The impact of the medicated air thus released passes through the tube and the catheter to the middle ear. This appliance affords a convenient and simple means of applying medicated vapors.

The diagnostic tube should be used in connection with these methods, and the character of the sounds transmitted through it noted for diagnostic and prognostic purposes.

Politzer's Method.—In 1863 Politzer[1] introduced a method of inflating the middle-ear cavities which still proves of the greatest utility in aural practice. It is performed with a pyriform rubber bag (Fig. 404), of about ten ounces' capacity, to which is attached a nozzle suitable for introduction into the anterior nares. The patient is seated in front of the operator, the nozzle inserted well into one nostril, while the opposite nostril is firmly closed. The index and middle fingers of the operator's left hand should engage the tip of the nose, while the thumb completes the closure of the nostrils. The patient is then instructed to swallow, and as the laryngeal box is observed to rise, the bag is forcibly compressed with the operator's right hand. The nozzle and the operator's fingers completely close the anterior nares, while the act of swallowing brings the muscles of the soft palate and of the posterior wall of the

Fig. 404



Politzer's bag and tips.

pharynx into apposition, thus completely walling off the respiratory path in that direction. The compressed air thus confined finds the point of least resistance *via* the Eustachian tubes, and is conveyed to the middle ear and inflation accomplished. The method is simple, the instruments of simple construction and slight expense, and the procedure is easily performed. The act of swallowing, if performed more than once or twice, becomes quite difficult for the patient unless aided by the use of a sip of water.

Miot introduced a simple expedient which in some respects is more convenient than water. Sugar lozenges are kept on the treatment table, and one given to the patient before performing inflation. As the lozenge is dissolved in the mouth of the patient the act of swallowing is easily

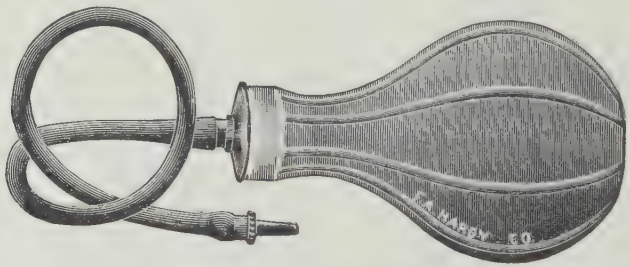
[1] Wiener med. Wochenschrift, No. 6.

and naturally performed as often as necessary without the inconvenience attending the use of water. The tubes may also be rendered patulous by uttering the sounds, och, k, king, kick, and by forcibly blowing out the cheeks.

The author, in using the Politzer bag, places a piece of soft-rubber tubing, one foot long, between the tip of the bag and the nozzle (Fig. 405). By this measure the liability of mechanical injury to the mucous membrane of the nose when forcibly compressing the bag is avoided, and the hand of the operator has great freedom of movement within a circle of twenty-four inches' diameter.

Auscultation during the use of the Politzer method shows two sets of sounds: one due to the entrance of air into the middle-ear cavity, the other to the escape of air in the epipharynx. The former is a soft, blowing murmur when the drumhead is intact, while the latter is rough, loud, and gurgling in character. After a little experience the tympanic sounds may be readily distinguished from the rough pharyngeal noises, and the latter are soon disregarded altogether. If for any reason the

Fig. 405



Politzer's bag and tube for use with a Eustachian catheter or nasal tip.

tympanic murmur is not heard, the use of the manometer tube should be resorted to in order to determine whether the air is forced into the middle ear.

It sometimes happens that inflation cannot be performed by Politzer's method, in which event the use of the catheter is usually indicated.

A Modified Politzer Method.—*The American Method.*—The author uses a modification of Politzer's method, in which the rubber bag is discarded and the compressed-air apparatus substituted. It is not only a more convenient, but also a more exact method of inflation. A suitable nose-piece adapted to receive the tip of the shut-off of the air tank tube, such as is used with spray bottles, comprises the outfit. The Buttles-Pynchon inhaler is one of the best for the purpose, as it is constructed to be used with the ordinary shut-off of a compressed-air apparatus. It is a Pynchon modification of the Buttles inhaler, in which the acorn-shaped nose-piece unscrews at about its middle portion (Fig. 399), thus affording an easy means of introducing pieces of sponge, gauze, felt, or cotton-wool upon which volatile medicaments may be dropped and blown into the tympanic cavity. By means of the com-

pressed-air tank with a pressure regulator, the exact amount of air pressure needed to inflate the ear may be established for each case at the time of the primary examination. This should be made a part of the record, and utilized in the future treatments. If it is found after a few treatments that inflation is accomplished with less air pressure than was at first required, a favorable prognosis may be given. The great advantage of this method over Politzer's is the fact that the amount of pressure used can be accurately estimated, regulated, and recorded. This method should be adopted in all modern offices, but for bedside practice and for home use the Politzer bag still holds a distinct and useful place in otological practice.

Thomas Hubbard has also devised an ingenious compressed-air apparatus for the graduated and scientific regulation of the air pressure in tympanic inflation. His apparatus is also provided with an air filter.

External Mechanical Massage.—In the hands of the author external mechanical vibration below the angle of the inferior maxilla has proved a valuable adjunct to the inflation of the middle ear. In some cases which resist successful inflation, mechanical massage applied in this region with the vibrator will bring about the desired result. The mechanical vibration thus imparted probably lessens the passive congestion of the mucosa of the pharynx, tonsils, and faucial pillars, and thus favorably influences the mouth and the lumen of the Eustachian tube.

Comparative Value of the Methods.—It may be said that no one method should be used to the exclusion of all others. Each will, under certain circumstances, answer the purpose better than another. The conditions favorable to the employment of any method cannot always be foreseen, but can only be ascertained by trial. The author has often found it impossible to inflate by catheterization when he could do it readily by the Politzer method, or *vice versa*. He has also found the Politzer method inadequate in some instances in which the modification described by the author, using the compressed-air tank and a nose-piece, did the work satisfactorily.

Valsalva's method is commended on account of its simplicity and the absence of instruments of any kind in its performance. On the other hand, it is to be strongly condemned on account of the ease with which it may be abused. It is done entirely by the patient, and the relief it affords may tempt him to resort to its use much oftener than is necessary or safe. There are few cases requiring inflation oftener than once on each alternate day for a period of six weeks. With Valsalva's method the patient often inflates his ears several times daily for many weeks or months, thus producing pressure atrophy of the drumhead. When this condition arises, the state of the patient's ears is worse than before treatments were given.

Catheterization is regarded by many as the most effective method of inflation yet devised. In the author's experience, a louder tympanic murmur is heard by this than by any other method. He believes, therefore, that where it can be used without great discomfort to the patient it should be given preference. However, there are certain nasal deformi-

ties which may prevent, or at least greatly hinder, its successful use. Some other method, preferably the tank and nose-piece, should then be used. Politzer himself claims more for his method than for any other, not excepting catheterization.

The Politzer method is extensively recommended and used on account of its simplicity and the ease with which it is practised. In those cases in which the catheter cannot be used, as in marked nasal obstruction, hypersensitiveness of the mucosa, timid patients, and children, it should be elected as preferable to catheterization.

Unless the diagnostic (auscultation) tube is used, the operator is never certain of the results obtained by any method whatsoever, the patient's statements often being untrustworthy.

The modified Politzer method, in which the compressed-air tank takes the place of the rubber bulb, is ordinarily preferable to the Politzer method, as the pressure can be accurately regulated to suit each case. The tympanic murmur is louder and is heard much longer and more continuously on account of the constant air pressure than with the short puff obtainable with the Politzer bag. The author believes, however, that where catheterization can be done with little discomfort to the patient, it should be given preference.

Recapitulation. –1. Catheterization is the most effectual method of inflation in most subjects.

2. The compressed-air tank and nose-piece are preferable if for any reason catheterization cannot be performed.

3. The Politzer method should be used in bedside practice and as a "home treatment," and in all other instances in which the compressed-air apparatus and nasal tip are not available.

4. Valsalva's method should only be recommended when the others are not available, and then only with strict instructions as to its possible evil results if the directions as to the frequency and period of use are not strictly followed.

CHAPTER XLI

INFLAMMATORY DISEASES OF THE TYMPANUM

ACUTE CATARRHAL OTITIS MEDIA

Acute catarrhal otitis media comprises about 13 per cent. (Hovell) of all ear diseases; it is, therefore, a very important division of otology, and should be considered in some detail, especially in view of the fact that the general practitioner is so frequently called upon to treat it.

General Etiology.—The causes of simple catarrhal otitis media are numerous, and may be considered under three different headings, namely:

1. *Exciting causes,* or pathogenic microörganisms.
2. *External influences,* or those conditions external to the body which act as predisposing causes.
3. *Internal influences,* or those conditions within the body which predispose to otitic inflammations.

1. **Exciting Causes.**—The exact relation of *microörganisms* to the inflammation of the middle ear is not yet fully determined. That they are found in healthy ears is probable, as the investigations by Zaufal have shown them to be present in the ears and epipharynx of rabbits. We know that the various infectious fevers, as scarlet fever, measles, diphtheria, etc., are often accompanied by acute catarrhal otitis media, although complications from these sources are very prone to take on the suppurative type. There is no special bacteria which causes catarrhal inflammation of the middle ear, but there is usually a combination of two or more, such as the Diplococcus pneumoniæ and the Streptococcus pyogenes. The Staphylococcus pyogenes albus and aureus, and the Bacillus pyocyaneus are next most frequently found in the middle ear. Friedländer's bacillus is less frequently found in combination with the Staphylococcus cereus albus, Bacillus pyocyaneus, and the Micrococcus tetragenus. These and other microörganisms may be present in the tympanic cavity without exciting inflammation. It is necessary that the conditions of the secretions and the tissues be favorable for their rapid propagation before they are able to excite an inflammatory process It has been found that the invasion of a new microörganism is sufficient, under certain circumstances, to excite inflammation. After the inflammation has subsided, the invasion of another type of microörganism may cause a recurrence of the inflammation. The question of microörganisms in their relation to inflammatory processes is still involved in so much speculation and doubt that it is impossible to give any definite statement as to the exact influence they have as etiological agents in

catarrhal inflammations. It seems that after the primary irritation of the tissues has subsided, the soil is prepared for other germs, so that upon their entrance there is a recrudescence of the inflammatory process.

It is well known that pathogenic microörganisms are more virulent at times than at others, hence the presence of microörganisms *per se* is not sufficient to cause acute inflammation. They must be of the proper virulency, the soil must be prepared to favor their activity, and the cellular structures must be so modified in their functional activity as to be unable to resist their influence. Even the tubercle bacillus may be found in the secretions of the middle ear without giving rise to pathological changes.

Channels of Invasion.—Microörganisms nearly always gain access to the tympanum through the Eustachian tube. There are several other routes, however, through which they may enter it. The bloodvessels may carry them to the mucous membrane of the tympanum, where they may be thrown out with the serum and mucus, and thus give rise to inflammation. They may also gain access through the drumhead, when it is perforated, either from congenital or pathological states. In rare instances they may gain entrance from the cranial cavity through the bony walls, or through the internal auditory canal and labyrinth.

As has been stated, they most frequently gain entrance through the Eustachian tube. This may occur in spite of the fact that the tube is lined with ciliated columnar epithelium, whose ciliæ create a current toward the epipharynx. The Eustachian tube is patent as it momentarily opens to admit air into the tympanum, and the microbes may be swept inward with the current of air to the middle ear. This may also take place during paroxysms of sneezing or vomiting. Hence, there is no absolute physiological barrier offered by the ciliated epithelium of the tube to the entrance of microörganisms into the middle ear.

The microörganisms excite catarrhal inflammation which may assume the suppurative type. They may also be present without exciting any pathological reaction.

2. **External Influences.**—The external causes of otitis media cannot be considered without also taking into account the internal conditions which predispose to it. It is convenient, however, for purposes of study to consider the external causes separately, and in so doing we shall have to take into consideration the local conditions of the upper respiratory tract, as well as certain constitutional states which will be considered in detail under the second type of general causes.

Exposure to the weather is a fruitful predisposing cause of otitis media, especially when the tone of the system is not up to the normal standard. If the patient has chronic rhinitis or obstructive disease of the nasal cavities, or has adenoids and epipharyngeal inflammation, exposure to the inclemencies of the weather is especially liable to result in acute catarrhal inflammation of the middle ear. Certain other factors enter into this proposition, as clothing, climate, zone, age, sex, and the occupation of the patient.

It seems appropriate, therefore, that these etiological factors should be

considered under this heading, rather than under separate paragraphs. It is evident that the effect of exposure to the weather will depend very largely upon the amount and kind of clothing worn, and the climate and latitude in which the patient lives, as well as upon his occupation. Age and sex will, also, largely determine this effect. The character and amount of clothing worn does not, *per se*, determine the influence that exposure to the weather will have upon the patient, as the habits of the individual and the character of the house in which he lives modify his susceptibility to such exposure. If he lives in a house that is but partially heated, and has been accustomed to sleeping in a bedroom which was never heated, the exposure to the inclemencies of the weather will not affect him as much as it will one who lives in a well-built house which is uniformly heated.

Many of our country homes are so loosely constructed that they are well ventilated through the crevices about the windows and doors. There is not, therefore, the extreme difference between the conditions indoors and outdoors found in the better portions of the large cities.

Those living in country houses are subjected to a more even temperature and atmosphere, within and without the house, than those who live in closely built and better heated houses. They are, therefore, not so susceptible to changes of the weather, and the amount of clothing they wear, when exposed, need not differ so much in quantity and character from that worn while indoors.

I have known patients accustomed to country life, who were exposed to the inclemencies of the weather a hundred times more than they were in after years when living in the city, to be entirely free from catarrhal conditions of the nose and ears while living in the country, and rapidly develop them after removing to the city.

The catarrhal inflammation developed, in spite of the fact that they were taking extraordinary precautions, in the way of *additional clothing*, to protect themselves while outdoors. It seems, therefore, that the habits of life which tend to lower cell vitality have more to do with the predisposition of the upper respiratory tract to catarrhal inflammation than the amount or character of clothing worn. Our modern dwellings with their superb heating plants, storm windows, etc., are, perhaps, less of a boon to humanity than is generally supposed. The more primitive style of living seems to accustom the system to the variations in the temperature and hygroscopic conditions of the atmosphere. It is not reasonable, however, to expect that we will return to that mode of living. We can only say in this connection that in the construction of our houses more attention should be given to the question of ventilation. It has been said that good ventilation and cheap heating do not go hand in hand. Within certain limits this is undoubtedly true. Nevertheless, the architect can do much toward the proper ventilation of dwelling houses without materially increasing the expense of heating.

The attention of the public should be frequently called to this fact until they are educated up to the point that they will demand that this problem receive appropriate attention at the hands of the architects.

The *climate* and *latitude* in which one lives influence, in a marked degree, the character and amount of exposure to which he is subjected. In the temperate zone the climate is usually variable and subject to very rapid changes in temperature and hygroscopic conditions of the atmosphere, and is, therefore, one of the factors in the etiology of acute inflammations of the upper respiratory tract and middle ear. Those living in the more frigid and torrid zones are less exposed to sudden changes in the temperature and atmosphere, and are, consequently, less subject to catarrhal inflammations. Those living near large bodies of water, as the ocean, or the chain of Great Lakes between Canada and the United States, are especially affected by climatic conditions, as the atmosphere is moist and penetrating. The skin is thereby chilled and the vasomotor nervous centres are disturbed, and many of the functions of nutrition and metabolism are modified in such a way as to excite inflammatory processes in the mucous membranes, especially those of the respiratory tract.

Certain *occupations* give rise to greater exposure than others, consequently *sex*, which largely determines the nature of one's occupation, must have some influence in the etiology of this disease. A greater proportion of *males are exposed* to the inclemencies of the weather; hence, catarrhal inflammation of the mucosa is more common with them than females.

Age also determines, to some extent, the amount of exposure. Young male adults in the vigor of life, full of ambition and enterprise, more often subject themselves to the inclemencies of the weather in the pursuit of their vocations than those who are younger or older. Hence, we find catarrhal inflammation of the middle ear and upper respiratory tract more common in young adulthood than at any other period of life.

A careful study of the above facts will demonstrate that *exposure to the weather* is a question of considerable complexity, as the effects of the exposure are largely determined by the mode of life, clothing, zone, age, sex, and occupation of the patient. It is not sufficient, therefore, for one to say to the patient, "You should not expose yourself to the inclemencies of the weather." All the facts pertaining to his mode of life should be taken into consideration, and advice given accordingly. It has become quite the fashion nowadays to tell patients that they should take a cold plunge bath each morning, and that they should walk at least five miles a day. This advice with certain limitations is sound, and is based upon the data given above. The attempt is made by this procedure to bring the patient for a brief time each day back to the primitive methods of living. It is well known that life in the *open air*, and a certain amount of exposure of the body to varying degrees of heat and cold, are favorable to the well-being of the system.

More attention should be given to this subject than is now done. The *influence of open air* upon the cellular vitality is greater, perhaps, than is generally appreciated. We know that many women work indoors all day, are constantly making physical exertion, and are anemic and

poorly nourished in spite of the fact that they have plenty of wholesome food. The same amount of exercise taken in the open air would transform them into robust, red-blooded women. Fresh air is the most potent therapeutic agent for the upbuilding of the system.

3. **Internal Influences.**—The internal conditions which predispose to catarrhal inflammation of the middle ear and upper respiratory tract have a more intimate clinical relationship to acute catarrhal otitis media than the external influences. It is well established that middle-ear disease is almost invariably preceded by some form of nasal or epipharyngeal disease. Whatever causes the preëxisting infection and inflammation of the nasal mucous membrane or the mucosa of the epipharynx will also directly or indirectly lead to a similar condition within the Eustachian tube and middle ear. This is easily accounted for when we remember that the mucous membrane of the Eustachian tube and middle ear is a continuation or reflection of that lining the nose and epipharynx. It is quite similar in physiology and structure, and inflammations therefore readily extend from one part of it to another. If there is a difference in the structure of the mucous membrane, as in the mesopharynx, where the epithelium is squamous, the inflammatory process does not readily extend to that part. The mucosa of the nose, epipharynx, Eustachian tube, and middle ear are lined by columnar ciliated epithelium; hence, there is no bar to the extension of the inflammatory process from one to the other.

In this connection it is of advantage to briefly refer to the diseases of the nose, epipharynx, and fauces which cause inflammatory diseases of the Eustachian tube and middle ear:

(*a*) *Nasal diseases* which cause pathological processes within the middle ear are either inflammatory or obstructive in character. The inflammatory diseases are acute rhinitis, acute fibrinous rhinitis, diphtheritic rhinitis, syphilitic rhinitis, tuberculous rhinitis, and catarrhal and suppurative sinuitis. The inflammation may extend to the middle ear through the Eustachian tube by continuity of tissue, or the pathogenic bacteria may invade the ear through the Eustachian tube or through the blood and lymph channels. They also influence the inflammatory changes in the middle ear by causing the closure of the Eustachian tube, thereby interfering with the ventilation of the tympanum. The oxygen is gradually absorbed from the middle ear, thus gradually rarefying the air. The blood within the vessels of the mucosa of the middle ear rushes in to fill the partial vacuum thus created, and congestion and engorgement of the mucous membrane follow. This leads to changed nutrition of the parts and to a disturbed relationship of the cellular structures, which after a time predisposes to an inflammatory process.

Nasal obstruction is also a fruitful source of ear disease. The presence of spurs, ridges, thickening, and deflections of the septum, and enlargement of the middle turbinate (see Vicious Circle of the Nose) cause stenosis of one or both nares or obstructs the ostia of the sinuses. As the nasal cavities are the natural channels for the respiratory and expiratory currents of air, any interference with their patency results

in physiological disturbances of a very pronounced character. When the diaphragm contracts, the thoracic cavity is enlarged and the air from without rushes in to fill the increased space. If the nasal chambers through which the air enters the respiratory tract are obstructed, the contraction of the diaphragm acts as the valve in a syringe when it is forcibly pulled out; the air is thus rarefied posterior to the point of obstruction. The partial vacuum thus created is attended with the rush of blood to the vessels of the mucosa. This condition after a time leads to tissue changes and predisposes to inflammatory processes. The patency of the Eustachian tubes is thereby diminished, which still further affects the middle ear. Hence, nasal and sinus obstruction is a constant menace to the middle-ear cavity.

All cases should be carefully examined for any diseased state of the nose, as the subsequent treatment of the case will depend very largely upon the successful treatment of the nasal conditions.

Ethmoiditis and sphenoiditis are a fruitful source of middle-ear inflammation. The morbid secretions from these cells flow into the epipharynx and excite an inflammation which in time extends by continuity of tissue to the Eustachian tube and middle ear.

(*b*) *Epipharyngeal diseases predisposing* to middle-ear catarrh may be studied under two headings; namely, postnasal adenoids, or neoplasms, epipharyngitis and adhesive bands in Rosenmüller's fossæ. The presence of postnasal adenoids in the vault of the pharynx gives rise to epipharyngitis, either of the catarrhal or suppurative type. For reasons already given, this inflammatory process may give rise to middle-ear inflammation. Postnasal adenoids may be so situated as to close the mouths of the Eustachian tubes, a common cause of middle-ear catarrh.

(*c*) *Enlarged* or *diseased faucial tonsils* have for many years been recognized as one of the principal etiological factors in the production of middle-ear disease. This relationship is readily understood when we remember that the tonsils are situated between the anterior and posterior pillars of the fauces (glosso- and pharyngopalatine arches). The posterior pillar embraces the palatopharyngeus muscle, which has some influence in controlling the patency of the Eustachian tube. It is apparent that when the tonsils are diseased the pillars are congested or inflamed and in time their muscular fibers undergo more or less degeneration and atrophy.

(*d*) *Tubal disease*, while intimately associated with middle-ear disease in nearly every case coming under observation, may be present without a similar process in the middle ear. In other words, there is a time when the inflammation extends from the epipharynx into the Eustachian tube, and does not yet involve the middle ear. Reference has already been made to the fact that congestion or obstruction of the Eustachian tube is a fruitful source of inflammatory diseases in the middle ear, and need not be dwelt upon at greater length in this place.

(*e*) Constitutional disorders, as anemia, scrofula, syphilis, and tuberculosis, lower the vitality and thus predispose the middle ear to inflam-

matory attacks. This has already been referred to under the external causes of otitis media.

After all that has been said as to the causes of otitis media, we may go back to the primary statement that those influences external to the body which, under varying circumstances, affect the vasomotor system, and certain diseased states of the epipharynx, cause obstruction of the Eustachian tube and subsequent infection and inflammation of the middle ear.

Pathology.—The cavum tympani contains serum admixed with mucus in varying proportions. Epithelial cells are also found in the secretion. They show evidence of having undergone degenerative changes peculiar to inflammatory processes. While the secretion cannot be said to be suppurative in character, it may contain a number of pus corpuscles. The mucous membrane of the middle ear, unlike that of the nose, has very few glands; hence, the mucus is formed from the chalice of goblet cells of the mucosa. In the nose the mucus is chiefly formed by the cells lining the glands, only a few goblet cells participating in its production. There is, therefore, in the middle ear a very rapid degenerative process (mucoid degeneration) going on during the acute inflammatory process. The intercellular spaces are filled with fluid, while the bloodvessels are very much congested, thus rendering the membrane very much swollen and thickened. The surface of the mucous membrane is denuded of epithelium in patches. Hovel calls attention to the fact that leukocytes are found mingled with the secretion in the immediate region of these patches.

Pronounced destructive processes are not commonly present in this type of middle ear disease. In rare instances the drumhead is perforated, while there is more or less maceration of the mucous membrane lining the tympanic cavity. After a few days the morbid changes described above rapidly disappear, the mucous membrane returning to its normal condition. There remains, however, a peculiar susceptibility to recurrent inflammations. This may be due to the fact that microörganisms of the proper virulency gain entrance to the cavity and, finding the soil prepared by the primary inflammatory process, readily excite a recurrence of the inflammation.

General Symptoms and Diagnosis.—Acute otitis media is usually due to a bacterial infection *via* the Eustachian tubes, though it occasionally enters *via* the blood current. The exudate may be simple or purulent. In simple catarrhal inflammation the drumhead rarely ruptures, no matter how intense the inflammation may be. If the exudate is purulent there is a tendency to rupture at the point of greatest bulging. Severe simple catarrhal cases begin with the same constitutional disturbances present in severe purulent cases, namely, chills, fever, vomiting, and prostration. It is often quite difficult to differentiate between acute non-suppurative and acute suppurative otitis media, until the drum membrane ruptures. Both types of inflammation are due to infection, one undergoing resolution before suppuration, and the other passing into the suppurative stage.

Intracranial complications never occur in acute non-suppurative otitis media, and somewhat rarely in the acute suppurative variety. Such complications occur more often in the chronic type, with acute exacerbations.

The exudate has a tendency to become organized into adhesive fibrous bands, hence it is very important that their absorption should be hastened as much as possible. The air douche, by means of the Politzer bag and the catheter, should be used to clear the middle-ear cavity of the exudate, or at least to spread it over a larger surface, thereby reducing the amount of exudate at any one point. The inflations should be repeated from time to time until the ear is free from the exudate, as shown by the auscultation tube. According to Edwin Pynchon, the use of the continuous air douche through a Eustachian catheter will abort acute otitis media. A pressure of about five pounds is required for this purpose. The compressed-air tank should be adjusted to this pressure and the current of air passed through the catheter into the tube and middle-ear cavity.

Infants often have acute otitis media of very short duration, probably of pneumococcal origin. Intestinal disturbances in infants are often accompanied by ear infection, and an examination of the ear should always be made. The exanthematous fevers of childhood are common causes of middle-ear infections, which in later years result in many deaths from meningitis, sinus thrombosis, brain abscess, etc. Great pains should be taken in these diseases to keep the nose and epipharynx clean during the fever. Scarlet fever and measles are especially destructive in this way. Diphtheria more rarely invades the middle ear.

Acute tuberculous otitis media is seldom accompanied by pain. This is in striking contrast to other types of acute infection. If an acute tuberculous otitis media begins with pain and other symptoms peculiar to the ordinary acute suppurative otitis media, the prognosis is much more favorable than in the non-painful variety.

Acute otitis media occurring during diabetes is not of diabetic origin. The occurrence of the two diseases is accidental. The diabetic disease, however, gives rise to constitutional disturbances which favor the long continuance of the ear discharge.

Neglected cases of chronic catarrhal otitis media result in shrinking and atrophy of the mucous membrane, or adhesions may form, thus causing permanent loss of hearing. The deposit of lime salts or adhesive processes may fix the ossicles or bind them to the contiguous walls of the cavum tympani.

Symptoms.—The symptoms of this disease vary according to the period of time which has elapsed since the onset. At the beginning they are much more pronounced than they are after a few days, when the more acute inflammatory process has begun to subside.

1. The onset of acute otitis media is usually signalized by a slight chill, which is quickly followed by a temperature ranging from 99° to 102°. The fever is, however, of such slight character in most cases that the attention of the patient is not usually attracted to it. The symptom

which quickly develops, and which should demand the attention of the attending physician, is the *pain*, which may be characterized as a dull, boring, aching sensation, or it may assume a more acute type, and become excruciating in its intensity. It is usually intermittent or throbbing in character, synchronous with the pulse beat at the wrist. It is due to the great swelling of the drumhead and mucous membrane of the middle ear, whereby the sensitory nerve filaments are put "on the stretch" with each arterial pulsation. It may also be due to the bulging of the drumhead outward into the meatus. There is a great amount of intercellular fluid thrown out at this stage of the disease, which together with the congestion of the bloodvessels renders the mucous membrane and drumhead very much thicker than normal.

In the first stage the *drumhead* is very red and thickened, and the *handle of the malleus* obscured from view. Its surface may present the appearance of a piece of raw beefsteak, except that it is more velvety in its texture. The drumhead may or may not bulge into the external meatus, depending upon the amount of secretion within the middle ear.

If the middle ear is filled with exudate, the drumhead is of necessity pushed outward. If, however, it is only partially filled, it may remain in its normal position or even be retracted.

Auricular tenderness is sometimes present, especially over the tragus. The mastoid process may or may not be tender upon percussion or pressure. Pressure over the mastoid antrum nearly always elicits tenderness, though it may be slight.

Bone conduction is increased on the affected side. The lower tone limit is lost, while the upper tone limit is not affected in those cases in which the labyrinth is not involved. If the disease is unilateral, the Weber experiment lateralizes to the affected side. The Rinné test is usually negative in character. By the term negative, I do not mean that it shows nothing, but that bone conduction for the tuning fork over the mastoid process is longer than by air conduction when the fork is held near the external auditory meatus. If the labyrinth is involved, bone conduction is diminished, and the Weber test shows the sound lateralized to the unaffected ear, while the Rinné test gives a positive sign. Labyrinthine involvement is, however, very rarely present in simple catarrhal otitis media.

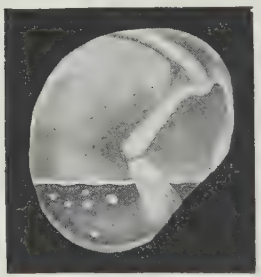
2. The second stage of this disease is characterized by the disappearance of the pain, fever, and redness of the drumhead. The congestive phenomena are lessened in intensity, hence the drumhead and mucous membrane are less thickened and swollen. The drumhead, instead of being beefy or purplish red in color, is yellowish or greenish in tint. The change in color may be explained by the fact that there is less blood in the drumhead, and the pale, slightly greenish secretion in the middle ear is seen through it. The greenish-yellow color often gives rise to the idea that there is pus in the middle ear.

Another symptom of considerable significance is the presence of a *dark wavy line* (Fig. 406) extending in a nearly horizontal direction

across the drumhead. This line, which is 1 to 2 mm. in thickness, is due to the peculiar refraction of light at the junction of the viscid secretion and the air in the tympanic cavity. If it is below the umbo, it is usually concave on its upper surface; whereas if it extends above the umbo, it is usually composed of two concave surfaces. The line will be higher or lower on the face of the drumhead according to the amount of secretion in the middle ear. If the middle ear is completely filled, the line will not be visible.

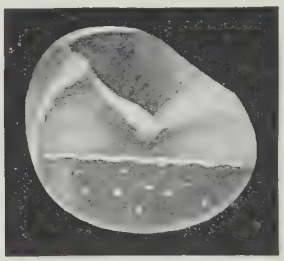
The *position of the head* determines the direction of the line, as the fluid gradually seeks the level of the new position (Fig. 407). The viscid nature of the secretion and the narrowness of the tympanic cavity interferes with the rapid change in the position of the secretion. The line is often not visible, on account of the great thickness and congestion of the drumhead.

Fig. 406



Right membrana tympani, showing mucus secretion and air bubbles after tympanic inflation.

Fig. 407



Right membrana tympani with mucus secretions and air bubbles after tympanic inflation, the patient having just arisen from the prone position.

Another symptom is the presence of *oval* or *round rings* (Figs. 406 and 407), which are due to the air bubbles in the viscid mucus. They may extend above the dark line, described above, or they may be within the field of the mucus itself. They may be single or multiple. After tympanic inflation the line disappears, while the entire field of the drumhead is occupied by the air bubbles. After several hours they will, in part, disappear, and the line will return.

Aural auscultation, if used during the process of tympanic inflation, shows the presence of moist rales, due to the air passing through the viscid mucus. They are very different in character from the soft, blowing murmur heard during inflation of the normal ear.

The first inflation may not be successful, as the Eustachian tube is filled with viscid mucus; hence, it should be repeated several times. The diagnostic tube should always be used in performing tympanic inflation.

The membrana tympani may or may not bulge into the auditory meatus, as this depends upon the amount of secretion within the middle ear. When it bulges into the meatus it is a positive indication that

paracentesis, or incision of the eardrum, should be performed. To neglect this subjects the patient to unnecessary pain and to spontaneous perforation of the membrane. Spontaneous perforation should not be allowed to occur, as the perforating process is due to necrosis. Not only is irreparable damage thus done to the drumhead, but other parts are subjected to pressure and to possible ulceration and necrosis.

Incision of the membrana tympani should, therefore, be done early, to prevent great destruction of tissue and to promote the reaction of inflammation. The incision does not result in scar tissue, which usually follows spontaneous rupture of the drumhead.

It should be made at the most bulging portion, and should be crucial or V-shaped in character and from $\frac{1}{8}$ to $\frac{3}{8}$ inch in length. Simple paracentesis, while often recommended, is not sufficient for free drainage of the tympanic cavity. If the incision is made straight and the drumhead is tense, the aperture for the discharge of secretion is very small, while the crucial or curved incision forms a slight flap, which permits a larger opening for the discharge of the tympanic contents.

Bone conduction is increased and the *Weber* and *Rinné experiments* give the results described under the *onset* of the disease.

Prognosis.—This is favorable or unfavorable according to the period at which treatment is instituted. If the case is seen early and appropriate remedies are used, favorable results will follow in nearly all cases. If, however, the case is allowed to run on for some time before treatment is commenced, changes of considerable importance may have taken place, such as adhesion of the contiguous parts, and ulceration in the superficial portions of the mucous membrane, the prognosis is not so favorable.

There are certain conditions which render the prognosis less favorable, as syphilis, tuberculosis, anemia, etc. It is obvious that if the diseases of the nose, epipharynx, and fauces, which predispose the patient to the primary attack, are present, there will be greater difficulty in effecting a favorable termination of the disease, and when it seems to have been cured there may be recurrences.

The *duration* of the acute type varies from one to six weeks, although in some cases it may be aborted in one or two days. The pain, which is one of the first symptoms to appear, is also one of the first to subside. Then the redness of the drumhead and the swelling of the mucosa, after which the hearing power begins to return. Later the tinnitus passes away. This symptom, however, often remains for several weeks, and in those cases which merge into the chronic form it may become a permanent symptom.

Treatment.—There are several influences to be considered in the treatment of acute catarrhal middle ear inflammation, as the causes are various and sometimes quite complicated. We are often called upon to relieve the patient of the pain or even of the acute inflammatory process, but we are not so frequently asked to treat the conditions which, if removed, would prevent a recurrence of the disease. This cannot be done without giving attention to the nasal, epipharyngeal, and faucial condi-

tions which are largely responsible for the middle-ear inflammation. The treatment should, therefore, be addressed to the relief of the acute inflammatory process in the middle ear and the upper respiratory tract in general, as well as to the complete removal of the morbid conditions of the nose, epipharynx, and fauces. The first duty of the attending physician is to allay the pain as quickly as possible.

General or hygienic treatment should first of all be considered, as the proper care of the patient will largely influence the progress of the disease. He should be kept in the house during the acute stage, and if fever is present he should remain in bed. The room should be well ventilated and exposed to sunshine. His food should be simple and nourishing, such as is usually given to bedridden patients. The bowels should be regulated with calomel and saline cathartics, while alcoholic beverages and tobacco should be forbidden. A light pledget of cotton should be kept in the external meatus to protect the drumhead and the middle ear from air currents.

Pain, being the most prominent subjective symptom, should receive appropriate treatment at once. It is often so excruciating that the patient is very restless. A mixture of equal parts of carbolic acid, glycerin, and the hydrochlorate of cocaine may be dropped into the external meatus, where it will, in most cases, afford relief within a few minutes. A mixture of laudanum and oil in the external meatus is not of very much value. The mixture is usually warmed in a teaspoon before use, and if there is any virtue in it at all, it is due to the warmth or protection it affords to the exposed and inflamed membrane.

Another remedy of value for the relief of pain as well as of the congestion is a 12 per cent. solution of carbolic acid in glycerin (Andrews). While this solution does not have as great anesthetic power as the one above recommended, it nevertheless aids materially in allaying the pain.

The author has often used the fumes of chloroform as a relief. There are a number of ways in which this may be applied, perhaps most conveniently with a pipe, in the bowl of which there is a small piece of cotton upon which a few minims of chloroform are dropped. The stem of the pipe should be placed to the meatus, while the bowl is placed to the mouth of the operator.

The fumes thus gently blown into the external auditory meatus usually afford relief in a very few seconds or minutes. Leeches applied to the tragus, or posterior to the auricle, also relieve the pain and promote the reaction of inflammation.

Cold may be applied over the ear, although the effect is neither good nor pronounced. Hovell recommends the use of blisters by means of plasters over the mastoid process, though they are liable to produce ugly sores. Their value is due to the fact that they promote the reaction of inflammation, but there are other remedies which are more efficacious and which do no harm, such as the leukodescent light from a 500 candle-power lamp.

Tympanic Inflation.—During the last few years the literature has shown a partiality for the use of glycerin and carbolic acid for the cure

of acute middle-ear inflammations. The remedy is a valuable one, but it does not meet all the indications, especially those which arise from the great tumefaction and adhesive processes. It is important that tympanic inflation be performed at frequent intervals, as the increase of the air pressure within the middle ear separates the inflamed surfaces. In this way *adhesions are prevented*, or, if formed, are broken down and a long train of symptoms and impairment of the auditory function, so often seen in the dry or adhesive types of chronic ear disease, are averted. The inflation also serves a very useful purpose in freeing the tympanic cavity from secretions and in maintaining the patency of the Eustachian tubes.

If the drumhead is very red and swollen, and there is great pain, the air douche should be used with great caution, as there is danger of perforation. Inflation should be chiefly limited to the second stage of the disease, and should be performed at frequent intervals. The patient should be provided with the Politzer air bag and instructed in its use. The frequency with which it should be used depends upon the rapidity with which the secretions are formed. In ordinary cases it should be used at intervals of one to three hours. In this way the tympanic cavity and Eustachian tubes are kept free from secretions. The hyperemia is reduced by the increased air pressure, and the adhesions between the ossicles and tympanic walls are prevented.

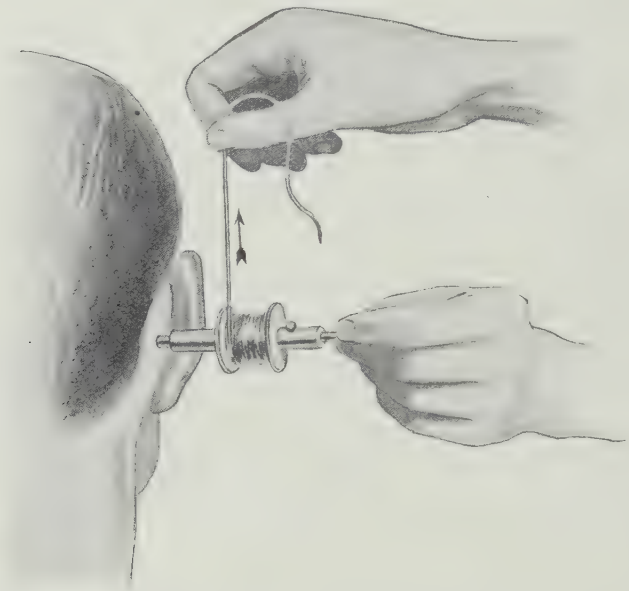
Inflation is most effective when performed through the Eustachian catheter, but this, of course, can only be done by the attending physician. The mucous membrane of the tube is usually quite swollen, and inflation difficult to accomplish on this account. Dr. Sidney Yankauer advises the preliminary use of cocaine to reduce the swelling. He first passes a standard catheter of virgin silver, through which he introduces an applicator wound with cotton saturated with a 5 per cent. solution of cocaine. The applicator is introduced as far as the isthmus or further, say 20 to 25 mm. beyond the tip of the catheter. It is advanced by stages every few minutes as the swelling subsides. A 25 per cent. solution of argyrol is then applied in the same manner. A single treatment, if applied before the membrana tympani bulges, or pus forms, is often followed by an immediate cessation of the middle-ear inflammation. If this does not occur the tube is rendered more patent and inflation can be effectively performed.

Leeching over the mastoid process and in front of the tragus is often attended with prompt and marked improvement. There is no other remedial measure that acts as promptly, and it would be a distinct advantage if leeches were used more frequently than they are at present. The artificial leech, as shown in Figs. 408 and 409, may be used instead of live leeches if desired.

Pneumomassage is a valuable adjunct to the treatment of the later stages of acute inflammations of the middle ear. During the acute or first stage it cannot be used on account of the pain and great swelling present, but later it is valuable, as it lessens the vascular and lymphatic engorgement of the tissues and prevents ankylosis of the ossicles. The

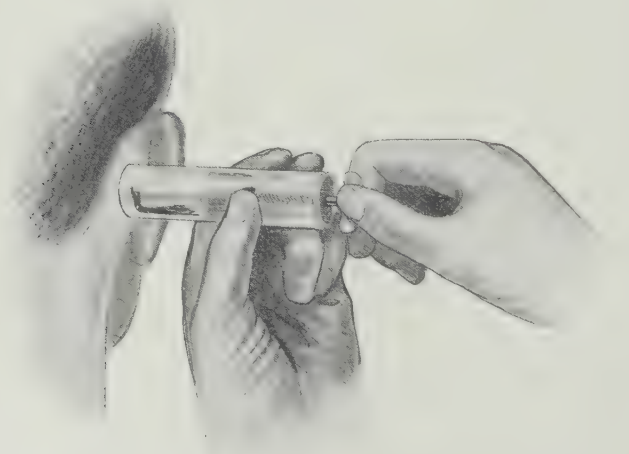
form of pneumomassage best adapted for use in these cases, at least in the secondary stage, is alternating compression and rarefaction of

FIG. 408



The application of the artificial leech to the mastoid process. The cord is drawn, thus rapidly rotating the circular knife applied to the skin of the mastoid process.

FIG. 409

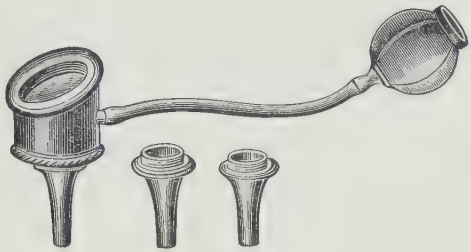


The exhaust pump withdrawing blood through the circular incision.

the air in the external meatus. With the Victor massage apparatus and the Pynchon modification of the pump (Fig. 13) any variety or character

of compression and rarefaction that may be desired can be produced. Care should be taken to adjust the piston to such a length of stroke as will cause no pain, as otherwise it may increase the inflammatory process or rupture the drumhead. The principle is the same as that relating to the use of massage in any other part of the body—namely, that it should be used with such force as not to produce contusion or injury to the tissues. If such an instrument is not available, Siegle's otoscope (Fig. 410) or the Delstanche masseur (Fig. 12) may be used. If neither of these are at hand, a simple rubber tube with a suitable metal tip, through which alternating compression and rarefaction may be produced with the mouth, will serve the purpose. These instruments have the advantage of being under the perfect control of the operator, while they have the disadvantage of imposing upon him the necessity of administering the treatment from one to fifteen minutes, as the case may require. Some otologists regard the massage machines, which are propelled by an electric motor, as being impressive pieces of machinery, which have but little actual value as therapeutic agents. The author's years of actual experience, however, with both kinds of apparatus has

FIG. 410



Siegle's otoscope.

proved that better results are obtained by the judicious use of the so-called "machines" than is possible with the hand devices. However, the hand instruments are especially well adapted for use in acute catarrhal cases, as pneumomassage is not usually applied for long periods at any one time. Pneumomassage is of little value in well-advanced adhesive processes, and in selected cases the only treatment is surgical.

ACUTE INFLAMMATION OF THE EXTERNAL ATTIC OF THE TYMPANIC CAVITY (POLITZER)

The external attic is sometimes the seat of a circumscribed acute inflammation. The exudate is thrown out into Prussak's space (Fig. 375) and partly into the spaces formed by the folds of mucous membrane between the malleo-incudal body and the external tympanic wall.

The disease is *characterized by* slight pain and deafness, with a tumor or blister-like formation on the anterior portion of Shrapnell's membrane (membrana flaccida); or if the posterior spaces are involved, the projection forms upon the posterior portion of the flaccid membrane.

Etiology.—The exciting cause of this rather rare condition is the same as in acute otitis media—namely, the specific bacteria of exanthematous fevers, epipharyngitis, and influenza. Sea bathing and cold solutions in the external canal act as predisposing causes. It is probable that the infection usually reaches Prussak's space through the Eustachian tube, although it is possible for it to pass through the Rivinian foramen.

Symptoms.—In the *mild form* there is a feeling of fulness in the middle ear, slight pain, deafness, and tinnitus. The membrana flaccida is reddened and bulging, or it may be yellow at its prominent portion. The upper wall of the meatus near the drumhead is red and slightly swollen. The membrana tensa usually appears normal. The process may run its course in a few days.

In the *severe form* the reactive symptoms are more pronounced, the hearing being temporarily more disturbed, although there is usually no permanent loss of hearing. The membrana flaccida is much more bulging, often completely covering the short process and handle of the malleus. The course in the severe form is prolonged, though it may end in complete recovery.

Treatment.—The treatment is the same as for acute otitis media and acute suppurative otitis media, except there is no need for tympanic inflation, as there is no deafness from swelling of the mucosa of the middle ear and Eustachian tube, and the tension of the membrana tensa and ossicles is not disturbed.

CHRONIC MOIST CATARRHAL OTITIS MEDIA

This disease is characterized by intermittent or remittent deafness and *tinnitus aurium*. It may follow acute catarrhal otitis media, or it may come on without any previous history of acute disease. In some cases deafness is progressive while in others it extends by leaps and bounds. The patient often makes the statement that he hears very well until after exposure, after which he is much more deaf. The acuity of his hearing is usually less during the damp, cool weather of late autumn and early spring.

Etiology. The etiology as given under Acute Catarrhal Otitis Media in a large measure applies to this disease. Therefore, a detailed statement is not given in this connection. It is sufficient to state that in most instances the chronic disease is an immediate result of the acute inflammation. This is especially true in those cases which are not treated early or in an appropriate manner. It is also especially liable to follow the acute type in those cases in which there has been previous chronic rhinitis, sinuitis, epipharyngitis, and obstruction of the Eustachian tubes. The obstruction of the tubes by adenoids, epipharyngeal catarrh, nasal and accessory sinus disease, etc., undoubtedly forms one of the chief factors in the production of the disease. (See Etiology, Acute Catarrhal Otitis Media.)

Symptoms.—Subjective Symptoms.—The chief subjective symptoms are deafness and tinnitus aurium. In addition to this, there is a feeling of fulness in the ears. Giddiness is present in a certain number of cases, but is by no means a constant symptom.

Deafness.—This is the chief symptom of the disease, and is usually the one which leads the patient to seek relief. In quite a number of cases, however, the tinnitus is so much more annoying than the deafness that relief is sought on this account. The deafness may at first be so slight and insidious in its progress that the patient is unconscious that his hearing is defective. He explains his inability to understand what is said to him by the slipshod way in which he is spoken to. It is not uncommon for such patients to feel offended when it is intimated that they do not hear well. They are very apt to reply that they can hear when they are spoken to in the proper manner. Later they notice slight subjective noises, after which it is only a question of a few months until they become conscious that their hearing is defective. In some subjects, however, the progress is not so insidious as that just described. On the contrary, it may be very rapid, then after a time seemingly remain stationary for months or years. The deafness may again suddenly become worse, and so continue throughout life. The rapid progress made is not indicative of the severity of the inflammatory process, but rather points to the fact that certain vital parts have become involved, thereby limiting the sound-conducting function of the auditory apparatus. If the changes which take place in the middle ear are limited to the mucosa of the tympanic cavity, the deafness is slighter and less rapid in its progress; whereas, if the ossicular chain and the round or the oval windows are involved in a marked degree, the deafness comes on suddenly and is more pronounced in character. It is important to bear this in mind, as otherwise it is not possible to understand why in one case of simple chronic catarrhal otitis media there is such slight deafness, while in another there is marked and sudden increase in the deafness.

Tinnitus aurium is a symptom which is almost constantly present in greater or less degree, causing the patients much annoyance. Their sleep and rest at night are interfered with. They sometimes become nervous and hysterical, and if relief cannot be afforded are apt to become morose. The noises in the head assume almost any variety of sounds or tones, ranging from simple pulsating murmurs to thundering noises, or reports like the shot of a pistol or cannon. In many cases they are of a whistling or singing character, while in others there is a buzzing, or dripping sound. They may be musical or simply noise. They may be mild or very intense. They may be constant, intermittent, or recurrent. It is doubtful if the noises in *simple* catarrhal otitis media ever assume the form of spoken language. Those who seem to hear voices and to receive messages and revelations probably have a central lesion of the cortex. The brain may otherwise be practically normal, so that the psychological phenomena referred to the organ of hearing may be the only evidence that the patient has departed from the normal mental state. The case of Joan of Arc, which has excited so much historic and romantic interest, possibly belonged to this class.

In some cases the tinnitus is synchronous with the heart beats, while in others it is very irregular in rhythm. Various explanations have been given to account for those cases in which the noises are synchronous with the cardiac pulsations, none of which seems to explain them satisfactorily. The most probable explanation is that in some way or other the vibratory thrill of the arteries of the tympanum is imparted to the membrana tympani and the ossicular chain in such a way as to be transmitted to the labyrinth, from whence the sensation is conveyed through the auditory nerve to the brain centre, where it is appreciated as sound. The tinnitus may be very high or low in pitch, and in either case is indicative of an advanced stage of the disease. If, on the other hand, it is medium in pitch, a less advanced stage is indicated. The state of the general health very materially influences the degree and the character of the noises. When the patient is fatigued or is affected by some disease which lowers his vitality, they are worse. I have seen patients who were the subjects of neurasthenia, in whom the pulsating noises were very pronounced. Some of these patients did not have ear disease, the pulsating tinnitus being only one of the symptoms peculiar to their nervous and anemic condition. In others, who were subject to catarrhal otitis media, the tinnitus was very much aggravated by the neurasthenia. The excessive use of alcohol and tobacco increases the intensity of the noises, and may even cause pulsating tinnitus, synchronous with the cardiac pulsations, even in persons who are not subject to otitis media.

Autophony consists of a vibration and echo-like reproduction of the patient's own voice. This symptom is sometimes present in the moist, but more particularly in the dry type of catarrh. It is most commonly found in those cases in which there is an undue patency of the Eustachian tube.

The *paracusis of Willis*, or "paracusis Willisii," is a symptom which is present in well-advanced cases. When present, it is an unfavorable sign, and should lead to a very guarded prognosis, as a more careful examination may reveal the presence of hyperostosis (spongifying) of the bony capsule of the labyrinth in addition to the middle-ear disease. Paracusis Willisii consists of an ability to hear better in the presence of noises than in a quiet place. Thus patients will hear better in a street car or train than they do in a quiet country home. It is a probable indication that the mobility of the ossicles is interfered with by ankylosis or adhesive processes, or the swelling of the mucous membrane of the tympanic walls, or it may point to hyperostosis of the bony capsule of the labyrinth.

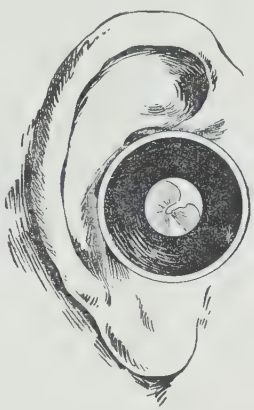
Objective Symptoms.—The *drumhead* should be examined with reference to its position, color, lustre, and reflection of light. In infants its position is normally at a very obtuse angle to the superior wall of the meatus, while in adults the obtuseness of the angle is much less pronounced. In other words, in adults the drumhead is more nearly at right angles to the axis of the external meatus than it is in very young children. In infants it is so nearly parallel with the superior wall of the meatus that it seems to be a continuation of it. As the tympanic ring develops

it rapidly assumes a more erect position, until it finally assumes that which is maintained throughout adult life. Its position will, therefore, depend upon the age of the patient and upon the completeness with which development has taken place.

If the Eustachian tube is closed for any reason, the drumhead will be drawn inward or retracted. This gives rise to a change in the contour of the drumhead, and consequently modifies the reflections from its surface. The cone of light which is normally present with the apex toward the lower end of the handle of the malleus, while its base is directed downward and forward toward the periphery, will either diminish in size, break into one or two whitish spots, or entirely disappear. These changes are, in most cases, indicative of retraction of the drumhead. If there are adhesions binding the membrana tympani to the promontory or other portions of the inner tympanic wall, its surface will present an uneven appearance, especially after inflation. At the points of adhesion it will appear whitish in color, whereas in the non-adherent portions there may be a slight reddish color, due to the reflection of light from the red mucous membrane of the inner tympanic wall.

The *color of the drumhead* has been variously described as of a pearl-gray, pinkish-gray, bluish-gray, or yellowish-gray membrane. Some of these observations have been made upon cadavers in which the normal colors were not present. By the use of such lights as are now at the command of most practitioners, the healthy membrane uniformly presents a pearl-gray color, with here and there a slight admixture of orange and purple. The orange is due to the red reflex of the inner tympanic wall, and is now regarded as a sign of spongifying.

FIG. 411



A normal membrana tympani of the right ear as viewed through a speculum.

Calcareous spots are sometimes found on the drumhead, even when there is no history of a previous suppurative process, and are undoubtedly the remnants of former inflammatory processes.

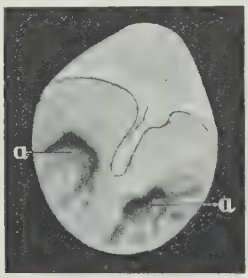
In the *normal drumhead* there is a *distinct luminous lustre* (Fig. 411), which is so modified in chronic catarrhal otitis media as to materially lessen its smoothness and brilliancy. The membrane appears whitish and velvety in texture in proportion to the amount of thickening it has undergone. The redness and the pinkish-gray color disappear because the vascularity and transparency of the drumhead are diminished.

The appearance of the drumhead may be modified by the presence of *tympanic secretion*. The dark line spoken of under Symptoms of Acute Otitis Media, which marks the upper limit of the secretion, may be present in these cases. Unless the thickening of the drumhead is so pro-

nounced as to interfere with its transparency, the bubbles of air spoken of in the same connection may also be seen. The presence of an appreciable amount of mucus in the middle ear is usually a sign of a subacute attack, but the drumhead may be so thickened that it is not easy to discern it. The opacity of the mucus increases with its viscidity, hence some estimate may be made by observing the character of the secretion present. In those cases in which the drumhead is atrophied in circumscribed areas, the secretion may be clearly seen at these points, while at the more opaque and thickened areas its presence cannot be detected. If there is a large quantity of mucus in the middle ear, the drumhead may bulge outward in its entirety if non-adherent, or in part if there are adhesions (Fig. 412).

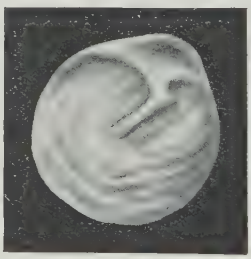
Prognosis.—The curability of chronic otitis media is somewhat in proportion to its chronicity and the pathological changes in the essential structures of the tympanic cavity. If the disease is of recent occurrence and the morbid changes are slight, the prognosis is quite favorable. If the disease is of long standing and pronounced degenerative changes in the mucous membrane covering the ossicles or the membrana tympani have occurred, the prognosis as to the restoration of hearing is not good.

Fig. 412



Adhesive retractions (*a*, *a*) of the membrana tympani.

Fig. 413



Adhesive processes affecting the membrana tympani.

Treatment.—The treatment should take two general factors into account, namely, the etiology and the pathological changes present. If the chronic disease is the offspring of an acute catarrhal process, the causes of the acute disease should be determined and eradicated if possible. If the patient has been subject to either of the forms of rhinitis or sinuitis, he should be treated accordingly. Ethmoiditis and sphenoiditis are particularly responsible for otitis media, and in a number of cases the chief cause. Too little attention has been given to these cavities in the treatment of ear disease. Appropriate treatment, surgical or otherwise, addressed to the sinuses, if given early, speedily relieves the ear disease. The symptoms of mild chronic ethmoiditis and sphenoiditis are not so obvious as to attract the attention of the physician unless he has had unusual opportunities for making such observations. The patient, perhaps, only complains of a "dropping" into the throat. An examination of the epipharynx and posterior choanæ may show a mucopuru-

lent secretion flowing over the posterior ends of the middle turbinals on to the posterior wall of the epipharynx. Anterior rhinoscopy shows the middle turbinal closely approximated to the septum. The divulsion of the middle turbinal away from the septum, or its partial or complete removal, will often exert a very favorable influence upon the course of the aural disease. In some cases, it may be necessary to make a total exenteration of the ethmoidal cells and to remove the anterior wall of the sphenoidal sinus.

If the ear disease is due to tonsillar disease, total ablation of the tonsil with its capsule intact is the best method of procedure.

Adenoids and inflammatory processes of the epipharyngeal mucous membrane, if present, should be treated. The presence of adenoids often perpetuates a chronic epipharyngitis, hence their removal exerts a favorable effect. As the pharyngeal inflammation extends by continuity of tissue to the Eustachian tube and middle ear, it is obvious that the removal of the adenoids or their remnants will exert a very favorable influence upon the course of the ear disease. McBride and Logan Turner have shown that adenoids often persist in adults, undiminished in size. In every case of chronic catarrhal otitis media, the otologist should examine the epipharynx, and if adenoids are present, they should be removed, even though they do not obstruct the nose.

When the structures adjacent to the Eustachian tube have been freed from morbid processes, the ear may be treated for the removal of the local morbid lesions and to restore the equilibrium of tension between the drumhead ossicles and the labyrinthine fluid.

The tympanic cavity should be inflated for three purposes, namely: (*a*) To force the secretions from the tympanic cavity and Eustachian tube; (*b*) to restore the equilibrium of air pressure on the two surfaces of the membrana tympani; and (*c*) to improve the arterial and lymphatic circulation of the lining mucous membrane. (See Principles of Tympanic Inflation and Methods of Tympanic Inflation.)

The air should be rarefied in the external meatus with Delstanche's rarefacteur after each inflation, as this increases the passive hyperemia of the inflamed membrane and promotes the absorption of the inflammatory exudates. It also reduces the annoying tinnitus usually present in this disease.

The mechanical removal of the secretions from the middle ear may be accomplished by paracentesis (Schwartze) or incision of the drumhead and by suction applied to the external meatus. This procedure is only indicated when the secretions are so heavy and tenacious as to resist being discharged through the Eustachian tube, or when the tube is obstructed by disease. The incision should be long and curved (see Incision of the Membrana Tympani), as in acute suppurative otitis media before perforation.

Even then the secretions will not appear in the meatus for several minutes or hours, unless the middle ear is forcibly inflated or suction is applied to the meatus. The meatus should be lightly packed with a strip of gauze for a few hours, at the end of which time it will be saturated

with the secretion. After thoroughly cleansing the meatus with a cotton-wound applicator, it should be refilled with gauze. The incision usually closes in from one to three days, and should be repeated if marked bulging of the membrana tympani reappears.

When the secretions are more serous in character, drainage is facilitated, as suggested by Politzer, by having the patient take a swallow of water in his mouth, then inclining his head well forward and somewhat toward the opposite side, thereby causing the axis of the Eustachian tube to stand perpendicular to the plane of the earth. The patient's head should be held in this position for two or three minutes, to allow the secretions in the middle ear to gravitate to the tympanic end of the Eustachian tube. At the end of this time he should swallow the water held in his mouth, thus opening the pharyngeal end of the tube and allowing the secretions to flow into the pharynx. As Politzer says, shortly after this procedure the membrana tympani presents a grayish color, whereas it was yellowish in color.

The passive hyperemia of the mucous membrane of the Eustachian tube gradually subsides during the treatment by inflation, and the patency of the tube is gradually restored. The secretions also diminish in quantity and in consistency, and the tube becomes adequate to carry on its drainage and ventilating functions.

In rare instances the swelling of the tube persists, and it may become necessary to make local applications of weak zinc, silver, ammonium chloride, ol. eucalyptus, and the vapors of menthol to the tube. Generally speaking, these remedies are of slight value, a better procedure being the administration of hepatic and saline aperients. Mechanical vibrations behind the angle of the inferior maxilla are very useful in opening the Eustachian tube when it resists the usual methods.

A. H. Buck has recommended the introduction of medicated bougies. Politzer uses a small violin string cut into suitable lengths for this purpose. They are soaked in a saturated solution of the nitrate of silver, dried, and introduced through a catheter as far as the isthmus tubæ, and left in position for from three to five minutes. Three to four applications often open the tubes.

Sidney Yankauer recommends the use of sounds daily through a standard Eustachian catheter if a firm stricture or obstruction is present. After a few days a larger sound may be introduced. The sound should be left in the tube for fifteen minutes.

ADHESIVE PROCESSES IN THE MIDDLE EAR

Synonyms.—Sclerosis of the middle ear; otitis media catarrhalis chronica; dry catarrh of the middle ear; otitis media catarrhalis sicca; otitis media sclerotica; proliferous inflammation of the middle ear.

Etiology.—The causes of adhesive processes in the middle ear are not fully understood. It is probable that several conditions are included under this title. Exudative catarrhs of the middle ear are often attended by the formation of adhesive processes, and these sometimes appear

without being preceded by a secretive or exudative catarrhal inflammation. The trophic centres or tracts seem to be at fault, and the onset and progress of the disease are insidious and result in pronounced deafness. The membranous labyrinth is often involved, probably from the same trophic influences. The mucous membrane around the oval window is especially affected, and the cicatricial contraction of the fibrous bands often fixes the stapes firmly in the window. Atrophy and fatty and colloidal degeneration of the labyrinth often occur simultaneously with or precede the sclerotic processes in the middle ear. The adhesive processes resulting from exudative catarrh of the middle ear are not attended with such pronounced deafness, and are marked by decided symptoms even in the early stages. In the trophic or insidious form, symptoms do not usually manifest themselves until the disease is well advanced.

The etiology may be summarized as follows:

(*a*) Exudative or moist catarrh of the middle ear. There is some doubt as to the causative influence, as in children in whom it most frequently occurs the adhesive processes are rarely found.

(*b*) *Trophic* disturbances affecting either the middle ear or labyrinth. It appears in some cases to affect the labyrinth first and extend to the middle ear. Probably both the middle ear and labyrinth are affected at the same time, although the symptoms may become manifest in one earlier than in the other. It is also quite probable that hyperostosis or spongifying of the bony capsule of the labyrinth is mistaken for an adhesive process, though the normal appearance of the drumhead should obviate such a mistake in diagnosis.

Pathology.—The adhesive processes may be classified as either diffused or circumscribed. The diffused type usually arises from an exudative chronic catarrh; the circumscribed type from trophic disturbances.

According to Politzer, "the structural changes in the mucous membrane consist in partial or total transformation of the new-formed round cells into fibrous connective tissue, interstitial hypertrophy of the mucous membrane with retrograde metamorphosis of the new-formed tissue, shrinking, sclerosis, atrophy, and calcification."

In those cases in which the secretions are still abundant, the mucous membrane is hyperemic, spongy, or gelatinous, and yellow or bluish red in color. The surface is uneven and ragged in appearance.

After the moist stage has subsided, the membrane becomes smooth, very thick, and firmly attached.

In the diffused or *insidious type*, the changes seem to proceed from the periosteum to the epithelial surface of the membrane. The favorite location for the adhesive process in these cases is about the oval window (spongifying?). The general appearance on inspection through an opening in the drumhead shows very little evidence of the true condition. The contraction and calcification take place in the deeper portions of the mucosa and fix the foot plate of the stapes in the oval window.

In another class of cases, numerous fibrous bands form in the middle ear. They may extend from the ossicles to the walls of the tympanum or

from ossicle to ossicle; or they may extend from the walls to the drumhead. The ossicles are thus bound together, and the drumhead is drawn by contracting fibrous bands to the fixed walls of the middle ear (Fig. 413). The normal tension of the ossicular chain and drumhead is thereby unbalanced, and serious disturbance of hearing occurs.

In *fetal life*, bands or folds of mucous membrane exist in the same places often occupied by fibrous formations in the adhesive process. They may be, therefore, only perversions of an earlier embryonal formation. According to Toynbee and von Tröltsch, the bands are sometimes transformed by calcareous deposits into bone-like processes.

In addition to the foregoing changes, the articulations of the ossicles may be ankylosed by fibrous formations or by the deposit of lime salts. In either event, the vibratory function of the chain of ossicles is impaired.

The mucous membrane of the entire attic in rare cases undergoes calcification, and a partial or complete obliteration of the attic results.

The changes in the Eustachian tubes are largely dependent upon whether the middle-ear disease is of the diffused or the circumscribed variety. In the diffused type, the tube is similarly affected, while in the trophic type, it is usually normal. The lumen is obstructed in the diffused variety, while it is unaffected in the circumscribed type.

Both ears are affected except in rare cases. This, together with the fact that it rarely occurs in children, in whom the moist or exudative catarrhs are most common, rather discredits exudative catarrh as the cause. When it occurs in children, it is usually easy to trace it to disturbances of nutrition, scrofula, etc.

Symptoms.—It is convenient to study the symptoms under the (*a*) drumhead, (*b*) the Eustachian tubes, and (*c*) the subjective symptoms.

(*a*) The *drumhead* is thickened, lustreless, and opaque. Areas of opacities more or less sharply defined may sometimes be seen. In some cases they are sharply defined, and appear as chalky white deposits, while in others they merge into the surrounding tissue with ill-defined borders. The spaces between the whitish deposits appear dark or bluish in color.

The *handle of the malleus* appears less distinct and wider than normal on account of the thickened condition of the drumhead. The cone of light is shortened, irregular, or broken. The handle of the malleus is drawn inward and backward, and is, therefore, foreshortened.

The *adhesive bands* may be attached to the drumhead and cause circumscribed retractions (Fig. 412). The retracted areas may also be due to atrophy or to direct adhesions of the drumhead to the inner tympanic wall. They appear as rounded, oval, or irregular depressions (Fig. 413).

Schwartze called attention to a distinct reddish glimmer around the umbo as indicating a circumscribed inflammation (insidious type) around the oval window. In these cases the drumhead is usually normal, although it is occasionally opaque or atrophic. Such cases are now generally recognized as hyperostosis of the bony capsule of the labyrinth.

The external meatus is usually devoid of cerumen, although it may be covered with a dense brown secretion.

(*b*) In the diffused variety the Eustachian tubes may be more or less obstructed by fibrous formations in their lumens. In the circumscribed variety they are usually normal.

(*c*) The subjective symptoms vary according to the degree of involvement of the middle ear and labyrinth. They also vary with the location and character of the lesion.

Perhaps the most common and pronounced subjective symptom is *tinnitus*. If the disease is well advanced it is continuous, although its intensity varies with the atmospheric conditions and constitutional vigor of the patient. If tired, worried, or weakened from the excessive use of alcoholic beverages, or illness, it becomes more pronounced. The noises vary in character and intensity even in the same individual.

Disturbances of hearing may appear simultaneously with the tinnitus, although the subjective noises usually appear first. The noises sometimes increase with the deafness, although in many cases they gradually diminish and cease altogether with complete deafness.

Pain is rarely present, although hyperesthesia acoustica is often a prominent symptom in the early stage of the disease. It is especially marked in the presence of shrill tones and loud speech.

More or less *giddiness* and *fulness in the head* are experienced in the cases in which there is continuous tinnitus. In some cases the Ménière group of symptoms is present, especially when there is a sudden increase in the deafness. It is probably due to a rapid deposit of an exudate in the labyrinth. The giddiness is sometimes persistent, while in others it gradually disappears without apparent damage. Aprosexia or difficulty in fixing the attention is sometimes complained of.

The *hearing is disturbed* in proportion to the interference with sound waves passing through the drumhead and ossicles and the degree of pathological changes in the labyrinth. The patient hears at a greater distance at one time than another, although the variation is not as great as is observed in ordinary catarrhal otitis media with secretion. The condition of the patient influences the hearing in a marked degree. He hears better in the morning when vigorous than he does toward evening when weary. Mastication of the food temporarily increases the deafness.

Hearing for speech may be very poor, while the finest variations in music may be distinguished, or the falling of a small instrument may be distinctly heard (Politzer).

Paracusis Willisii, or ability to hear better in a noisy place, as in a street car, is quite characteristic of this affection. It is important to ascertain in every case whether or not this symptom is present, as it gives a fair indication as to the prognosis of the disease. It should not be assumed, however, that the patient cannot be benefited by treatment because this symptom is present. The ordinary treatment by inflations and massage will usually fail to afford relief, but more radical measures, to be described, will in rare instances prove effective.

The Course of the Disease.—The course of the disease is *progressive*, although it is not steady in its advancement. It rarely progresses by gradual increase in the deafness, but goes by leaps and bounds. It often

remains stationary for years and then suddenly becomes worse. It is always progressive, as it is due to degenerative pathological changes in tissues, as contraction, calcification, and ossification. These conditions develop slowly, on account of the nature of the pathological process. They progress by leaps because the changes may involve portions of the tissue but little concerned in the function of hearing, until finally it encroaches upon tissue intimately concerned in audition, and hearing suddenly becomes impaired. This does not necessarily mean that the pathological process has suddenly increased, but that it has invaded the functionating tissue. The disease rarely causes complete deafness.

In the *insidious* or *trophic* type of the disease, persistent tinnitus often of a most aggravated character, may exist for years without deafness. The trophic interstitial changes are chiefly about the fenestra of the vestibule (oval window). Finally, the foot plate of the stapes is ankylosed, and deafness becomes a pronounced symptom. These cases are often mistaken for *nervous tinnitus* until the deafness sets in.

Politzer says that the greater number of cases in which ankylosis of the stapes was observed post mortem, he found from the history of the patient that the decrease of hearing occurred after the existence of subjective noises for ten or fifteen years, and the progressive increase of deafness was very gradual. In these cases there was generally a marked negative Rinné, with sometimes lengthened and sometimes diminished duration of perception through the cranial bones, the latter, especially when the disease had existed for a long time, and in old age.

When unilateral adhesive inflammation has existed for a long time and the other ear subsequently becomes involved, the progress in this ear is quite rapid, in contradistinction to the progress in bilateral involvement.

In rare cases a change for the better takes place spontaneously. This may be permanent, or it may be followed by a sudden increase of the deafness and tinnitus.

Diagnosis.—(*a*) Thickening, contractions, and chalky deposits in the drumhead.

(*b*) The drumhead often presents a ground-glass appearance.

(*c*) Marked negative Rinné with loss of hearing for low tones shows middle-ear involvement.

(*d*) Adhesive bands may be present, and the Rinné test does not show a marked negative result. Labyrinthine involvement probably present.

(*e*) High tones are heard better than low ones. In some cases, however, there is loss of hearing for high tones, thereby indicating labyrinthine involvement.

(*f*) By the use of Siegle's otoscope (Fig. 410), the drumhead may be made to move back and forth under alternate suction and pressure. If adhesions are present, the drumhead remains fixed at these points.

(*g*) Inflation of the middle ear causes the thin portions of the drumhead, when present, to bulge outward like bubbles. Improvement of hearing usually lasts while the bubbles remain inflated. The adherent parts remain unmoved under inflation.

(*h*) Marked movement of the handle of the malleus precludes ankylosis of the malleus and incus. Ankylosis of the incus diminishes the movement of the malleus.

Prognosis.—The prognosis will be studied under two headings, namely: (1) The more favorable signs, and (2) the unfavorable signs.

The More Favorable Signs.—(*a*) Fibrous bands following the secretive form of catarrh are more favorable than those from the insidious type which are more often associated with labyrinthine disease. (*b*) If the case has not progressed to a high degree of deafness, the prognosis is more favorable. (*c*) If subjective noises have been but little manifested, the prognosis is more favorable. (*d*) Good bone conduction is also a favorable sign. (*e*) Improvement in hearing and tinnitus after inflation is a good sign.

The Unfavorable Signs.—(*a*) Early deafness. (*b*) Slight or no increase in the hearing distance after inflation of the middle ear. (*c*) Diminished bone conduction. (*d*) Advanced age. (*e*) Constitutional ailments. (*f*) Heredity.

It should be said that complete restoration of hearing is not possible in any of the cases, as the changes have been of long duration and are retrograde in character. Indeed, few cases are benefited by treatment.

Treatment.—This is most conveniently divided into (*a*) non-surgical and (*b*) surgical treatment. The purpose of treatment should be threefold, namely, to improve the hearing, mitigate the tormenting subjective noises, and check the progress of the disease.

Non-surgical Treatment.—The form of treatment most in vogue among physicians in America is *inflation of the middle ear*, by either the Politzer method or through the Eustachian catheter. Politzer claims better results by his method than by the use of the catheter. This is probably due to the fact that the Eustachian tubes are usually quite patent and easily inflated by the bag. Those cases which show improvement after the use of the air-bag are more favorable for treatment than those which show no improvement. The longer the improved hearing continues after each inflation the more hopeful is the prognosis. The object of middle-ear inflation is to restore the normal air pressure to the cavity of the middle ear and to stretch or break down recent adhesions. It is quite probable that but little effect of this kind is produced by this procedure, except in the early stages while the adhesive bands are slight and fragile. The chief use, therefore, of intratympanic inflation is to equalize the air pressure, and thus overcome in some measure the pressure upon the labyrinthine fluid and auditory nerve endings.

Local medical treatment has but little if any curative effect. The medicated vapors and nebulæ, so much extolled in the medical literature a few years ago, have no appreciable effect whatever, except such as may be explained by the inflation which usually accompanies their use. We may say the same in regard to many of the medicines injected through the Eustachian tubes, as their use is usually preceded by inflation.

Numerous *injections* have been recommended for adhesive processes in

the middle ear, some of which seem to be followed by good results. Only those which have proved of special value will be referred to here.

The following formula has been used extensively by Politzer through a catheter with favorable results:

℞.—Sodii bicarb. . . . . gr. x
Glycerini . . . . . ♏ ix
Aquæ des. . . . . q. s. ℥j—M.
Ft. sol.
Sig.—Inject 5 to 8 drops into the middle ear through a catheter 2 to 3 times per week.

It acts mildly and does not cause irritation.

Pilocarpine is another popular remedy, and should be used in a 2 per cent. solution, 5 to 6 drops being injected into the middle ear. Perspiration and salivation usually occur while the patient is still in the office, especially in those cases in which the membrane of the middle ear is still boggy and well supplied with bloodvessels. In the dry or trophic type these symptoms may not occur. It should not be used in patients with weak hearts.

Delstanche recommended the injection of *liquid vaseline* into the middle ear through a catheter. M. A. Goldstein has also reported favorable results from its use. It is claimed that it lubricates and softens the fibrous tissue, and that the force used in its introduction stretches the fibrous bands and liberates the ossicles. Probably the only benefit is from the simultaneous inflation of the middle ear.

Caution.—Whatever method of medication is used, extreme care should be exercised lest too great an irritation be produced by the remedy. Temporary improvement only follows excessive irritation. The case then rapidly passes into a worse condition than before treatment.

Massage.—The alternate rarefaction and condensation of the air in the external acoustic (auditory) meatus moves the drum membrane back and forth. As the handle of the malleus is located between the layers of the drum membrane, it is also propelled inward and outward with the movements of the drumhead. If there are firm adhesions binding it to the promontory, it will not perform these excursions.

Bing has recommended prolonged rarefaction of the air in the external auditory meatus by the use of an olive-tipped instrument inserted into the meatus. The tip is perforated and has a valve at its inner extremity. The air is withdrawn from the meatus through the rubber tubing, whereupon the air pressure closes the valve. In this way rarefaction can be maintained for one-half to one hour. He thinks that in some cases this is an advantage over simple alternating rarefaction and condensation of the air in the meatus.

Lucae has devised a spring probe with a cup-shaped extremity to fit over the short process of the malleus. Pressure is exerted upon the short process, and then released, repeating the motion a number of times. This motion is transmitted to the other ossicles, the ankylosis and cicatricial adhesions being stretched or broken down. The treatments are very painful, and are, therefore, not used to any great extent. If this difficulty could be overcome, the use of the probe would prove

of greater value. It might be advisable to administer nitrous oxide gas and use the probe during the brief anesthesia. There is little danger or inconvenience connected with this anesthetic, and the exigencies of the case often warrant its use. The injection of a 2 per cent. solution of cocaine into the middle ear through a catheter may also be practised to mitigate the pain. The use of Lucae's probe in suitable cases at intervals of seven to ten days, inflation being practised on alternate days, is sometimes helpful. If the element of pain can be eliminated, it is the remedy *par excellence* in cases in which the adhesive processes are not too far advanced. The hearing is sometimes improved to a remarkable degree, and the subjective noises correspondingly diminished. The improvement is not permanent in a majority of cases, nor is there any method of treatment known which will make it so.

The *length of time* during which any of the aforesaid treatments should be continued varies. It should only be continued while the hearing distance continues to increase. This usually ranges between two and six weeks. The greatest amount of improvement occurs during the first six or eight days. *To continue the treatments longer than improvement of the hearing distance increases often leads to ill effects.*

As the improvement in hearing is temporary, it becomes necessary to give occasional treatments to maintain the beneficial effects realized. Politzer thinks his method of inflation the best adapted for the after-treatments.

Stenosis of the Eustachian tube may be overcome by inflation if due to accumulated mucus, or by the use of soft rubber sounds (Yankauer) if due to fibrous bands or rings within its lumen. They should be introduced through the Eustachian catheter. In the adult the tube is about one and one-half inches long, and the sound should be passed through its entire length. Sounds may be made of whalebone, catgut, celluloid, or rubber. If for any reason it is desirable to locate the stricture, an olive-tipped bougie should be used, whereas to secure therapeutic effect a sound with a filiform tip should be used. Medicated bougies (silkworm gut) may be used and left in place for twenty or thirty minutes. A solution of the nitrate of silver is the astringent chiefly used for this purpose.

The introduction of the sound should be done with extreme caution and gentleness, as force may cause it to penetrate the mucosa of the tube. This would be unfortunate, as subsequent inflation might cause emphysema of the submucous tissues. This accident occasionally happens in catheterization of the tubes through abrasions made during the manipulation of the bougie. The dilatation with the rubber sounds should be made daily for a few weeks, larger and larger sounds being used every few days until the stricture is completely overcome. The intervals between treatments may then be prolonged, until finally they are weeks or months apart.

Internal medication is of value in those cases suffering from constitutional diseases. I have seen cases resist all treatment until iron and arsenic were administered. Others will improve in hearing when the

iodide of potash or tonics are given. But even these cases do not entirely recover; they only become somewhat improved in hearing and tinnitus.

I am indebted to Dr. Geo. F. Suker for the following analysis of the conditions of the ear in which thiosinamin is indicated. In 1897–98 he used it in a number of such cases, and bases his conclusions upon this experience together with the literature concerning its use in other conditions:

The class of cases in which thiosinamin has been found of value come under the following heads:

1. So-called catarrhal deafness in which there is a diapedesis of leukocytes into the meshes of the membrana tympani which ultimately cause cicatricial-like thickening.

2. Subacute suppurative otitis media with a small perforation of the drum. The latter is thickened by infiltrations, but there is no true fibrous ankylosis of the ossicles.

3. Inflammation of the middle ear, suppurative or otherwise, leading to a fibrous ankylosis of the ossicles and with very slight structural changes of any kind in the membrana tympani.

4. Deafness, rather a loss in the acuity of hearing, due, as we have reasons to suppose, to some fibrous changes in the auditory nerve or its endings.

5. Cases in which two or more of the above-mentioned conditions are present in the ear.

6. Suppurative otitis media with extensive loss of drum substance and the formation of fibrous bands which impede the free action of the ossicles.

7. Cases in which there is a transudation of the lymph into the substance of the drum, which, instead of being absorbed, remains and becomes partly organized, thus causing drum thickening, and, therefore, interferes with the transmission of sound waves.

All such cases, if the thiosinamin is persistently given in alternating periods of time, will be markedly benefited. It may be administered by the mouth or hypodermically. If by the mouth, the dose should be rapidly increased until 6 to 10 grains per day are taken. If employed hypodermically, use a 10 per cent. solution in equal parts of glycerin and water. Of this give 12 to 18 c.mm. three times a week.

Thiosinamin acts as a glandular stimulant; at first it causes a breaking down of the exudate. Its powers of removing or absorbing an exudate is not unlike that of potassium iodide and mercury, peptone, pepsin, sodium urate, and allied bodies.

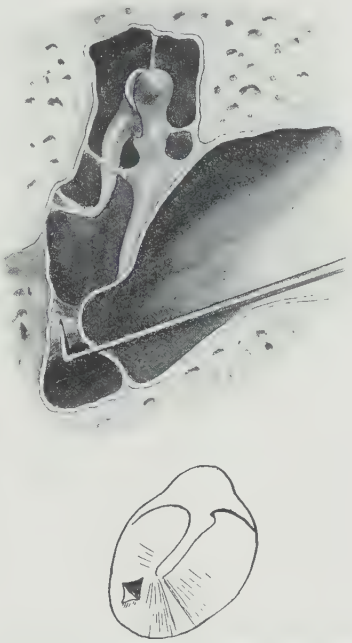
In employing the thiosinamin treatment, the hygienic and other needed *regime* must not be overlooked. Give it for periods of six to eight weeks, and then cease for a week or ten days, after which begin again.

Whether or not larger experience will support the claims thus clearly set forth remains to be demonstrated. Enough evidence is available, however, to justify extended trials of it. Its favorable action on keloids and lupus is well known.

Rest is another therapeutic measure of special value in neurasthenic cases. I have seen cases make material improvement both in hearing and in the severity of the subjective noises under this mode of treatment. J. A. Stucky reports good results following rest in bed, with massage of the body.

Surgical Treatment.—*Operations on the drumhead* for the relief of deafness have been performed for more than a century. Himly and Astley Cooper, in 1795, removed portions of the drumhead and strongly recommended the procedure as a means of admitting sound waves to the labyrinth and of relieving the increased tension of the ossicular chain. Others soon followed in their wake, all to meet with ultimate disappointment, as the relief was only temporary. It was found impossible to keep the wound open for any length of time. Later, vulcanite and metal eyelets were used with unsatisfactory results. All efforts to maintain the opening in the membrana tympani (drumhead) have failed. The difficulty has been to secure the epidermization of the edges of the wounded membrane. The author suggests the use of small skin grafts on the margin of the perforation, after the Thiersch method.

Fig. 414



Severing an adhesion of the membrana tympani to the promontory. A small triangular flap is made in the drumhead and the right-angle knife introduced through the opening thus made and the adhesive band severed.

Malherbe recommends lifting the auricle forward and the removal of the posterior wall of the meatus external to the annulus tympanicus, as in the meatomastoid operation. He then establishes communication between the middle ear and the meatus *via* the antrum and the aditus ad antrum. The opening is maintained by inserting a celluloid or gold tube through the opening in the wall of the meatus. He only recommends this procedure in cases of moderate severity. An improvement over this method would be to form the Ballance plastic flaps and reflect them through the opening in the meatus, as described under the meatomastoid operation. This would obviate the necessity of wearing the vulcanite tube recommended by Malherbe.

Section of the posterior fold of the drumhead (Fig. 397) was first suggested by Politzer in 1871: "It is advisable in all cases where the objective signs of an abnormal inward curvature of the membrana tympani are present, where the inferior extremity of the handle of the

malleus is, therefore, abnormally inward, and the short process of the malleus and the posterior fold of the membrane extending from it project strongly toward the external meatus. If these changes are combined with a disturbance of hearing of a high degree and loud subjective noises, which cannot be materially improved by the local methods of treatment, an experimental section of the posterior fold is justifiable in such cases."

The operation is simple and consists of a section of the fold just posterior to the short process of the malleus or midway between it and the peripheral extremity of the fold. The knife should not penetrate deep enough to sever the chorda tympani nerve in its passage between the malleus and incus.

The handle of the malleus should immediately drop downward and forward as the tension is relieved. The tinnitus is usually most relieved, although in some cases there is also an improvement in hearing. The benefit lasts only a few weeks or months in most cases.

Adhesion of the drumhead to the promontory may be overcome by making a small triangular opening in the drumhead and introducing a right-angle knife through it. The adhesion is then severed, as shown in Fig. 414.

Dr. Sidney Yankauer has, perhaps, devised the best means of treating obstinate old strictures at the isthmus *via* the external auditory canal. As the drumhead is rarely perforated in adhesive processes, it is necessary to incise it anterior to and on a level with the umbo of the malleus, this being in a position corresponding with the tympanic orifice of the Eustachian tube. Through this opening the probe is introduced and directed downward, inward, and forward to the isthmus of the tube. The presence of a dehiscence or caries of the bony wall may be detected. The first by the softness of the tissue and the latter by the rough grating of the probe over the surface of the bone. Having explored the tube, the salpingitome should be introduced in a similar manner until its end passes through the isthmus. The instrument should then be pressed backward and downward to force the blade through the stricture; it is then withdrawn, incising the mucous membrane to the bone.

The above procedure should be preceded by the application of a 5 per cent. solution of cocaine to the pharyngeal end of the tube through a catheter, and to the middle-ear cavity through the incision in the drumhead.

CHAPTER XLII

OTOSCLEROSIS; SPONGIFYING OF THE BONY CAPSULE OF THE LABYRINTH[1]

Synonyms.—Otitis media insidiosa; hyperplasia of the bony capsule of the labyrinth; capsulitis labyrinthii.

In a recent article read by Denker before the International Otological Congress at Boston the following definition was presented:

We understand by otosclerosis a disease in which there is a permeable tube and a normal tympanic membrane, accompanied by a definite and characteristically marked clinical picture of a progressive difficulty in hearing, as shown by the functional tests. As a pathologic-anatomic foundation for the disease, investigations have revealed a loss of movement of the stapes brought on by bony ankylosis in its framework or in the niche of the oval window, and a progressive spongification of the bony capsule of the labyrinth.

To this definition, upon the ground of our present knowledge, must be added that, in addition to the disease of the bone, there is to be found, as shown by a histologic examination of a large number of cases, an atrophic degenerative process in the nerve endings in the membranous labyrinth.

Only the forms of disease which clinically and pathologic-anatomically correspond to the above definition will be dealt with, and not those forms of chronic affections accompanied by thickening of the interstitial connective tissue, which, because of changes on the tympanic membrane are recognized as chronic adhesive processes, or as the residues of long-standing middle-ear suppurations.

Etiology.—There are two schools of thought in reference to the causes of otosclerosis,

1. *The disease of the bony capsule is secondary to an inflammation of the membrane lining the tympanic cavity.* Habermann examined 13 cases and arrived at the conclusion that the disease of the bone is secondary to middle-ear inflammation which extends along the larger bloodvessels and is disseminated in the bone through the medullary spaces. Katz and Toynbee consider it rheumatic inflammation of the stapediovestibular articulation and call it "arthritis rheumatica." Schilling and Scheibe also regard it as of inflammatory origin.

2. *The disease of the bony capsule originates in the bone.* In 1867 Moss first expressed the opinion that in view of the absence of any change in the mucous membrane of the middle ear, the disease must be an ostitic process in the temporal bone. Bezold and Scheibe, after examining a number of temporal bones affected by this disease, said

[1] I am greatly indebted to an article by Henry J. Hertz, wherein he reviewed the work of Continental observers, for most of the data presented in this chapter.

that the disease must originate either in the bony capsule of the labyrinth or in the periosteum of the niche of the oval window. Alfred Denker quotes Politzer and J. Möller as saying that the greatest changes were found in the deeper layers of the bone, most of the cases failing to show pathologic changes in the mucous membrane. Hanan, a pathologic-anatomist, who examined the specimens from the cases reported by Hartmann from the Siebermann clinic, contends that the process at the stapes and niche of the oval window is a hyperostosis with a rebuilding of the bone, and designates this new-formed bone as metaplastic connective-tissue bone, originating from the periosteum. Siebermann, however, arrived at the conclusion that the spongification does not arise in the periosteum, nor does it develop primarily from the labyrinth capsule, but that the oldest parts are to be found at the junction of the labyrinth capsule, which is formed out of the endochondrial substance, with the connective-tissue bone arising secondarily from the periosteum (probably in the last-named structure itself). Brühl also supports this opinion. Hegener is of the opinion that the bony change and the disease of the auditory nerve must be differentiated from each other, and that the acoustic affection is not a result of the bony changes. Manasse also agrees with Siebermann that the disease originates in the labyrinthine capsule. Denker is likewise strongly committed to this view, though he points out that there is in some cases an association of bony and mucous membrane disease, but that the disease of the mucous membrane might be secondary or accidental.

Manasse, believing the bone changes to be inflammatory in character (originating in the bone and not in the mucous membrane of the middle ear), has designated the process "ostitis chronica metaplastica of the labyrinth capsule." Brühl and Orth, according to Denker, believe that the disease is a "spongy hyperostosis," brought about by means of the vessels of the hyperplastic periosteum, and the vessels of the Haversian canals absorbing the old bone, which is followed by a regular, even, excessive formation of connective-tissue bone.

Siebermann, after careful study of a large number of temporal bones, comes to another conclusion. According to him, the beginning of the progressive spongification of the labyrinth capsule occurs, not as Manasse supposes, in an extension through new bone, but in the inward growth of cells from the periosteal tissue through the bone covering into the normal Haversian canals of the labyrinth capsule. Lacunar resorption from the walls, with the aid of mono- and polynuclear osteoblasts, goes hand-in-hand with it, and fails in no case. New bone apposition results only where resorption processes have taken place. The newly apposed bone contains more chalk than the old, and does not deserve to be known as the osteoid substance. As a differentiation from ostitis fibrosa, the spongifying process of the labyrinth capsule occurs, without inflammatory symptoms. Granulation tissue, leukocytes, and plasma cells are not present.

That trauma lead to stapes ankylosis, with the functional symptoms of otosclerosis was shown by a case published by Politzer in 1862. If we review what has been written concerning the etiology of otosclerosis,

it seems correct to state that in the majority of cases, at least, we have to do with an hereditary analogue as the cause of the disease. This predisposition lays the foundation upon which, under the influence of certain stimuli, there arises the affection with the characteristic symptoms and pathologic-anatomic changes. These stimuli are the increased bony formation during puberty, and the bony changes during pregnancy and the puerperium, which are probably dependent on the hyperplasia of the hypophysis, which appears during pregnancy. Furthermore, Denker believes the circulatory disturbances such as are present in arteriosclerosis, vasomotor neurosis, and lues may give the impetus to the development of the disease. It has not yet been proved that the continuous movement of the stapedial foot plate and the permanent contraction of the musculus tensor tympani can be regarded as a cause for the fact that the bony alterations which develop by predilection at the anterior circumference of the vestibular window, as Brühl claims.

The dense bone of the osseous capsule of the labyrinth contains cartilaginous cells, hence it is the area of election for the transformation of the cartilage into bone. The ossicles also have cartilage cells in them, and may be the seat of this disease. The distribution of the cartilage cells is most constant in the posterior half of the margin of the oval window (fenestra of the vestibule). They are also found in the capsule of the semicircular canals and the upper and lower walls of the cochlea. Any or all of these points may be affected and give rise to symptoms peculiar to the physiological bearings of the various structures. That is, the hyperostosis may be limited to the ossicles, the oval window, the cochlea, or to the semicircular canals, or it may involve two or more of them at once.

Age exerts a positive influence upon the development of the disease. It usually begins between the eighteenth and the fortieth years of life. Heredity has been noted as a rather common factor in the etiology, many cases giving a history of other members of the family having had the disease. In a noted American literary family several members were affected by it. The majority of the cases occur in young women. Sexual intercourse and parturition aggravate the symptoms, probably on account of the increased hyperemia produced by these acts, or it may be due to the hyperplasia of the hypophysis which occurs in pregnant women. The marriage of women affected by this disease should, therefore, be carefully considered before being consummated.

Pathology.—According to Denker, the osseous changes may be divided into two stages, the first of which consists in an active proliferation of all the cellular elements within the bone. New vascular and cellular tissue is formed in the narrow spaces and in the Haversian canals. Among the new-formed bone cells may be found giant cells, under the influence of which the basement of the bone substance is principally absorbed. Hollow spaces are formed, and areas of erosion gradually undermine the original compact bone, which becomes traversed by irregular and abnormal channels. With the absorptive process there is the

formation and apposition of new bone, which is unlike the original, in that it is more voluminous and porous. The second stage is ushered in when the progressive changes cease and when the new bone assumes a lamellar structure. Then the abnormally large and thick bone corpuscles are found concentrically arranged, and the nuclei undergo atrophy. The vascular system is likewise gradually altered by the formation of connective tissue, in which at times may be found fat cells. The Haversian canals and spaces have been changed in structure by this resorptive and appositional process, and all the cartilaginous elements have been metamorphosed into osseous tissue, as it cannot be found in the newgrowth. Thus the process constitutes not only hyperplasia and hyperostosis, but also a metaplasia.

The new structure differs from the normal by its affinity and greater absorptive power for carmine stains, which fact is utilized in the differential diagnosis. The microscopic evidence of this new formation is the osteophytes, situated usually near the stapes articulation. Frequently the stapes is partially absorbed by penetrating bloodvessels and replaced by osseous formations, and sometimes a dislocation of the stapes is produced by an encroachment of the osteophytes. The functions of the oval and round windows may also be seriously interfered with by the hyperostosis producing partial or complete occlusion. When the process invades the base of the cochlea, the patency of the Eustachian tube is threatened, and the microscope shows its lumen to be narrowed by thickening of the periosteum. Owing to the great vascularity attending the process, especially in the first stage, it is probable that the distressing tinnitus of progressive deafness may have its origin in the increased capillary circulation.

The structural alteration consequent upon an invasion of this bone into the cochlea and the semicircular canals may cause a change in the pressure of the labyrinthine fluid. The mechanical and physical qualities of the endolymph and perilymph may be so altered as to interfere with the nutrition of the parts and induce disease. The detonating sounds heard by some patients and Ménière's symptom complex may be ascribed to a perforation of the septum dividing the endolymph and perilymph.

While the histological alterations are found to be identical by different authorities, yet their designation of the bone hyperplasia differs and new terms are consequently introduced. Politzer defines it as capsulitis labyrinthii or otosclerosis. Siebenmann, noting the resemblance to sponge because of the rarefied spaces and porous structures, designated the new formation as spongification. Katz compares the process to Volkmann's osteitis vascularis chronica.

Symptoms and Diagnosis.—The symptoms, while more or less constant, vary with the anatomical structures involved. If only the ossicles are affected, the ankylosis of the stapes may be partial or complete; if the posterior bony margin of the oval window is the seat of the changes, the ankylosis may be complete and the stirrup pulled posteriorly by the stapedius muscle; if the cochlea is involved, the signs of nervous deafness

are present, *i. e.*, diminished bone conduction and the loss of hearing for the upper tone limit; if the process is in the semicircular canals, giddiness and nausea may be present. In mixed cases there may be a combination of these symptoms.

In the cases commonly recognized in practice, the disease is characterized by the signs of middle-ear disease without the objective appearances of it. That is, there is (*a*) loss of the lower tone limit, (*b*) a negative Rinné, and (*c*) an increased duration of hearing by bone conduction, all symptoms found in middle-ear disease, but upon inspection of the membrana tympani its appearance is normal, or is so slightly changed that it cannot account for the marked degree of deafness present, and the Eustachian tube is normally patent.

When the hyperostosis is located exclusively in the ossicles, and the ankylosis is partial or complete, the symptoms are those of ordinary middle-ear disease, except the membrana tympani is normal in appearance and the Eustachian tube open.

When the hyperostosis is limited to the cochlea, the usual signs of nervous deafness, loss of hearing for the upper tone limit, positive Rinné, and shortened and diminished bone conduction are present.

When both the oval window and the cochlea are involved, it is practically impossible to make a diagnosis. This is also true when the oval window is affected by hyperostosis (spongification) and the middle ear is simultaneously diseased. Tinnitus is present in nearly all cases, and is sometimes very pronounced. The paracusis Willisii is more pronounced than in any other form of ear disease.

Summary of Symptoms.—As the spongifying or hyperostosis affects various parts of the ear structures, the symptoms vary accordingly.

The following classification includes the chief clinical characteristics of each subdivision:

Hyperostosis about the Oval Window (Fenestra of Vestibule).—1. Loss of hearing for one-half to one and a half octaves of the lower tone limit in one or both ears.

2. Negative Rinné in varying degree.

3. Prolongation of hearing by bone conduction for fork A of the Edlemann-Bezold set of forks (Schwaback test).

4. Hyperemia of the promontory, appearing as a yellowish-red glow through a membrana tympani otherwise normal in appearance. The handle of the malleus may be foreshortened, but is not rotated.

5. Patency of the Eustachian tubes.

Hyperostosis of the Stapes.—The same as the preceding except in a less degree.

Hyperostosis of the Cochlea.—1. Loss of hearing for the upper tone limit, and slightly for the lower tone limit. Shambaugh reported a case in which there were islands of deafness, thereby showing that the hyperostosis may be limited to definite isolated areas in the cochlea.

2. Positive Rinne.

3. Shortened duration of hearing by bone conduction for fork A, on account of the degeneration of the membranous cochleæ.

4. Hyperemia of promontory showing through an otherwise normal membrana tympani.

5. Patency of Eustachian tubes may be affected by the thickening of the periosteum.

6. Loud, subjective noises are often complained of.

Hyperostosis of the Semicircular Canals.—1. Giddiness or dizziness at times.

2. Nausea may or may not be present.

3. Perhaps slight deafness.

4. Membrana tympani and Eustachian tubes normal.

5. Subjective noises, often of a loud, popping character.

Hyperostosis Around Oval Window Combined with Catarrhal Otitis Media or Other Middle-ear Diseases.—1. Loss of hearing for one-half to two octaves of the lower tone limit.

2. Negative Rinne in varying degree.

3. Prolonged hearing by bone conduction for fork A.

4. Retraction of the membrana tympani.

5. Foreshortening and rotation of the malleus.

6. Eustachian tubes obstructed.

7. Subjective noises are usually present.

A positive diagnosis of spongifying in a case with the above symptom complex is impossible except at the postmortem examination, as it is masked by the catarrhal otitis media which presents the same symptom complex.

Prognosis.—The cure of the disease appears to be impossible. In a few cases slight or temporary improvement follows treatment, and in the early stage of the disease certain medicinal, mechanical, and surgical procedures afford relief. In the later stages all remedial measures fail, unless, indeed, Lake's exposure of the horizontal canal and vestibule is resorted to on account of the distressing tinnitus and vertigo. (See Intractable and Unbearable Vertigo, p. 975.)

Treatment.—Medicinal.—Small doses of phosphorus, gr. $\frac{1}{200}$, three times daily, for six months of the year, have given the best results. The treatment acts best in the early stages during the active proliferation of the cellular elements within the bone, when new vascular tissue is being formed in the narrow spaces and Haversian canals, and the absorptive processes and apposition of new bone is in progress.

Thyroid extract has likewise occasionally given good results under the same conditions.

Iodine, in the form of the iodide of potash, and mercury have been given by Politzer with good results when the diagnosis was made early on account of other members of the family having had the disease. That is, its appearance was carefully watched for, because of the known hereditary influence present. When a father or mother is known to have the disease, they should be warned that their children are liable to the same trouble, and that they should be periodically examined after puberty for its earliest expression. In this way there is some hope of modifying its progress by the administration of phosphorus, iodide of potash, or

thyroid extract, and by the correction of inflammatory diseases of the tonsils and adenoids, and of rheumatic, gouty, and scrofulous diseases. Thyroidectin in five-grain doses may be given three times a day. Depletion of the vessels of the head may be produced by the administration of cathartics and by hot foot and sitz baths.

To accomplish anything of importance an early diagnosis is positively necessary, and heredity should give warning of the impending disorder.

Mechanical.—Pneumomassage with the Delstanche rarefactor (Fig. 14) may be used to mobilize the ossicles when they are not excessively ankylosed (Hartz).

Clarence Blake calls attention to the fact that in practising pneumomassage, gentleness should be observed in its application, as otherwise the whole ossicular chain may be dislocated and irreparable damage done. He also calls attention to the fact that the posterior segment of the membrana tympani may become relaxed by excessive massage. Indeed, great damage may be done by any treatment addressed to the Eustachian tubes and middle ear. Aurophones are also damaging in this disease. The massage should, therefore, be gently administered, preferably with a hand pump, for one to two minutes, two or three times a week, for two months. After a rest of two months the massage may be tried again, provided improvement followed the first course of treatment. Further massage may be given at the discretion of the aurist. As soon as the nature of the disease is known, the patient should be advised to begin a systematic course in lip reading.

Surgical.—Stapedectomy has been tried with almost universal failure. Jack has performed the operation a number of times with but one or two permanent improvements. In some cases, stapedectomy is followed by the formation of scar tissue over the oval window, thus rendering the hearing worse than before the operation.

Prophylactic.—"An ounce of prevention is better than a pound of cure," is eminently exemplified in this disease. The prevention of conception, the avoidance of consanguineous marriages, catching cold, cold douches on the head, sea voyages, etc., will greatly diminish the number of cases, and modify the intensity of expression of those already developed.

CHAPTER XLIII

ACUTE AND CHRONIC SUPPURATIVE OTITIS MEDIA CHOLESTEATOMA

ACUTE SUPPURATIVE OTITIS MEDIA

This type of inflammation of the middle ear is characterized by marked hyperemia of the mucous membrane of the middle ear, including the inner wall of the drumhead. This may be followed by pain and perforation of the drumhead, through which the pus discharges into the external auditory meatus.

Etiology.—The exciting cause of this disease is the presence of pathogenic microörganisms in the middle ear, as already described under Acute Catarrhal Otitis Media; indeed, the catarrhal inflammation often assumes the suppurative type after a few days. In many cases the inflammation remains catarrhal in type until the drumhead is perforated, the microörganisms thus receiving the required environment to promote their rapid propagation, though spontaneous rupture sometimes promotes a rapid reparative process, due to good drainage and the increased reaction of inflammation. (See Chapter VI.) The perforation may occur either spontaneously or by surgical intervention. Incision of the membrana tympani is not contraindicated, as, if it is done under aseptic conditions, the danger of increased infection is reduced to the minimum; indeed, the reaction of inflammation is promoted, and the infection is thus overcome instead of being increased. Some cases are undoubtedly suppurative in type from the beginning, the inflammation, temperature, and pain being more pronounced than in the simple catarrhal inflammation.

Arthur B. Duel arrives at the following conclusions in reference to the relation of the infectious fevers to acute suppurative otitis media, his conclusions being based upon a study of 6000 cases of scarlet fever, measles, and diphtheria in the Willard Parker Hospital:

Acute purulent otitis developed in about 20 per cent. of the scarlet fever cases, in about 10 per cent. of the diphtheria cases, and in about 5 per cent. of the cases of measles. There were 26 mastoid cases, 2 in measles, 2 in scarlet fever, and about 20 in combined scarlet fever and diphtheria. Two were complicated with thrombosis of the lateral sinus.

Time of appearance: In scarlet fever the ear complications occurred the second or third week; in diphtheria, during the acute symptoms; in measles, during the acute stage, fever still being present.

In scarlet fever cases there was usually much greater destruction of

tissue than in those due to diphtheria or measles. A combination of two or more of the infectious diseases increased the danger, nearly one-half of such cases developing acute suppurative otitis media, and mastoiditis was a frequent sequela.

The Rivinian segment as an etiological factor: In children under five years of age, Duel found postauricular swelling present most frequently, which, he thinks, was due to the escape of pus through the unclosed Rivinian segment. Between the ages of five and ten the postauricular swelling was due to perforation of the thin mastoid cortex. In older children mastoid swelling was rare, except in those cases in which the external meatus was greatly inflamed. In all cases there was sagging of the postsuperior wall of the meatus near the drumhead.

The *predisposing causes* are colds, exposure, chronic rhinitis, chronic and acute epipharyngitis, adenoids, enlarged or inflamed tonsils, syphilis, tuberculosis, and other constitutional diseases. The acute exanthematous fevers, as scarlet fever, measles, diphtheria, whooping-cough, and influenza, are also responsible for many cases. The use of the nasal douche sometimes causes the disease. The author formerly used the nasal douche quite frequently in office practice, but abandoned it after seeing two or three cases of acute suppurative inflammation resulting directly from it. Cold injections into the meatus, bathing, diving, and snuffing cold fluids into the nose also act as causes.

Age has a direct influence, a large majority of the cases being in children. The damp, unsettled weather of autumn and spring also favors its occurrence.

Those cases occurring independently of any other disease are usually unilateral, while those occurring in connection with scarlet fever, diphtheria, measles, epipharyngitis, and adenoids are usually bilateral.

Finally, it may be stated that all conditions which lower the resistance of the tissues of the middle ear predispose to infectious inflammation. The exciting causes are the pathogenic microörganisms. The various constitutional diseases and the local diseases of the fauces, nose, and epipharynx produce lowered cell resistance, and predispose to the infection.

The *indications*, in view of the foregoing facts, are to remove the predisposing causes and increase the reaction of inflammation, in order to increase the resistance of the tissues to the bacteria and their toxins. (See Inflammation, and the Methods of Promoting the Reaction of Inflammation, Chapters VI and VII.)

Symptoms.—The symptoms may be grouped under pain, temperature, the appearance of the membrana tympani, the character of the secretions, the subjective noises, and the disturbances of hearing.

Pain.—The pain is sometimes preceded by a feeling of heaviness in the ear, or by a severe headache. It may be piercing, tearing, boring, or throbbing in character, and is more severe in children than in adults. It is continuous, but becomes less severe toward morning, when the patient falls into a sound sleep. Photophobia, edema of the eyelids, and conjunctivitis occasionally complicate severe inflammation prior

to the time of perforation of the drumhead. Facial paralysis and trigeminal neuralgia occasionally complicate the disease.

Temperature.—The temperature at the onset is elevated from 1° to 3° F., and is sometimes preceded by a slight chill, or creepy sensations, and, occasionally, in very young children, by convulsions. After the suppurative process is well established, and drainage is taking place through the perforation in the drumhead, the temperature subsides to about 1° above normal.

Membrana Tympani.—In the early stages, the membrana tympani presents the appearances found in acute catarrhal otitis media. It is scarlet red, ecchymotic, swollen, and more or less bulging. The handle of the malleus is obscured by the swollen drumhead. In the postsuperior quadrant of the membrana tympani a blister is sometimes present, giving a pearly lustre to this area. If the case is seen quite early, the round spots due to the bubbles of air in the viscid mucus may be seen through the still transparent drumhead. In the influenzal cases a hemorrhagic bleb often completely covers the drumhead. After a day or two the posterior half of the drumhead becomes covered with dead, cracked epithelium, beneath which there is a serous infiltration. Politzer was the first to show that the light reflexes on the bulging portions of the posterior segment of the drumhead sometimes pulsate. The yellow purulent secretion behind the membrana tympani does not show as often as one might expect, on account of the swollen and reddened condition of the drumhead. Occasionally, however, a greenish-yellow bulging spot may be seen, and when it appears, perforation is imminent.

In diabetic patients, and occasionally in others, small interlamellar abscesses form in the posterior segment of the membrana tympani, or near the umbo. They are of the size of a millet-seed, and rupture early in the course of the disease.

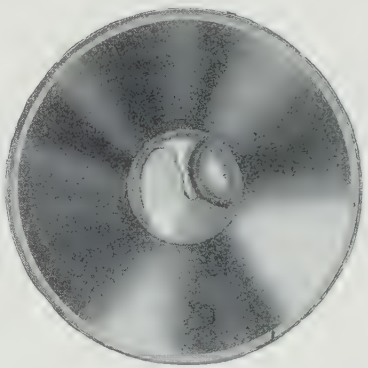
External Auditory Meatus.—The osseous portion of the meatus is almost always hyperemic, and is sometimes infiltrated, and more or less covered with blisters. The cartilaginous portion of the meatus is injected and painful in severe inflammations, the infection taking place through the numerous anastomoses of the capillary bloodvessels between the mucous membrane of the tympanic cavity and the skin of the meatus. The swelling and redness, or the so-called "sagging" of the postsuperior portion of the osseous meatus, near the membrana tympani (Fig. 415), occurs in those cases in which there is a marked suppurative process in the border mastoid cells (the cells along the posterior border of the meatus). When it occurs it is usually a positive indication for the mastoid operation.

Perforation.—Perforation takes place at the seat of one of the interlamellar abscesses, or at the most bulging portion of the drumhead, generally in the anterior half, although it may occur in the posterior segment. The size and shape of the perforation varies, usually being an ill-defined area with irregular edges, while in others it appears as a small dark round spot, with a pulsating drop of mucus covering it. In still other cases the opening cannot be located. Inflation sometimes

enables the observer to distinguish its edges. The same is true when the air is rarefied in the external canal with Siegle's otoscope (Fig. 410). The perforation is usually single, except in tuberculous patients, when it is multiple and near the margin of the drumhead (Fig. 417). In influenzal otitis, the perforation often occurs on the apex of a nipple-shaped elevation. Such a perforation is, therefore, significant of serious mastoid disease. Even under favorable conditions, the nipple-shaped perforation persists for some time. In those cases occurring independent of one of the infectious diseases, the perforation rarely exceeds the size of a millet-seed, whereas in cases secondary to the infectious fevers it may be so large as to destroy the entire membrana tympani. The membrana flaccida (Shrapnell's membrane) is rarely perforated in acute suppurative otitis media.

Secretions.—The secretions may be serous, seromucous, serosanguineous, seropurulent, mucopurulent, or mucohemorrhagic. If it is purulent, it often runs a more favorable course than the mucopurulent type. The quantity of pus, serum, and mucus varies greatly at different times, and one form of secretion may alternate with another. In nephritic, cachectic, leukemic, hemophilic, and traumatic cases, the hemorrhagic secretion is usually present.

FIG. 415



Bulging or sagging of the posterior superior wall of the meatus; an imperative indication for the mastoid operation.

Subjective Noises.—Pulsating noises sometimes occur in acute suppurative otitis media, although they are not always present. They are due to the increased pressure within the cavum tympani from the hyperemia and excess of secretion. The labyrinth is also hyperemic and somewhat infiltrated, the noises being thereby augmented. Autophony is sometimes present.

Hearing.—The hearing is impaired somewhat in proportion to the amount of congestion and secretion present. As the disease progresses, and the membrane becomes more congested, and the cavity filled with the secretion, the deafness, which at first was slight, becomes quite pronounced. In scarlatinal and diphtheritic infections involving the labyrinth, the deafness may be profound.

Hearing by bone conduction for the watch, tuning-fork, and acoumeter remains intact, except in those cases wherein the labyrinth is involved. In the Weber test the sound is lateralized to the diseased ear, except in the aforesaid labyrinth cases, in which it is lateralized to the sound ear.

Course.—Taking the perforation of the drumhead as one of the early milestones in the progress of the disease, we may subdivide it into three classes, namely: (*a*) Those cases running a very rapid and

destructive course, wherein the drumhead is perforated within the first one or two days; (*b*) those cases wherein perforation occurs on the third or fourth day (primary suppurative otitis media); (*c*) and the more chronic type, in which perforation occurs within the second or third week of the disease.

Perforation usually ameliorates the symptoms, especially the pain and temperature. Improvement does not always follow, however, as the mastoid antrum and cells may also contain pent-up secretions, and thus give rise to pain and elevation of temperature, in spite of the lowered tension within the tympanic cavity. The fever, headache, and subjective noises are also abated when perforation and drainage into the meatus take place.

After a variable time, the discharge ceases and the perforation closes. In the cases occurring independently of the infectious fevers, this will usually take place in from one to three weeks; sometimes, however, it may take as many months. In those cases due to the infectious fevers and to influenza (nipple-shaped perforation), the perforation only closes after a protracted period.

I have seen a fatal type of mastoiditis develop seven years after an attack of mild scarlet fever. In one case, seven years after the scarlatinal infection, cavernous sinus thrombosis complicating mastoiditis occurred, and was speedily followed by death. In another case, one year after a very mild attack of measles, suppurative labyrinthitis developed very suddenly, deafness being almost complete. Pachymeningitis, followed by death four days later, terminated the case. There is great danger for the safety of those patients whose ears become infected during the course of the exanthematous fevers. A latent or concealed inflammation so often persists, which after a lapse of a few years becomes very active and destructive. It is, therefore, always best to give a guarded prognosis in otitis media secondary to the eruptive fevers. The prognosis in those cases occurring independently of the exanthematous fevers is much more favorable.

Another type of otitis having dangerous tendencies, is that running an irregular or intermittent course. The discharge ceases, and then, after a variable interval, reappears. Pain also occurs at irregular intervals. In other words, the acute type becomes chronic and somewhat latent in character. Necrosis of the bony tissue takes place, and mastoiditis, complicated with sinus thrombosis, brain abscess, or meningitis, occurs.

Terminations and Sequelæ.—This phase of the subject is of great importance, on account of the apparent harmlessness of the disease in many cases, whereas it is in reality a most grave and destructive one. It is not so much the disease that is to be feared as its sequelæ. The terminations and sequelæ should engage the thoughtful consideration of the attending physician quite as much as the primary otitis. For convenience of discussion, Politzer's classification of the terminations will be followed:

(*a*) **Cure.**—That many cases terminate in a positive cure, no vestige of the disease remaining, cannot be questioned. That many are pro-

nounced "cured" when in reality a serious sequela is left as a heritage, is also unquestioned. A careful analysis of the case, its etiology, course, etc., should be considered in arriving at a correct conclusion as to whether or not it is "cured." What, then, are the points that should be considered in arriving at such a conclusion? In the first place, if the case is primary, or independent of a preceding infectious fever, and has run a mild and rapid course, and if there are no demonstrable ear lesions, it is safe to pronounce the case as probably cured. Such an opinion should, however, be based upon accurate and intelligent observations. I have seen many cases pronounced cured in which subsequent results demonstrated the opinion to have been erroneous.

(*b*) **Catarrhal.**—A catarrhal termination is not attended with immediate serious consequences, but it may in time produce pronounced impairment of hearing. The perforation may become completely closed by cicatricial tissue and a seromucous secretion, with slowly increasing deafness and tinnitus as the chief symptoms.

(*c*) **Adhesive Processes.**—This form of termination is comparatively common. The thick mucoid secretion or exudate becomes organized, the adhesive bands binding the ossicles to each other or to the wall of the tympanic cavity. The drumhead may also be involved by adhesions to the inner tympanic wall, forming ridges and folds toward the wall from which the adhesive bands spring. The deafness and tinnitus are usually progressive, although they may increase by bounds. In the earlier stages, bone conduction is increased, Rinné (see Functional Tests of Hearing) being negative, while in the more advanced stages Rinné is positive. The positive Rinné in the later stage is accounted for by the extension of the sclerotic process to the labyrinth.

(*d*) **Permanent Deafness.**—Permanent deafness is usually a result of the secondary infection from scarlet fever, measles, diphtheria, etc., the membrana tympani and ossicles being partially or entirely destroyed. I have seen cases, however, in which the drumhead and ossicles were entirely destroyed and the inner wall (promontory) of the tympanic cavity plainly visible, in which the hearing was remarkably acute. The chief loss of function seemed to be an inability to locate the direction of sound or speech. After once grasping the fact that they were being addressed, these cases seemingly hear with almost normal acuteness. Another cause of permanent, and often very pronounced, deafness is the panotitis of Politzer, wherein the whole auditory apparatus is involved in the infective process. In these cases there is caries of the bone separating the tympanic cavity from the labyrinth (promontory), or there is a perforation of the round window leading to the labyrinth. This condition is usually secondary to the infectious fevers.

(*e*) **Mastoiditis.**—While mastoiditis nearly always complicates middle-ear infection, it is not always severe enough to cause serious symptoms. In some cases, however, notably those due to the infectious fevers, influenza, and typhoid fever, the mastoid involvement often becomes the chief problem in the management of the case. In mastoiditis having its origin in influenza, the abscess is usually circumscribed, and is located

in the mastoid process, the tympanic cavity containing no pus. In children, the mastoid process is often perforated through the external plate, thus giving rise to a subperiosteal abscess.

(*f*) **Loss of Mucous Membrane, Ossicles, and Infection of the Labyrinth.**—Labyrinthitis, described under (*d*) Permanent Deafness, is found following mild infectious fevers, typhoid fever, and tuberculosis. The tympanic cavity is denuded of mucous membrane, and the ossicles are necrosed. A probe introduced into the cavity through the external meatus shows bare, comparatively smooth bony walls. The labyrinth may be exposed by necrosis of the promontory or inner wall of the middle ear, or the wall of the horizontal semicircular canal may be perforated. The hearing in these cases may not be as profoundly affected as in (*d*), except when the cochlea is involved.

(*g*) **Chronic Suppuration.**—This sequela is not so much to be dreaded as the more latent form, in which there seems to be a cure, when in fact necrosis may be steadily progressing. In the plainly manifested chronic suppuration the physician and patient are not so readily deceived, but recognize the possible danger still attending the further progress of the disease.

(*h*) **Death.**—A fatal issue may result early in the disease from meningitis, sinus thrombosis, septicemia, or brain abscess. The infection may reach the meninges through the labyrinth, the tegmen antri or tympani, or through one of the open sutures of the temporal bone in infants and young children.

Diagnosis.—The diagnosis of acute suppurative otitis media in the early stage is neither easy nor simple. The apparent difference between it and acute catarrhal otitis media is often so slight that only a careful and intelligent examination will enable the surgeon to make a correct diagnosis.

(*a*) **Pain.**—In suppurative otitis media the pain previous to perforation is very intense and boring in character, especially in children.

(*b*) **Temperature.**—The temperature ranges from 1° to 3°, or even more, above normal in children, but may not run so high in adults. In catarrhal otitis media the temperature does not usually exceed 1° or 2° above normal.

(*c*) **Appearance of the Drumhead.**—In suppurative otitis media before perforation, the drumhead is quite similar in appearance to that in catarrhal otitis media. The perforation may appear as a dark spot or it may not be visible. A pulsating droplet of mucus or pus is, however, significant of perforation. If the drumhead is destroyed, the red promontory may be seen when the pus is cleared away.

(*d*) **The Probe.**—The probe may be used to differentiate between a reddened promontory wall and a reddened drumhead. The promontory is firm and unyielding, while the drumhead is resilient. With the probe, a flake of mucus or pus may be brushed away, and thus show whether a perforation is present. Necrosis of the promontory or cochlear wall may also be demonstrated with the probe. In the acute stage

nystagmus, nausea, and vomiting may be present when the labyrinth is involved. (See Tests of Vestibular Apparatus.)

(*e*) **Inflation.**—Inflation of the middle ear and the simultaneous use of the diagnostic tube will produce a whistling tympanic murmur when perforation is present, and a soft, blowing tympanic murmur when the drumhead is intact. Inflation should be practised with caution in acute cases, as the infectious material may be forced into the deeper recesses of the tympanic and mastoid cavities. If during inflation, the distal end of the diagnostic tube is dropped into a basin of water, bubbles of air will arise in the water if perforation is present. A manometric tube partly filled with water and inserted into the external meatus during inflation will cause the column of water to rise in the distal arm of the U-shaped tube during inflation if a perforation is present.

(*f*) **Compression of Air in the Meatus.**—Compression of the air in the external canal will force air through the perforation into the middle ear. The sound may be heard by inserting one end of the diagnostic tube into the nose of the patient (one nostril being closed), the other end being placed in the external auditory meatus of the observer.

Prognosis.—The prognosis has already been quite fully considered under Terminations and Sequelæ.

Treatment.—The treatment will be considered in connection with the subject of middle-ear suppurations in general. A brief *resume*, however, will be given in this connection.

(*a*) Complete asepsis or cleanliness and drainage should be striven for, to prevent the otorrhea becoming chronic. To fail in this regard subjects the patient's life to great hazard. If thorough asepsis is maintained, a secondary staphylococcus infection will be prevented. Staphylococcus infection means chronicity. Do not allow it to occur.

(*b*) In the early stage, before perforation occurs, a 12 per cent. solution of carbolic acid in glycerin should be dropped into the meatus. It is also a valuable remedy after perforation, as it is hygroscopic, reduces the edema of the mucous membrane, and thus establishes a more rapid flow of blood through the tissues. The resistance of the tissues is thus raised and the infection checked.

(*c*) Early incision of the drumhead should be practised at its most bulging portion. The incision should be free and curved to allow of good drainage. Simple puncture, the so-called paracentesis, is never indicated. It is an obsolete procedure. Drainage is the object sought for, hence use the lance with a free hand. Incision also promotes the reaction of inflammation, and thus favors a speedy resolution (Fig. 416).

(*d*) If the secretion is thick and tenacious, the syringe may be used to remove it. A sterile alkaline solution should always be used for this purpose, as it thins the secretion and facilitates its removal.

(*e*) An aqueous solution of the peroxide of hydrogen may also be used to break down the secretion, after which it may be more readily wiped away with a cotton-wound probe.

(*f*) The cotton-wound probe should be used gently, but repeatedly, at each sitting. In the author's experience, this is the most effectual method

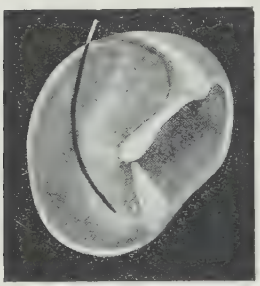
of removing the secretion in those cases in which the perforation is of large size.

(*g*) Inflation of the middle ear may be practised with caution after the pain and other acute symptoms have subsided.

(*h*) A safer procedure is to use suction with Siegle's otoscope in the external auditory canal.

(*i*) Constitutional treatment: Calomel may be given in $\frac{1}{10}$ grain doses three to ten times a day. For the relief of the pain, 1 grain of codeine or 3 to 6 grains of phenacetin, may be given. The epipharynx should be frequently gargled after the von Tröltsch-Swain method, the patient lying upon his back.

FIG. 416



A long, curved incision extending across the drumhead and into the meatus at the upper portion.

(*j*) Six weeks of daily inspection and appropriate treatment will, in most cases, result in a complete cure. Less faithful and intelligent attention will result in many cases becoming latent or chronic, with the usual sequelæ so unfortunate in their effects.

(*k*) In those cases in which there is sagging of the postsuperior meatal wall the simple mastoid operation should be performed at once. Delay is dangerous. If the infection is staphylococcal, the urgency for the operation is not so great as in streptococcus infection. In the latter type, local treatment is usually unavailing, surgical procedures being required to effect a cure.

(*l*) The ice-bag may be used over the mastoid process for one-half to two hours when great pain is present. If no improvement follows, it should be discontinued and operative measures considered. Discontinue the ice when pus flows freely and the pain subsides. If the infection is streptococcal, its use will be unavailing. If it is staphylococcal, it may abate the infective process.

(*m*) Artificial or natural leeches, applied over the mastoid process and in front of the tragus, afford the most effectual method of promoting the reaction of inflammation and aborting the disease. (See Chapter VII.)

ACUTE SUPPURATIVE OTITIS MEDIA IN INFANTS AND CHILDREN

In view of the fact that in 50 per cent. of the cases of measles in infants and young children there is an inflammatory affection of the middle ear, and that with all infectious diseases in young patients there is more or less inflammation of the ears, a brief consideration of these inflammations is in order.

The pathological changes found vary all the way from a simple catarrhal inflammation, with swelling and cloudiness of the mucosa, to infil-

tration and purulent secretion. This secretion is usually serous or seromucous, with some pus cells.

The embryological conditions influencing the occurrences of the process in infants are: (*a*) The presence of an opening in the upper or Rivinian segment of the drumhead, which does not always close before birth. In bathing, water may thus gain entrance into the tympanic cavity and excite an inflammation. (*b*) According to Weiss, the mucous membrane of infants is embryonic in type, and is, therefore, more liable to become infected.

The cachexia of infancy, bronchitis, the infectious fevers, and chronic intestinal catarrh are also special causes.

Coughing, vomiting, sneezing, and other violent respiratory efforts may force infected matter through the Eustachian tubes into the middle ears and excite catarrhal and suppurative inflammations.

Otitis media is sometimes present in the newborn, and is probably due to the forcible entrance of amniotic fluid into the middle ear during delivery.

Adenoids, enlarged or diseased tonsils, epipharyngitis, and coryza are common diseases of childhood, and contribute toward the causation of otitis media.

Symptoms.—In infants with cachexia there are often no subjective symptoms. Objectively, the drumhead may be a little reddened, especially about the short process and along the handle of the malleus. A small amount of slimy secretion may be found in the canal. It may be questioned whether the cachexia is the cause of the ear disease, or the ear disease is the cause of the cachexia. It is quite certain, however, that even a mild suppurative process in infants is quite sufficient to cause pronounced disturbances of nutrition. Every case of malnutrition, peevishness, twisting of the head, or dropping it to one side should lead to the careful inspection of the ears of these young patients. Boring the head, or occiput, into the pillow, hanging it to one side (affected ear), placing the hand to the affected ear, going to sleep when lying on the ear toward which the head is inclined, refusing to take the breast except on the side which allows the patient to lie with the affected ear against the bosom, all point to acute inflammation of the middle ear. The infant cannot tell of its sufferings, but if the physician carefully observes its actions, they will often speak louder than words.

In older children, the symptoms are more pronounced, and just prior to perforation of the drumhead the pain is often excruciating. There may be nystagmus, vomiting, unconsciousness, and convulsions. In other words, signs of labyrinthine and meningeal irritation are often present.

When perforation takes place, there is immediate relief, although the patient is by no means necessarily out of danger, especially if labyrinthine and meningeal symptoms are present.

The tendency to frequent relapses is a prominent characteristic of otitic inflammations in infancy and childhood. After the tenth to the fifteenth year of age this tendency is not so marked.

Treatment.—The treatment is almost the same as in adults, with the exception that tympanic inflation is usually followed by great relief. When the inflammation is suppurative in character, the external meatus should be thoroughly cleansed with cotton-wound probes. The same treatment described under Acute Suppurative Otitis Media and Acute Mastoiditis is applicable to these cases. The removal of adenoids, when present, is usually followed by great improvement or a cure of the otorrhea. Many cases of chronic otorrhea in children cease after the removal of the adenoids. If, however, the otorrhea is secondary to scarlet fever, measles, or diphtheria, it is often necessary to perform a mastoid operation to effect a cure. If nystagmus and meningeal symptoms were present, the case should be carefully watched and free drainage maintained, and, if necessary, suitable surgical procedures adopted.

CHRONIC SUPPURATIVE OTITIS MEDIA

Owing to the faulty instruction, or, more properly speaking, to the lack of systematic instruction in otology in most American medical colleges, false ideas are prevalent concerning the true importance of chronic suppurative otitis media. The acute exacerbation is the only phase that ordinarily attracts serious consideration. When we recall the fact that none of the prominent life insurance companies will accept an applicant who is affected with chronic or intermittent otorrhea, we are brought face to face with the business man's view of the disease. He has found after a careful study of the mortality tables, that applicants thus affected do not live to the full term of their natural lives. Both clinical observation and pathological findings bear out this conclusion. Clinically, we find chronic otorrhea attended with a sallow, muddy complexion and acute exacerbations, during which there is pain and mastoid tenderness, and an increased flow of pus, which subsides only to return again after many weeks, months, or years. In other cases, sinus thrombosis, septicemia, labyrinthitis, meningitis, brain abscess, etc., which often lead to a fatal termination, are associated. Bearing these facts in mind, and their relation to what seems to be a simple and harmless chronic otorrhea, it becomes apparent that chronic suppurative otitis media is not to be thought of as a trivial or an unimportant disease.

Symptoms.—The symptoms vary with the nature and location of the pathological process, as well as with its acuteness or chronicity. In some cases the signs of the ear disease are so latent that but little thought and less attention are given to them. In others, there is a constant or intermittent flow of pus or mucopus into the external canal, with occasional twinges of pain. In still others, there are acute exacerbations, characterized by profuse pus discharge, often admixed with blood, and attended with pain, mastoid tenderness, and swelling. The chief difference between the types is in the degree of obstruction to free drainage and in the virulency of the microörganisms in the tympanic cavity and mastoid cells. So long as there is free drainage, and there are

no virulent microörganisms jeopardizing the middle-ear and cranial contents, the symptoms are not alarming in character. On the other hand, when free drainage is interfered with and virulent infection supervenes upon the preëxisting less virulent infection, the symptoms assume a most aggravated and alarming character. In other words, the so-called chronic suppurative otitis media assumes the proportions of an acute mastoiditis with threatened intracranial complications.

The Latent Form.—The symptoms in this type of middle-ear suppuration are scarcely appreciable to the patient, as there is little discharge and no pain or tenderness over the mastoid process. The patient often says there is no discharge, nor has there been for many months or years. Ocular inspection, however, will often show a small amount of pus in the middle ear and external auditory meatus. The amount is so small that it does not reach the concha, but is evaporated in the meatus, the dried remains being thrown off with the cerumen and epidermis. In these cases there is a central perforation of the drumhead, the size varying from a millet-seed to almost the entire membrane (pars tensa), though frequently cases of latent otorrhea are observed in which the perforation is marginal.

The Chronic Discharging Form.—There is a profuse but intermittent purulent discharge, sometimes admixed with mucus and blood. Acute coryza, epipharyngitis, and exposure to inclement weather increases the amount of discharge and its purulency. Pain may be present, especially when aggravated by either of the foregoing conditions. There is, at these times, a slight tenderness over the mastoid process, especially over the antrum. Inspection of the fundus meati shows pus completely filling it, or oozing through the perforation in the drumhead. If the drumhead is largely destroyed, and the pus has its origin in the attic, it may be seen to trickle down the long process of the incus into the atrium of the middle ear. After removing all the pus from the middle ear, the promontory appears as a yellowish-red reflex. Granulations or polypi may be present, filling the middle-ear cavity, or even protruding into the external meatus.

I have seen cases in which the polypus protruded into the choncha of the auricle. When polypi are present, blood is often admixed with the secretions.

There is more or less elevation of temperature during the subacute exacerbations. The skin is yellow and muddy, the whites of the eyes are slightly discolored, and a feeling of lassitude and mental inertia possesses the patient.

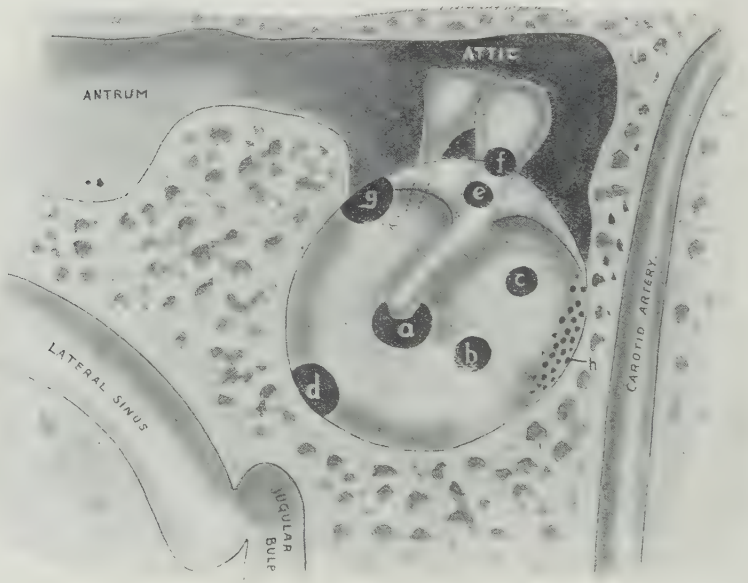
Chronic Otorrhea with Acute Exacerbations.—This form of chronic suppurative otitis media attracts attention on account of the exacerbations of pronounced pain, mastoid tenderness, and elevation of temperature. The patient and attending physician become conscious of the danger, which may have existed for some weeks, months, or even years previously. What was previously regarded as a simple harmless discharge is now recognized as a threatened mastoiditis. There is a profuse flow of pus, perhaps admixed with blood, the mastoid is tender to the

touch, either at its tip or over the antrum, and the temperature ranges from 1° to 4° above normal. There may be no distinct chill.

The patient complains of lassitude, and is disinclined to pursue his vocation. He may be apprehensive of impending danger.

Having thus characterized the more obvious symptoms of the three most common types of chronic suppurative otitis media, the further study of the signs of this disease, and their significance in estimating the nature and location of the pathological changes, will be based upon the location of the perforation in the drumhead.

Fig. 417



The significance of central and marginal perforations of the membrana tympani.

Perforations, their Location and Significance.—To Leutert, Zaufal and others we owe our knowledge of the pathological significance of the location of the perforations in the drumhead. It may be said, in general, that if the perforation is marginal, there is bone necrosis in the region of the perforation; and if the perforation does not involve the margin of the drumhead, but is near its centre, bone necrosis is absent, the case being one of simple suppurative otitis media. The information thus afforded, while not absolute, is nevertheless very valuable in arriving at a full knowledge of the case.

The Clinical Significance of Chronic Perforations of the Membrana Tympani.—A central perforation (Fig. 417, *a*, *b*, *c*) usually signifies inadequate drainage and ventilation through the Eustachian tube, the perforation occurring at the point of least resistance. A central per-

foration is rarely attended with necrosis of the bony walls of the cavum tympani or of the ossicles, and may be successfully treated without major surgical interference. According to Leutert, all central perforations indicate tubal infection.

(*c*) This is a central perforation (Fig. 417), located over the tympanic orifice of the Eustachian tube, and is the result of continual middle-ear infection from the tube. The Eustachian tube is probably infected from the epipharyngitis, if present. The epipharyngitis may be due to the presence of adenoids or their remnants, or to diseased tonsils, or to ethmoiditis and sphenoiditis. A perforation of the membrana tympani over the tympanic orifice of the Eustachian tube should, therefore, direct the attention of the aural surgeon to the epipharynx and the contiguous structures, rather than to the tympanic cavity. *A radical mastoid operation upon a case with a perforation at this point would, in all probability, fail to check the otorrhea.* An attempt to close the tympanic orifice of the Eustachian tube at the time of the radical operation would, in all probability, meet with failure, as the continued infection from the epipharynx would prevent closure. The rational treatment of such a case would be to cure the sinuitis, remove the adenoids and tonsils, or to adopt such other remedial measures as will cure the epipharyngitis.

A perforation of the inferior margin of the membrana tympani (Fig. 417, *d*) signifies necrosis of the inferior wall or floor of the tympanic cavity. The only vital structure in this region is the jugular bulb. As the bony wall separating the tympanic cavity and the jugular bulb is usually quite thick, the perforation may signify nothing more than necrosis of the floor of the tympanic cavity, a region which is accessible to curettement through the external meatus. In rare instances, however, the jugular bulb is separated from the tympanic cavity by only a thin bony wall, or the wall may be entirely absent. A marginal perforation at this point should, therefore, be regarded as suspicious of necrosis from jugular bulb disease, especially if septic symptoms are present. The exploration and curettement of the floor of the tympanum should in such cases be prosecuted with caution.

A perforation of the membrana flaccida immediately above the short process of the malleus (Fig. 417, *e*) usually signifies necrosis of the head of the malleus, a structure in close apposition to the perforation.

A marginal perforation immediately above the short process of the malleus and extending to the superior wall of the meatus (Fig. 417, *f*) usually signifies necrosis of the tegmen tympani (roof of the attic).

A perforation of the membrana tympani at the margin of the posterior quadrant of the membrana tympani (Fig. 417, *g*) usually signifies necrosis of the incus and of the walls of the antrum.

Numerous small perforations near the margin of the membrana tympani (Fig. 417, *h*) are usually significant of a tuberculous otitis media.

From the foregoing data it may be inferred that a central perfora-

tion signifies a simple infectious process in the cavum tympani, probably of tubal origin, whereas a marginal perforation usually signifies bone necrosis. Marginal perforations are, therefore, indicative of a more serious process in the middle ear (cavum tympani) than is indicated by a central perforation. The entire absence of the membrana tympani is equivalent to a marginal perforation, and is strongly suggestive of bone necrosis.

While the significance of chronic perforations is generally to be interpreted as given in the foregoing paragraphs, it should not be inferred that the location of the perforation is an infallible guide to the condition present in the middle-ear and mastoid cavities. All other clinical phenomena should be taken into consideration, and a conclusion be drawn from the entire symptom complex.

Prognosis as to Hearing.—In simple or central perforations the hearing may be but slightly affected after the suppurative process is relieved. In the complex or marginal perforations, with bone necrosis, the hearing is usually diminished after the radical operation, whereas it is greatly improved after the meatomastoid operation. The patient should be made to understand that, while every effort will be made to maintain or improve the hearing, the chief concern is to check, or to cure, the suppurative process, which, if allowed to run its course, may jeopardize both the health and life of the patient.

According to Clarence Heath, of London, many of the cases heretofore operated by the radical method may be cured by a less radical operation. (See Meatomastoid Operation.) In addition to a less radical procedure, he claims that the hearing is not only conserved, but that it is usually restored to near the normal. The author's experience with the meatomastoid operation is limited to twenty-five cases, and thus far the results obtained have been excellent. The twenty-five cases selected for this operation have been those in which the ossicles were not markedly necrosed, though the perforation in some was marginal. The prognosis as to the permanent cure of the disease by this operation is still open until further experience demonstrates its exact place in otological surgery. That the hearing is temporarily preserved, and usually greatly improved is fairly well demonstrated.

Treatment.—The treatment of chronic suppurative otitis media does not offer a brilliant therapeutic field. In spite of all that can be done with local treatment, the discharge often persists, or, if checked, recurs within a few weeks or months. Many so-called "cured cases" are in reality only latent, and with the first "cold in the head," or other local irritation, become active again. This tendency is so strong that many otologists have regarded the persistence, or the tendency to recurrence, as an indication for the radical mastoid operation. While this is probably an extreme view, it is, nevertheless, a more rational one than the view held by some, that most cases of chronic otorrhea may be cured by simple local treatment, or by simple operative measures through the external auditory meatus (Hotz, Theobald). As a matter of fact, each case should be diligently studied as to the local morbid conditions, and

as to the main etiological factors. Furthermore, the pathological laws underlying infectious processes in cavities lined with mucous membranes should be well considered. (See Chapter VI.)

The treatment of chronic suppurative otitis media will be studied, with the foregoing facts in mind, under the following headings

The Treatment of Chronic Otorrhea with a Central Perforation of the Membrana Tympani.—Chronic suppurative otitis media with a central perforation of the membrana tympani (Fig. 417, *a*, *b*, *c*) usually signifies a simple infection of the mucous membrane of the Eustachian tube and middle ear without involvement of the bony tissue of the tympanic walls, or of the ossicles, and is, therefore, often amenable to simple local treatment. Non-marginal perforations indicate a suppurative process in the Eustachian tube, hence the middle ear cannot be cured while the tubal infection continues. In such cases the first attention should be given to the Eustachian tube and the conditions giving rise to its involvement.

The otorrhea is perpetuated by the discharge of infected secretion from the Eustachian tube into the tympanic cavity, and cannot be cured without first overcoming the infection and discharge from this source. The mucous membrane of the Eustachian tube, when normal, is covered by ciliated columnar epithelium, which propels the secretions toward the pharyngeal orifice of the tube. In chronic infectious processes the cilia are lost, or their wave-like motion is inhibited, and the secretions flow in the direction of least resistance. The isthmus of the tube forms a partial barrier to the downward flow of the secretions from the tympanic end of the tube, hence they are retained in the tympanic cavity. The constant irritation of the membrana tympani opposite the tympanic orifice of the tube leads to perforation at this point. The first indication in these cases is to remove the cause of the tubal infection and inflammation.

If the tubal infection is due to a constriction at the isthmus of the tube, the tube should be dilated with bougies, and astringent and antiseptic solutions forced through it with a Weber-Liel catheter.

If the infection is due to the presence of epipharyngeal adenoids, or their remnants, they should be removed.

If the infection is due to an epipharyngitis, it should receive appropriate treatment.

Finally, if the tube is infected by the discharge from diseased nasal sinuses, especially the posterior ethmoidal and sphenoidal sinuses, this condition should receive appropriate treatment.

Having removed the cause of the tubal infection, that in the tympanic cavity tends to disappear with little or no other treatment. In some cases, however, the infectious process in the Eustachian tube is attended by such pronounced tissue changes that additional local treatment of the middle ear is necessary.

Removal of Adenoids.—If adenoids are present, it may be assumed that the ear disease cannot be permanently cured until they are removed; hence, the first indication is to remove them and then address the treatment to the ears. The tonsils may also require attention.

Epipharyngitis.—Epipharyngitis is usually caused by adenoids, hence the adenoids should be removed and the epipharyngitis treated with weak silver solutions. When overcome, address the treatment to the middle ear and Eustachian tube.

Sinuitis.—Chronic posterior ethmoidal and sphenoidal infection cause swelling and infection of the Eustachian tubes and thus perpetuate middle-ear infection. Give appropriate attention to these conditions and then direct the treatment to the ears.

If the above courses of treatment are consistently pursued, many cases may be cured without a mastoid operation.

Dry Gauze Dressings.—In 1880–82, Dr. Spencer, of St. Louis, advocated the use of strips of dry gauze in the treatment of acute and chronic suppurative otitis media. Since then the same method of treatment has been urged by Gradinego, Pierce, Gradle and others.

The fundus of the meatus should be mopped dry with a cotton-wound applicator before the strip of gauze is applied.

The end of the gauze is then carried to the membrana tympani with a probe packer (Fig. 418). The meatus is then *loosely* packed with the gauze and a small piece of cotton placed over it. The gauze should be removed every twenty-four hours and the secretions thoroughly removed with a cotton-wound applicator. A new strip of gauze is then applied as before.

Fig. 418

Bane-Allport gauze packer.

In some cases the drainage and protection afforded by the gauze leads to the rapid disappearance of the infection and to repair, the perforation often voluntarily closing by granulating from its edges. In other cases it persists, and may be closed by the application of a 33 per cent. solution of trichloracetic acid to its edges at intervals of a few days. No attempt should be made to close the perforation until the secretion is normal.

In addition to the foregoing method of treatment, alcohol in varying strength may be instilled into the middle ear through the meatus.

The middle ear may also be cleared by inflation through the Eustachian tube if the otorrhea persists after several treatments.

Treatment via Weber-Liel Catheter.—The local treatment of the infected Eustachian tube and tympanic cavity consists in the use of the dry gauze treatment and in the use of mild astringents and antiseptic solutions through the Eustachian tube, a Weber-Liel catheter being used for this purpose. The Weber-Liel catheter consists of a small, long, flexible rubber catheter, placed inside of a larger catheter of the usual length. The larger catheter is passed to the pharyngeal orifice of the tube, and the smaller one is introduced through it to the isthmus of the Eustachian tube. A small syringe filled with an alkaline antiseptic solution is then attached to the smaller catheter and

the fluid forced into the middle ear. This course of treatment, following the removal of the conditions causing the tubal and middle-ear infection, is often attended by a complete cure of the chronic otorrhea.

Treatment of Chronic Otorrhea with Marginal Perforations of the Membrana Tympani.—As marginal perforations of the membrana tympani usually signify necrosis of the ossicles, the bony tympanic walls, the tegmen tympani or tegmen antri, and the other contiguous bony structures, the treatment of chronic otorrhea thus characterized is not as simple as in central perforations. The same fundamental principles of treatment should, however, be observed. The drainage and the removal of the morbid material are absolutely essential to success.

The methods of establishing drainage and of removing the morbid material are radically different, for anatomical and pathological reasons, from those pursued in otorrhea with central perforations. It is obviously impossible to materially facilitate drainage by dressings in the external auditory meatus when the obstruction is in the antrum or aditus ad antrum. It is equally obvious that the morbid material cannot, under such conditions, be removed through the auditory meatus. Surgical measures are usually required in these cases, as follows:

1. When the perforation is just above the short process of the malleus (Fig. 417, *e*), the head of the malleus is probably necrosed, and the malleus should be removed. (See Ossiculectomy.) A 2 per cent. solution of the nitrate of silver may, however, be injected through the perforation to promote healthy granulation, with the hope of healing the diseased ossicle and thus avoiding the necessity of removing it.

2. When there is a perforation at the upper margin of the membrane (Fig. 417, *f*), and it involves not only the membrana flaccida but the superior wall of the auditory meatus, the tegmen tympani is probably necrosed. Even in these cases, ossiculectomy is sometimes attended by a cure of the chronic infection and otorrhea. If the floor of the attic is blocked, the removal of the malleus and incus may establish free drainage, and thus effect a cure. In other instances, ossiculectomy will not effect a cure, probably because the case is complicated by epipharyngitis, salpingitis, or necrosis of the antrum walls. Ossiculectomy is, therefore, only applicable to those cases in which the tegmen tympani is alone necrotic, the complicated cases being amenable to the meatomastoid and the radical operations.

3. When the chronic otorrhea is attended by a marginal perforation at the postsuperior quadrant of the membrana tympani, as shown in Fig. 417, *g*, necrosis of the antrum is probably present. The incus also may be necrosed. To establish drainage, and to remove the morbid material, either the radical or the meatomastoid operation should be performed. It is barely possible, however, that by irrigating the attic through the perforation, drainage may be established through the aditus ad antrum and a cure effected. To these cases the meatomastoid operation appears to be well adapted.

4. With a perforation at the inferior margin of the membrana tympani (Fig. 417, *d*), the necrosed bone may be removed with a curette introduced through the auditory meatus. If septic symptoms are present, the floor of the tympanic cavity should be cautiously explored, as the necrosis may be due to an extension from the jugular bulb. If septic symptoms are present in such a case, the rational procedure would be to perform either the radical or the meatomastoid operation, and then expose the sigmoid portion of the lateral sinus and the jugular bulb. If septic symptoms are absent, the floor of the tympanum should be explored with a blunt probe for necrotic bone, and if found, it should be carefully removed through the perforation with a bent curette. The perforation should be previously enlarged by two divergent incisions. After curettement, the meatus should be loosely packed with sterile gauze, as recommended in simple central perforations. The gauze should be removed daily, the meatus freed of secretions, and repacked with gauze, until the necrotic area is healed and the perforation closed. If the secretions disappear and the perforation persists, the perforation may be closed by the application of a 33 per cent. solution of trichloracetic acid to its margins.

5. Otorrhea attended by a perforation of the membrana tympani at its anterior margin usually signifies necrosis in this region. As the carotid artery passes upward through the temporal bone near the anterior boundary of the cavum tympani, curettement should be cautiously performed in this region (Fig. 417). (See Surgical Treatment.)

Other Methods of Treatment.—Curettage of the attic *via* the external auditory meatus should be undertaken with great reluctance and caution. If granulations are present, it is quite probable that the tegmen tympani is necrosed and that the granulations are thrown around and over it to wall off the invading pathogenic bacteria from the meninges. The removal of the granulation tissue without at the same time establishing free drainage of the secretions from the tympanic cavity might lead to infection of the meninges. Such a condition may be much more successfully, safely, and conservatively treated by either the radical or the meatomastoid operation.

The *alcohol treatment* has been held in high esteem in chronic suppurative otitis media. Its field of usefulness is chiefly limited to central perforations, especially after the causes of the tubal infection have been removed (see p. 757).

In otorrhea with a marginal perforation, alcohol only relieves the symptoms, but does not cure the disease.

The alcohol may be used in various dilutions, ranging from 25 to 95 per cent., beginning with the milder solution and gradually increasing the strength. The alcohol should be left in the cleansed ear for twenty minutes at each treatment.

Alcohol holding boric acid or iodoform in solution or suspension may be used in otorrhea with a central perforation, though it is probable that its therapeutic value is not increased by the addition of the boric acid or iodoform.

In fetid otorrhea the instillation of the *compound tincture of benzoin* may be used to remove the fetor. It is also an antiseptic and astringent, and acts favorably upon the diseased tissues. The fundus of the meatus should be mopped dry before applying the compound tincture of benzoin.

When there are exuberant granulations in the middle ear, a 95 per cent. solution of carbolic acid may be applied, care being exercised to prevent the acid coming into contact with the meatal skin. At the expiration of one minute, alcohol should be instilled into the ear to check the action of the acid, after which the ear should be mopped with a cotton-wound applicator. The meatus should then be loosely packed with dry, sterile gauze.

CHOLESTEATOMA

Cholesteatoma of the middle ear is characterized by the formation of masses of epidermoid cells arranged in concentric layers, between which are found cholesterin crystals.

Etiology.—About the year 1840, J. Müller described new formations in the temporal bone, resembling pearly growths. They were composed of concentric layers of epidermoid cells with cholesterin crystals between them. They are commonly found in the atrium and attic, and are covered with a delicate membrane which is closely adherent to the periosteum of the bone to which they are attached. This variety is known as primary cholesteatoma, as it seems to have its origin in the cavity where it is found. The secondary and most common type is due to an extension of the epidermis of the external meatus and membrana tympani into the middle ear through a perforation in the drumhead.

Primary Cholesteatoma.—Primary cholesteatoma is variously believed to be heteroplastic, possibly arising from the epithelium of the ductus vestibule; that is, it is a remnant of the second visceral cleft left behind after its closure. Mild inflammatory action in the middle ear favors its growth, whereas severe inflammation hinders it. Primary cholesteatoma is probably quite rare. Its existence might well be doubted if it were not for the fact that eminent observers have made full and detailed reports of such cases. Other equally eminent observers claim there is no such condition, all cases being secondary to suppurative processes in the tympanic cavities. Von Tröltsch, Habermann, Politzer and others hold this opinion.

Secondary Cholesteatoma.—This is the type found in practice, the primary form being chiefly limited to literature. The masses in all probability have their origin from extensions of epidermis from the external meatus and drumhead. The conditions favoring this extension are:

(*a*) A marginal perforation of the drumhead.

(*b*) A mild chronic suppurative inflammation of the mucosa of the middle ear.

(*c*) A fistulous opening in the posterior or superior wall of the meatus.
(*d*) Adhesions at the margin of the perforation.
(*e*) Adhesion of the end of the handle of the malleus to the promontory.
(*f*) Aural polypi.

Perforations in the posterior portion of the membrana flaccida are especially liable to be followed by cholesteatoma on account of the tongue-like thickened extension of epithelium from the superior wall of the meatus to the drumhead at this point. Politzer reports a case in which the growth seemed to have its origin in a fistulous opening in the mastoid process.

The cholesteatomatous masses are of a pearly gray color, and slightly lustrous. Upon section they are found to be composed of concentric layers of epidermic cells intermixed with detritus and cholesterin crystals. If the conditions are favorable, the masses grow larger and larger, and cause eccentric pressure atrophy of the bony walls of the cavity involved. In some cases the bone is necrosed, exposing the brain, lateral sinus, and labyrinth. The masses are broken down in their centres, richly odorous, and loaded with pathogenic microörganisms. The central breaking down is due to putrefaction.

Aural polypi, with mild suppurative inflammation, are often attended with cholesteatomatous formations. If there is an active or profuse pus discharge, the growths are checked or altogether dissipated. The free drainage incident to a profuse discharge seems to prevent the further inward extension of the epidermic process, the masses gradually disappearing, and the cavity healing with a layer of flat epithelial covering or matrix. The size of the cholesteatomatous masses varies from a hemp-seed to a large walnut. Their shape either conforms to that of the cavity in which they form, or they are round, oval, or very irregular in outline.

Extensions of the cholesteatoma into the Haversian canals have been demonstrated, which may, in part, account for the marked tendency to recurrences in spite of thorough operative interference. E. B. Dench has called attention to the presence of small masses of cholesterin crystals without epithelial cells, the etiology and pathology of which are not known. He reported two such cases operated by the radical method, with good results.

Symptoms.—The masses may be present for years without giving rise to distinct symptoms. Sudden swelling of the mass from the entrance of moisture into the external meatus, as from sweating, bathing, syringing, etc., may cause pressure symptoms, as pain and necrosis. In this there may be a feeling of fulness or pain in the affected ear, with headache, nausea, vomiting, nystagmus, staggering gait, fever, and aprosexia. The moisture causes the horny cells to swell, and the sudden pressure thus exerted causes the above signs of pressure and of intracranial irritation.

Inspection of the meatus shows it to be more or less filled with a pearly gray mass, admixed with granulations or aural polypi. If a portion is removed and placed in water, it appears as shreds of delicate tissue

with the golden grains of cholesterin, which are characteristic of this growth. If the mass is favorably located, it may be removed with the syringe or ear spoon. In other cases it is necessary to resort to the radical mastoid operation. Even then it may be necessary to repeat the operation one or more times before a satisfactory result is obtained.

The termination of cholesteatoma may be (*a*) by epidermization after the spontaneous or instrumental removal of the mass; (*b*) by forcing it through the Eustachian tube into the epipharynx, or into the maxillary articulation through the anterior wall of the meatus; (*c*) by its breaking through the walls of the semicircular canals (Jansen); (*d*) in some cases by pushing its way through the external plate of the mastoid process and presenting the appearance of a mastoid abscess; (*e*) in still other cases by causing necrosis of the tegmen antri and tympani and causing death from involvement of the cranial contents; (*f*) sepsis arising from the absorption of the retained secretions, causing death; (*g*) and from meningitis, brain abscess, sinus thrombosis, or thrombosis of the jugular vein with a similar result.

Diagnosis.—The diagnosis may be made by the removal of the growth and subjecting it to microscopic examination. It may be removed with a curette, probe, or syringe when the growth is in the middle ear. If in the antrum, it can only be removed by a mastoid operation. Sydacker has called attention to the sedimentation of the washings of the ear, which, when microscopically examined, show the epithelial cells with nuclei staining very faintly. Particles of bone dust are also shown as highly refractile crystals. Bruhl and Politzer have called attention to the use of a chloroform solution of the cholesteatomatous masses in which the cholesterin produces a greenish discoloration.

Prognosis.—The prognosis is bad. In those cases in which there is a spontaneous or instrumental expulsion of the cholesteatoma, the cavity usually becomes refilled. Even after the most thorough radical operation, the disease may persist. This is not at all difficult to understand when we recall the fact that the cholesteatoma forces its way into the Haversian canals of the bone, thus effectually forming focal centres from which it may extend again. Sac-like prolongations into the bone have also been observed, thereby making it difficult to entirely eradicate the process. The uncertainty of cure leaves the possible complications, as meningitis, brain abscess, pyemia, sinus and jugular thrombosis, a menace to the health and life of the patient. A cure is, however, usually effected, and we are warranted in attempting thorough surgical measures for its relief.

Treatment.—The treatment in uncomplicated cases may be begun by the removal of the cholesteatoma through the perforation in the drumhead with small curettes, ear hooks, etc., or with a syringe. In some instances it is found to be advantageous to force sterile fluid through the Eustachian tube into the middle ear, thus getting the force of the stream of water behind the mass, and forcing it into the external meatus.

Should polypi be present, they should be removed. If there is necrosis of the ossicles, they should be removed. Adhesion of the edges of the

perforation to the inner wall of the tympanum or adhesion of the end of the handle of the malleus to the promontory should be overcome. After having removed the tumor the parts should be dusted with an antiseptic powder.

Should these simple measures prove ineffective, recourse must be had to the radical mastoid operation, elsewhere described in this work. The meatomastoid operation is not indicated, as the chief object of this operation is to preserve or improve the hearing. In these cases this object is defeated by the unavoidable dislocation of the ossicles in removing the cholesteatoma.

CHAPTER XLIV

THE SEQUELÆ OF SUPPURATIVE OTITIS MEDIA, MASTOIDITIS AND CHOLESTEATOMA. SUPPURATION OF THE LABYRINTH

DISEASES OF THE MASTOID PROCESS

Primary infection and inflammation of the mastoid process is very rare. Disease of the mastoid is usually secondary to a suppurative process in the middle ear, but there are cases of pneumococcus, and more especially influenza infection, which sometimes appear in the mastoid process without first affecting the middle ear. As a matter of fact, all, or nearly all, suppurative middle-ear inflammations probably also involve the mastoid cells.

It is difficult to separate the suppurative processes of the middle ear from those of the mastoid cells. Clinically, the disease is subdivided upon an arbitrary basis according to the focal manifestations present. The anatomical distribution of the pneumatic spaces of the temporal bone is so complex that it is advantageous to subdivide suppurative inflammations within them according to the focal centre of involvement, while, on the other hand, it is more rational to regard the process as one disease regardless of the focal symptoms. The antrum is perhaps the axial centre of the pneumatic spaces of the ear, the mastoid cells communicating with it, while the attic and atrium (middle ear) communicate with it anteriorly through the aditus ad antrum. If the case requires external surgical treatment, it is most centrally attacked by way of the antrum, the operative field being extended posteriorly into the mastoid cells and anteriorly into the middle ear, according to the conditions present. If the disease is focalized in the middle ear without mastoid symptoms, it may be regarded as middle-ear disease. In those acute cases terminating without focal mastoid symptoms, it has been customary to speak of them as acute otitis medias, regardless of the fact that the mastoid cells were also involved.

With this understanding, the various diseases of the mastoid process will be described.

ACUTE SIMPLE MASTOIDITIS WITHOUT INTRACRANIAL LESIONS

Symptoms.—It is probable that in nearly every case of acute infection of the middle ear, the mastoid cells and antrum are also involved. It is chiefly in those cases in which free drainage is interfered with that the mastoid symptoms become manifest. These symptoms are chiefly

those of pressure from retention of the secretions within the cells. They are pain, redness, swelling, and tenderness upon pressure or percussion over the mastoid process. When such symptoms supervene, the original disease sinks into a place of secondary importance, while the secondary condition comes forward as the object of greatest interest. The disease is no longer called otitis media, but is called mastoiditis.

There is a sudden rise of temperature accompanied by rigors of varying intensity. Many cases, however, have but slight elevation of temperature at any time during the disease. In others, the rise is as high as 104° F.

The pain originates behind the auricle and radiates toward the teeth and shoulders (Politzer), the occiput, neck, and face. Mastication may be painful on account of an involvement of the bony portion of the external meatus, which is in close proximity to the glenoid fossa

The sternocleidomastoid and the other muscles of the neck attached to the mastoid account for the pain upon movements of the head. Torticollis may be present, and is due to a fixation of the muscle to avoid pain upon movement. It has been shown by others (Broca and Lubet-Barbon) that it is sometimes due to enlargement of the cervical glands and to infection from measles, in which otitis media was not present. In measles the torticollis is probably due to glandular enlargement from infection.

Schwartze called attention to the intolerance of pressure over the whole mastoid, but more particularly immediately below the zygomatic ridge (antrum), as a symptom of mastoiditis.

The skin over the mastoid process may become red and swollen. In some cases the auricle stands forward, even approaching a right angle to the side of the head. In these cases a subperiosteal abscess is present.

The aural discharge may be scanty or profuse. Redness and swelling of the posterior wall of the external meatus near the drumhead are commonly present. This condition is variously spoken of as the "dip," "chute," or "bulging" of the postsuperior wall. Under the pathology of the mastoid reference has already been made to the presence of pneumatic mastoid cells (the border cells), which are found between the antrum and meatus. These break down, and the retained secretions cause the wall to thus "dip" or "bulge." This sign is pathognomonic of mastoiditis of a destructive type, and is, therefore, a strong indication for an immediate operation.

The diagnostic value of this sign has been emphasized by Schwartze, Macewen, Holmes, Sheppard, Duplay and many others. Politzer thinks it is not necessarily an indication for the mastoid operation, while Schwartze, Broca, and Lubet-Barbon hold the contrary view.

Delay in operating subjects the patient to almost certain danger, even though it does not become apparent for years. The author can recall but one case (following an attack of influenza) in which the "dip" and all other signs of middle-ear and mastoid disease seemed to disappear. The word "seemed" is used advisedly, for there is little doubt as to a subsequent recurrence in such cases. There are exceptions to all rules

and the case just mentioned was probably one of them. Nevertheless, the rule and not the exceptions should guide us.

A central perforation of the drumhead nearly always exists. It is usually small and filled with pus and debris, which pulsates synchronously with the heart beat. Should the infection be very intense, great destruction of tissue may result, in which event the perforation may be marginal.

Granulations sometimes protrude through the opening and block the discharge of the secretion. The removal of the granulations is often sufficient to establish free drainage and relieve the acute mastoid symptoms. It may be doubted whether it really cures the mastoiditis, as this may remain in a latent form for years before culminating in an alarming exacerbation.

In still other cases the perforation is large and discharges but little pus. In these cases the aditus ad antrum is obstructed, and pain is pronounced. This is of interest as a diagnostic and prognostic point. It enables the attending physician to locate the obstruction prior to the operation, and to determine whether relief may be expected from a simple middle-ear operation (incision of the membrana tympani) or whether it will be necessary to perform a postauricular mastoid operation.

Spontaneous cures should be looked upon with suspicion, as in nearly every case it amounts to nothing more than a remission. Politzer, Schwartze, Duplay, Holmes, Ballenger, Stucky, Macewen, Dench, St. John Roosa, Hollinger, Pierce, Whiting and many others report recurrences in cases which had seemed to be cured.

One should be extremely modest in claiming to have "cured" mastoiditis without surgical intervention. That such terminations occur cannot be denied, but they are rare.

Treatment.—If the case is seen before spontaneous perforation of the eardrum has occurred, the drum should be freely incised at the point of greatest bulging. This is done to promote the reaction of inflammation and to relieve the pressure and the tissue necrosis. The tissues in the presence of an acute infectious process are very susceptible to necrosis while pressure is maintained, hence the necessity of an early incision. The incision should be a long and curved one, so as to make as free an opening as possible. Some writers advise carrying the incision into the meatus, thus cutting through the annular plexus of vessels surrounding the attachment of the membrana tympani. The free bleeding thus produced acts favorably upon the progress of the inflammatory process; that is, it promotes the reaction of inflammation and favors free drainage. Some writers condemn the extension of the incision through the annular plexus of vessels, on account of the liability of extending the infection through these vessels. If there is a virulent streptococcus infection, the incision should not be thus extended, while in the milder infections it is safe to do so. The author does not often carry the incision into the external meatus. If the destructive process is not great, there is no necessity for so doing, whereas if it is great, there are dangers attending such a procedure.

Cold applications by means of an ice-bag or a Leiter coil may be made over the mastoid process if the case is seen within thirty-six hours of the onset, and if there is great pain and scanty discharge of pus. Cold reduces the inflammatory reaction, diminishes the swelling of the mucous membrane, and thus overcomes the obstruction to the flow of the secretions. If these applications fail to remove the tenderness and pain, and to establish a better discharge of secretions, they should be discontinued and leeches applied. Leeching is much more efficacious than ice. In some cases the cold applications mask the symptoms and lead the surgeon to believe the disease is conquered. The real problem in acute mastoiditis is not to bring about an abatement of the acute symptoms, but to relieve the patient of the suppurative process by promoting the reaction of inflammation. Even though the acute symptoms disappear and the patient appears to be well, but still has an ear discharge, a cure is not effected. Too much attention has been given to the relief of the acute symptoms, and too little to the cure of the suppurative process. The acute symptoms will usually subside if nothing is done for the patient, but in most cases less damage follows if appropriate attention is given during their manifestation. Eradication of the suppurative process should be the ultimate aim of the treatment. The attending surgeon should not be satisfied, therefore, to relieve the pain, redness, tenderness, and temperature, but should also institute such remedial measures as will modify the acute symptoms and at the same time eradicate the infection.

To accomplish the foregoing results, it may become necessary to perform a mastoid operation, which, if done at a sufficiently early period, need not be an extensive or formidable affair. On the other hand, the delay of a few days or weeks may make it necessary to perform a radical operation. The cold applications, the incision of the eardrum, leeching, etc., should therefore be tried early, so as to determine as quickly as possible whether the disease can be aborted. If the mastoid is still tender upon pressure and the discharge continues, there is a strong probability that the acute process will merge into a chronic one if surgical interference is not instituted. The point to be emphasized is that the simple operation may be performed within the first three or four weeks of the onset of the disease, whereas if delayed to a later period the meatomastoid operation may be necessary. There are hundreds of cases of chronic otorrhea which would never have existed had they been operated on sufficiently early, or had the meatomastoid or the radical operation been performed when, on account of delay, a cure by the simple mastoid operation was impossible. Just when to operate, and the kind of an operation to perform, is the great problem in acute suppurative otitis media complicated by mastoiditis. It should also be stated in this connection that all cases do not need to be operated upon. Many get well without such interference. If the pain over the mastoid persists after the incision of the membrana tympani and the use of the leeches, an operation is indicated; that is, the disease will probably persist as a chronic otorrhea unless an operation is performed. The object of the operation is to prevent

further mischief, rather than to avert immediate danger. It is not good practice to wait for dangerous symptoms, as the mortality under these conditions is much higher. Chronic otorrhea is a signal of impending disaster, and every effort should be exerted to prevent it, even though a mastoid operation is necessary to accomplish it.

The Leiter coil should be connected by rubber tubing with a tank or bucket of iced water, and the water passed through it by siphonage and allowed to escape into a vessel through another tube attached to the opposite end of the coil. The iced water should be renewed each time the tank becomes empty, and continued for about one hour, or until the pain ceases and the purulent discharge becomes more profuse.

An ice-bag filled with cracked ice, and fastened over the mastoid process by bands of linen, may be used instead of the Leiter coil. The ice should be renewed as often as it becomes melted.

Hot irrigations of the bichloride of mercury solution, 1 to 5000, may be used every hour to promote the reaction of inflammation.

Bier's treatment by constriction of the neck, if judiciously applied, often exerts a favorable influence upon the course of the disease. The patient should be placed in a bed, the foot of which is raised several inches from the floor, and an Esmarch elastic band applied around the neck. It should produce no pain or discomfort, and only slight cyanosis of the face. It should be applied four times daily, with two-hour intervals between applications. If the bandage is applied tight enough to produce pain, it may do great damage.

The object of Bier's treatment is to promote the reaction of inflammation; that is, to increase the passive hyperemia and the migration of leukocytes, so as to remove the bacteria and their toxins. Ice, in view of these principles, is usually not indicated, as it diminishes the reaction of inflammation. Encapsulated organs, such as the mastoid, however, sometimes become so distended by inflammatory swelling that the flow of blood through them is very much blocked. Ice relieves the distention and establishes the flow of blood, and is indicated under the circumstances. When the distention or pressure symptoms (excessive pain and scanty discharge of pus) are relieved, ice should be discontinued and measures adopted that promote the reaction of inflammation.

Other methods of promoting the reaction of inflammation are leeches, light, heat, hot poultices, etc. (See Chapter VII.) Of these, leeching, the leukodescent light, and Bier's treatment are of special value in the treatment of acute mastoiditis.

Leeching should be more generally used, as it is one of the best means of promoting the reaction of inflammation. Cases following measles running a temperature of 102° to 104°, often rapidly subside after the use of leeches.

Should these simple measures fail, the simple mastoid operation should be performed. (See Chapter XLVIII.)

Subacute Mastoiditis.—This form of mastoiditis has been referred to under Acute Mastoiditis as the stage following the subsidence of the acute symptoms. It should be regarded as a chronic disease even if

the conditions present are of recent origin, as it only responds to treatment suited to chronic cases. The infectious agent is usually the staphylococcus, the usual germ of chronic suppuration.

Subacute mastoiditis is, therefore, the persistent remains of an acute mastoiditis, in which the more active microörganisms have disappeared, the staphylococcus perpetuating the inflammatory process. It is amenable to such treatment as is recommended for chronic mastoiditis.

ACUTE PERIOSTITIS OF THE MASTOID PROCESS; SUBPERIOSTEAL MASTOID ABSCESS

Subperiosteal mastoid abscess is characterized by a pronounced bulging outward of the affected ear. The auricle at its superior portion stands well out, while its entire free border is almost at right angles to the plane of the side of the head. In other words, the outline of the ear as seen from either the front or the rear, falls from the upright toward the horizontal plane of the head.

Upon manipulation, the swelling above the auricle fluctuates more or less in proportion to the amount of pus beneath the soft tissues. Duplay says that before the pus forms externally, one feels the elevation and depression, under pressure, of the external table of the mastoid.

The alarm occasioned by an abscess of this type is out of proportion to the danger attending it, as it rarely proves fatal.

Etiology.—It usually has its origin in an infectious otitis media, which extends to the antrum and mastoid cells. In young children, the middle ear and antrum alone are involved, as the mastoid cells are not yet formed.

The periosteum over the squamous portion of the temporal bone is more easily separated (Macewen) than over the mastoid process. In consequence, the pus passes upward and causes the outward bulging of the upper portion of the auricle.

Chronic otitis media suppurativa predisposes to the formation of the abscess. A low stage of vitality is usually present. It occurs more often in children, on account of the loose articulation of the bony plates.

Treatment.—In acute cases it is often only necessary to make a free incision through the skin and periosteum covering the mastoid process and evacuate the purulent accumulation. As the abscess is of otitic origin, it may in some cases be necessary to perform a mastoid operation either at the time of the incision or subsequently. In chronic subperiosteal abscess, the simple incision (Wilde's) may not effect a cure, as the ear disease is well established and may require an operation.

CHRONIC MASTOIDITIS

Symptoms and Diagnosis.—Chronic mastoiditis is not necessarily characterized by any special symptom other than those present in chronic suppurative otitis media. Mastoid pain and tenderness and

other focal symptoms are often absent. The mastoid bone often undergoes an eburnizing sclerosis in the course of the disease, the cortex becoming quite dense and the cells replaced by dense bone. It is not unusual to find the mastoid process with a few small cells, while the remainder of the process is as hard as ivory. In this case, the antrum may be smaller than normal. When the cortex is dense, external, pressure symptoms are not present. The cranial aspect of the mastoid process does not always undergo the sclerosing process; hence, intracranial complications, as sinus thrombosis, meningitis, brain abscess, etc., may be the first focal symptoms to develop. A neuralgic pain often accompanies the osteosclerosis of the mastoid process, which may be relieved, according to Schwartze, by the removal of a wedge of bone from the process.

The inspection of the drumhead and the middle-ear cavity often affords useful information as to the diagnosis. The drumhead is usually almost or entirely destroyed. Usually the short process and the head of the malleus are present, while the handle is gone. The incus is often entirely destroyed, though it may be present in the more recent cases. A fetid purulent secretion fills the meatus and the middle-ear cavity. When this is removed and suction is applied with Siegle's otoscope, the secretion may be seen trickling from the attic into the atrium. After the middle ear cavity is thoroughly cleansed, a fetid odor from the foul pus which continues to enter the antrum from the inaccessible attic and antrum is present, giving evidence of mastoid involvement.

Another evidence of chronic mastoiditis is the necrosis or entire destruction of the incus. In the section on perforations of the eardrum, attention was called to the significance of a marginal perforation in the postsuperior quadrant of the eardrum and the associated necrosis of the incus, as signs of necrosis in the antrum. An increased quantity of purulent secretion is also a sign of mastoid involvement, although such an involvement may be present with scanty discharge. Macewen calls attention to the fact that in many cases the discharge is so slight as to escape attention. In some of the cases, granulations or polypi are the only evidence of mastoid disease. The attachment of the polypi, when examined with a delicate curved probe, may be traced to the attic. Polypi generally signify bone necrosis. If, after cleansing the atrium of all secretions, suction is applied through the Siegle otoscope, and pus trickles down one of the fragments of the ossicles, attic and antral involvement may be safely inferred. The presence of a persistent purulent discharge unchecked by local treatment is fairly good evidence of chronic otitis media plus mastoiditis. Macewen also calls attention to the fact that chronic suppuration of the middle ear extending over a period of two or more years is usually attended with necrosis. Neuralgic pains in the mastoid region occur in those cases attended with eburnizing osteosclerosis of the mastoid process. In cases in which acute exacerbations occur, there may be headache, especially at night. The mastoid skin may be slightly red, swollen, and hot, and the tem-

perature rises 1° or 2° above normal. The meatus is slightly swollen and hyperemic, and the postsuperior portion near the eardrum is tense and swollen or distinctly bulging. A cessation or diminution of the discharge is attended with pain, and signifies an obstruction to the discharge, the obstruction being due to acute swelling of the mucosa or to the formation of polypi.

The progress of the disease varies greatly in different cases. In some it runs a long and uneventful course without distinct symptoms other than the intermittent discharge. In others, acute exacerbations occur every few weeks or months with the acute symptoms described under acute mastoiditis. In still others the discharge is so slight as to escape attention, unless the attic of the tympanum is explored with a probe. Any of these types may develop one or more of the labyrinthine or intracranial complications and become a very serious disease.

Caries and necrosis of the mastoid process frequently follow the retention of the purulent secretion. Most cases of two or more years' duration are thus affected. Such destruction may take place without marked symptoms. The insidious progress of the disease makes it a formidable process. As Macewen has so well said, one with a chronic otorrhea is likened unto one with a charge of dynamite in the head: he does not know when it will explode. Safety lies in removing the "charge" or diseased process. Tuberculous patients are especially subject to caries and necrosis, and do not heal so readily after operation. One of the author's cases on whom a radical operation was performed, could not be removed from the hospital for six weeks. Subsequently a secondary operation was performed, and it was again six weeks before it was possible to remove her from the hospital. At the second operation, Thiersch grafts were applied, with success, the entire cavity being thus covered by epidermis.

In caries and necrosis, careful examination will generally develop tenderness upon pressure, as the periosteum is apt to be swollen and inflamed. If in such cases the temperature is recorded every four hours, the record will show a typical septic curve. In cases attended with necrosis, paralysis of the facial nerve may be present. A bony sequestrum sometimes becomes separated and may be removed through the meatus. Goldstein reported a case in which the entire cochlea was exfoliated.

Prognosis.—The prognosis varies with the focal centre of the disease, the extent of the necrosis, and the presence or absence of intracranial involvement. When there is free drainage and only the mucous membrane is involved, the disease is not essentially a serious one. When extensive necrosis and intracranial complications are present, the danger to life is imminent. Chronic sepsis, as evidenced by a yellow pasty skin and an increased leukocytosis, while not serious, undermines the general health and paves the way for the development of other serious diseases. According to T. Mark Hovell, attacks of partial or complete unconsciousness, restlessness, and feverishness are always of grave import when occurring in a person suffering from disease of the mastoid process.

Treatment.—The local medical treatment of chronic mastoiditis is the same as that given for chronic suppurative otitis media. When this has been tried for a few weeks without effecting a cure, the mastoid antrum and cells and the middle ear may be opened. The object of this mode of treatment is to (*a*) establish free drainage, and (*b*) remove the morbid material, and establish the reaction of inflammation.

General Indications for the Radical Mastoid Operation.—There are practically but three general types of mastoid operation now practised: one, the simple mastoid operation for acute mastoiditis, wherein only the mastoid antrum and cells are opened; another, the radical mastoid operation for subacute and chronic mastoiditis, wherein the mastoid antrum and cells and the middle ear are thrown into one large irregular but freely communicating cavity; the other the meatomastoid operation, which may sometimes be used instead of the radical operation. The indications for the mastoid operations are in general those phenomena present in a persistent otorrhea which do not yield to local treatment (including the associated nasal and throat diseases) or which do not yield to operations through the external auditory meatus. The more specific indications are as follows:

1. Persistent tenderness over the mastoid process, with or without copious ear discharge.
2. Persistent ear discharge and polypi.
3. Fistulous opening into the roof or postsuperior wall of the external auditory meatus.
4. Caries of the attic, as shown by probing or by bone dust in the ear washings.
5. Facial paralysis.
6. Labyrinthine involvement, as shown by nystagmus, dizziness, nausea, staggering gait, and profound deafness.
7. Chronic ear discharge with neuralgic pains over the mastoid process.
8. Chronic ear discharge and septicemia.
9. Intracranial complications and a history of chronic otorrhea.

These and other signs may indicate the same type of mastoid operation. In view of the fact that life insurance companies refuse to insure persons affected with chronic otorrhea, the otorrhea alone may be a positive indication for the radical operation.

CHAPTER XLV

PRINCIPLES OF TREATMENT AND GENERAL CONSIDERATIONS IN SUPPURATIVE OTITIS MEDIA

THERE are four cardinal principles to be considered in the treatment of suppurative inflammations of the middle ear and mastoid cells, namely: (1) The promotion of the reaction of inflammation to aid Nature in combating The host of invading pathogenic microörganisms; (2) the establishment of free drainage and the reduction of pressure; (3) the removal of the morbid material; and (4) the maintenance of asepsis while repair is taking place.

1. **Promotion of the Reaction of Inflammation.**—As shown in Chapter VI, on inflammation, the reaction of inflammation is a beneficent process, the object of which is to combat the infectious microörganisms. It is a threefold process, namely: (*a*) Increased hyperemia, (*b*) increased nutrition, and (*c*) increased leukocytosis in the affected tissues.

The increased hyperemia floods the tissues with nutrition and thus raises their resistance. The increased migration of leukocytes into the tissues provides a fighting force which destroys the pathogenic bacteria and disposes of the dead cells of the tissues. As the reaction of inflammation is usually inadequate to successfully and quickly destroy the pathogenic bacteria, the therapeutic indications are to adopt measures which will increase or promote this reaction. Various modalities may be used for this purpose, some of which are, for anatomical and physiological reasons, especially well adapted to the treatment of the ear. (See Chapter VII, also *Vaccine Therapy*, at end of Chapter X.)

As stated in Chapter VII, heat, irrigation with alkaline solutions, incisions, leeching, massage operations, and radiant energy may be used to promote the reaction of inflammation.

Heat has long been used in the treatment of inflammation. Everyone has observed the increased redness of the skin under its influence. The hyperemia thus produced increases the nutrition, and it is now believed increases the migration of leukocytes into the tissues.

There are differences in heat, as there are differences in silk and calico. Heat is produced by a wide range of vibrations. Some wave-lengths of wide amplitude and slow vibration produce heat of slight penetrating power. Other wave-lengths of short amplitude and rapid vibration produce heat of high penetrating power. The shorter the wave-length and the more rapid the vibrations the higher the penetrating power. Heat from a hot-water bag or low candle-power incandescent lamp is of long wave-length and slow vibration, and, therefore, of slight

penetrating power. Heat from a 500 candle-power incandescent lamp is of short wave-length and rapid vibration, and is consequently of high penetrating power. The therapeutic value of heat is proportionate to its penetrating power. In selecting the modality for the application of heat these principles should be borne in mind. If the inflammation is superficial, a hot-water bottle or a low candle-power (16 to 100) lamp may be used, though a higher candle-power lamp will produce better results in a shorter time. If the inflammation is deep-seated, a high candle-power incandescent lamp (300 to 500 candle-power) or an arc light is indicated.

Radiant light as given by the leukodescent lamp is a remedy of some value in suppurative otitis media. It not only gives off heat of high penetrating power, but it gives off rays possessing a high degree of chemical activity. The spectrum of the leukodescent lamp is rich in the blue violet rays which effect chemical changes in the tissues exposed to them. Such a lamp is, therefore, a mechanical device furnishing two powerful therapeutic agents—namely, heat with high penetrating power, and blue-violet rays of chemical activity. In the opinion of the author, however, the leukodescent light is not as good or as quick a remedy in acute suppurative otitis media as incision of the membrana tympani and leeching. The progress of the disease is so rapid, and the structures of such vital physiological importance, that it is imperative that immediate improvement be obtained.

Incision of the inflamed tissue has long been a therapeutic measure of acknowledged efficacy. In the treatment of acute catarrhal and the pre-perforative stage of suppurative otitis media, incision of the membrana tympani is one of the most efficient modes of treatment. The good effects following such an incision are not altogether due to the increased hyperemia and leukocytosis, though this influence is greater than is generally believed. In addition to the increased reaction of inflammation, the incision establishes free drainage, relieves the pressure, and favors the removal of the morbid material.

Incision of the membrana tympani is an almost ideal therapeutic measure in the early or pre-perforative stage of acute suppurative otitis media, though it is of little value in the later stages of the disease, and in the chronic type. Little can be done by promoting the reaction of inflammation in chronic suppurative otitis media. In such cases the establishment of free drainage and the total removal of the morbid material should be accomplished. In acute cases, the incision of the membrana tympani should be long and curved, or V-shaped, to permit the secretions to flow through it.

Leeching is another old and all but discarded remedy in the treatment of acute inflammation. In the author's hands it has proved one of the most satisfactory methods of combating acute catarrhal and suppurative otitis media. It is best to apply from three to five leeches over the mastoid process and one to the tragus in front of the ear. If applied in the pre-perforative stage, or when the mastoid is swollen and tender, or when pain is present, the improvement is usually prompt, the case often proceeding toward rapid resolution.

Leeching increases the hyperemia and the migration of leukocytes into the inflamed tissues, and thus favors the destruction of the pathogenic bacteria and the repair of the tissues.

Artificial leeching is, perhaps, of equal value, and is easier of application. The skin over the mastoid process should be incised, as shown in Fig. 408, the circular knife being adjusted with a set screw so as to cut the desired depth. When the incision is made the exhaust pump should be applied, as shown in Fig. 409, and the air exhausted by turning the hand screw. An ounce of blood may thus be drawn from the inflamed tissues. The effect of this procedure is to overcome the venous stasis and edema, thus establishing a more rapid arterial flow of blood through the tissues. The nutrition of the tissues is raised and the migration of leukocytes increased.

Massage is of little value in promoting the reaction of inflammation in otitis media. In tubal catarrh, however, external mechanical vibratory massage under the angle of the jaw over the course of the Eustachian tube will often quickly relieve the edematous obstruction to this tube.

Vaccine and Leukocyte Extract Therapy.—1. Endotoxin is a toxic substance elaborated within the pathogenic bacteria at the time of their dissolution or death. Each type of germ produces its own peculiar endotoxin.

2. Each class of bacteria gives rise to antibodies peculiar to its class, and the antibodies counteract the endotoxins.

3. Pathogenic bacteria cause pathologic processes by (*a*) the irritation produced by the presence of the bacteria in the tissues and (*b*) by the presence of the endotoxin in the circulating blood.

The irritation produced by the presence of the pathogenic bacteria in the tissues, and of endotoxin in the blood is counteracted by (*a*) the antibodies given being by the parent pathogenic bacteria, (*b*) by the phagocytes, a type of blood corpuscle which actually destroys bacteria. Phagocytes are normally present in the blood, and, in the presence of infection, they are concentrated at the area of infection, and are greatly increased throughout the blood currents. Under ordinary conditions of infection, the increase in white blood corpuscles corresponds to the increase in the bacterial development. This is especially true in reference to the increase in the polymorphonuclear cells. If the bacterial increase remains long in excess of the white blood corpuscle increase, the corpuscle-producing organism becomes exhausted, and we have the clinical phenomenon of an increasing sepsis with a diminishing number of polymorphonuclear cells. (*c*) The destruction of the bacteria is also aided by the opsonins which are normally in the blood. They act by reducing the vitality of the pathogenic microörganisms, thereby rendering them easy preys to the phagocytes.

Recapitulation.—1. Under normal conditions of health, pathogenic bacteria are present in the body in more or less quantities and morbid processes are prevented by the (*a*) phagocytes, more particularly the polymorphonuclear cells), the (*b*) opsonins aiding the phagocytes by reducing the resistance of the bacteria, and (*c*) the antibodies given

being by the presence of the pathogenic bacteria counteracting the poisonous effects of the endotoxins liberated in the blood stream at the death or dissolution of the pathogenic microörganisms. The pathogenic bacteria are usually produced at more or less regular intervals, swarm, so to speak, so that there are periods of bacterial activity followed by periods of bacterial death. During the periods of bacterial activity the phagocytes and opsonins are especially active in the destruction of the bacteria, and the antibodies are especially active in counteracting the endotoxin liberated in the blood streams. Another feature not always considered in infective processes is, that the amount of blood is increased in the infected organ or tissues. This is of clinical importance because it means that an increased food supply is given to the histologic cells of the tissues during the term of stress placed upon them by the pathogenic bacteria and endotoxin. Should the pathogenic bacteria for any reason increase out of proportion to the phagocytes, antibodies, and blood (food) supply, we have the clinical phenomenon of increasing sepsis, and a diminishing polymorphonuclear count. A histologic cell soldiery, succumbing to an overpowering invading pathogenic bacteria host.

Autogenous Vaccines.—An autogenous vaccine is a preparation of dead bacteria held in suspension in normal salt solution. A culture is made from the pus of the infected area, the bacteria responsible for the pathologic process isolated, and submitted to a temperature of 60° C. for forty-five minutes. According to Dr. Nagel, of Boston, if the bacteria are overheated the vaccine loses some of its therapeutic qualities, a fact which may account for many of the failures attending its use.

Other factors also influence the success or failure of autogenous vaccines in the treatment of infections. Of these we may mention the drain upon the vital forces, attending the administration of a vaccine. If the patient is in a relatively good condition, *i. e.*, if his vital forces are not depleted, the use of vaccine may be followed by good results. If, on the contrary, his vital forces are at a low ebb, the vaccine will reduce them so much lower that he can not bridge over the depression gap, and he gravitates to a still lower plane of resistance, and the infection and sepsis become more and more pronounced. These facts may throw light upon the relative failure of vaccines in acute infections, and in cases in which there is great debility and profound sepsis, hence the added stress attending the injection of the vaccine is followed by evil results. If, on the contrary, the disease is pursuing a subacute or chronic course, and the vital forces are strong, the depression gap produced by the vaccine is bridged over and the antibodies are produced in quantities sufficient to neutralize the specific microörganisms and their endotoxin.

Vaccines are, therefore, of greater value in subacute and chronic infectious processes. In my experience they have been of special value following operations upon chronic nasal sinus, and aural disease. I have also had good results following operation upon pachymenin-

gitis. From one to two weeks should elapse after the operation before the vaccine is administered, as the stress attending the administration of the vaccine should not be added to that attending the operation. When the surgical shock has subsided, and the vital forces have been renewed the vaccine may be given with advantage.

Another factor of great clinical significance is that each type of pathogenic bacteria (streptococcus, staphylococcus, pneumococcus, etc.) produces antibodies peculiar to itself, and which are corrective only to its kind. Hence, the antibodies liberated in the blood by the staphylococcus vaccine would exert no corrective influence upon streptococci or pneumococci, whereas it would exert a favorable or restraining influence upon staphylococci. There are various strains of streptococci, staphylococci, etc., each possessing a virulency peculiar to itself, and arousing strains of antibodies, which exert a restraining influence only upon the peculiar strain of cocci giving it birth. It is, apparent, therefore, that the best results can only be obtained by using vaccines made from the germs obtained from the diseased area of each patient. In other words, only autogenous vaccines should be used.

The injections should be given in increasing doses every three or four days, and as soon as reaction symptoms such as a feeling of exhaustion, etc., are noticed, the injections should be stopped for a week or two, or until the vital forces have been reëstablished.

Stock Vaccines.—Stock vaccines are usually prepared by the various pharmaceutical houses from the various pathogenic microörganisms, as streptococci, staphylococci, pneumococci, etc. However well they may be prepared, they do not meet the therapeutic indications as well as the autogenous vaccines, as they (stock vaccines) do not give being to antibodies exactly suited to counteract the specific bacteria and endotoxin present. In addition to the foregoing objections the stock vaccines more or less deplete the vital forces without giving any beneficial results in return, as is done when an autogenous vaccine is administered.

The Leukocyte Extract of Hiss.—Conceive the soldier's combative outfit to consist of food, a rifle, cartridges, and a saber. During times of peace he makes but little use of his accoutrement. In war he not only uses his armament, but he needs and receives renewals of the same, as the exigencies of the occasion require. Conceive the leukocyte to be a soldier guarding the frontiers of the system against an invading host of pathogenic bacteria. In times of health it has potentialities within itself to maintain the balance of power. In times of stress it may or may not have enough potentialities within itself to restrain or destroy the invading host. It needs more food, rifles, and cartridges than the organism can supply. Hiss has attempted to meet this emergency by making an extract containing the essential properties of the leukocytes. That is, the extract contains the elements necessary to combat the bacteria and endotoxin, and its administration does not use any of the vital forces for their production, as in the adminis-

tering of autogenous or stock vaccines. The Hiss leukocyte extract seems to be food and artillery combined. The extract is readily diffusible through the system when injected into the subcutaneous tissue. It is especially indicated in (*a*) acute infectious processes in which the autogenous vaccines are contraindicated. It is also indicated (*b*) in those cases in which profound sepsis and depression of vital forces are present. In such cases there is an increasing sepsis, and a diminishing leukocyte count and diminishing polymorphonuclear percentage.

While the exact status of either the vaccine or leukocyte extract is not yet established, each has a field of usefulness of no mean proportions. Vaccine and leukocyte therapy are distinct steps in the right direction, even though we do not as yet know the ultimate goal to which they lead.

2. **Establishing Free Drainage.**—The second principle of treatment, the establishment of free drainage, is a very important part of the treatment of suppurative otitis media. If free drainage is maintained, pressure necrosis is not apt to occur; indeed, if present, it may disappear.

In the early stage of acute otitis media free drainage may be established by incising the membrana tympani, the Eustachian tube being, for the time, inadequate to carry away the excess of secretions. A free incision of the membrana tympani affords an accessory outlet for the secretions, and, in addition, it promotes the reaction of inflammation and relieves the pressure and attending necrosis.

If the obstruction is in the aditus ad antrum, incision of the membrana tympani may fail to establish free drainage, in which case it may be necessary to perform a mastoid operation. In some cases of chronic otorrhea, the obstruction is due to the heads of the malleus and incus, together with the ligamentous bands and adventitious cicatricial tissue resulting from the inflammatory process. In such cases the removal of the malleus and incus establish free drainage. Heath claims that the Eustachian tube is usually adequate to drain the tympanic cavity, even when diseased, but that it is inadequate to also drain the diseased mastoid antrum and cells. He therefore recommends that the secretions from the antrum and mastoid cells be diverted from the aditus ad antrum to the external auditory meatus, as described in the meatomastoid operation.

3. **Removal of Morbid Material.**—Whatever method of treatment is adopted, earnest effort should be made to remove all obstruction to the flow of secretions from the tympanic cavity. In infants and children the removal of the adenoids may accomplish the purpose by unblocking the Eustachian tubes. The removal of aural polypi or granulations may temporarily establish drainage. Incision of the membrana tympani, leeching, hot irrigations, dry heat, etc., may act favorably, but in many cases it will be necessary to resort to a mastoid operation. In simple cases the morbid material consists of the purulent secretions, which are successfully removed by drainage. In the more complicated cases, in which granulations and necrosed bone are present, an operation may be required to accomplish the result.

gitis. From one to two weeks should elapse after the operation before the vaccine is administered, as the stress attending the administration of the vaccine should not be added to that attending the operation. When the surgical shock has subsided, and the vital forces have been renewed the vaccine may be given with advantage.

Another factor of great clinical significance is that each type of pathogenic bacteria (streptococcus, staphylococcus, pneumococcus, etc.) produces antibodies peculiar to itself, and which are corrective only to its kind. Hence, the antibodies liberated in the blood by the staphylococcus vaccine would exert no corrective influence upon streptococci or pneumococci, whereas it would exert a favorable or restraining influence upon staphylococci. There are various strains of streptococci, staphylococci, etc., each possessing a virulency peculiar to itself, and arousing strains of antibodies, which exert a restraining influence only upon the peculiar strain of cocci giving it birth. It is, apparent, therefore, that the best results can only be obtained by using vaccines made from the germs obtained from the diseased area of each patient. In other words, only autogenous vaccines should be used.

The injections should be given in increasing doses every three or four days, and as soon as reaction symptoms such as a feeling of exhaustion, etc., are noticed, the injections should be stopped for a week or two, or until the vital forces have been reëstablished.

Stock Vaccines.—Stock vaccines are usually prepared by the various pharmaceutical houses from the various pathogenic microörganisms, as streptococci, staphylococci, pneumococci, etc. However well they may be prepared, they do not meet the therapeutic indications as well as the autogenous vaccines, as they (stock vaccines) do not give being to antibodies exactly suited to counteract the specific bacteria and endotoxin present. In addition to the foregoing objections the stock vaccines more or less deplete the vital forces without giving any beneficial results in return, as is done when an autogenous vaccine is administered.

The Leukocyte Extract of Hiss.—Conceive the soldier's combative outfit to consist of food, a rifle, cartridges, and a saber. During times of peace he makes but little use of his accoutrement. In war he not only uses his armament, but he needs and receives renewals of the same, as the exigencies of the occasion require. Conceive the leukocyte to be a soldier guarding the frontiers of the system against an invading host of pathogenic bacteria. In times of health it has potentialities within itself to maintain the balance of power. In times of stress it may or may not have enough potentialities within itself to restrain or destroy the invading host. It needs more food, rifles, and cartridges than the organism can supply. Hiss has attempted to meet this emergency by making an extract containing the essential properties of the leukocytes. That is, the extract contains the elements necessary to combat the bacteria and endotoxin, and its administration does not use any of the vital forces for their production, as in the adminis-

tering of autogenous or stock vaccines. The Hiss leukocyte extract seems to be food and artillery combined. The extract is readily diffusible through the system when injected into the subcutaneous tissue. It is especially indicated in (*a*) acute infectious processes in which the autogenous vaccines are contraindicated. It is also indicated (*b*) in those cases in which profound sepsis and depression of vital forces are present. In such cases there is an increasing sepsis, and a diminishing leukocyte count and diminishing polymorphonuclear percentage.

While the exact status of either the vaccine or leukocyte extract is not yet established, each has a field of usefulness of no mean proportions. Vaccine and leukocyte therapy are distinct steps in the right direction, even though we do not as yet know the ultimate goal to which they lead.

2. **Establishing Free Drainage.**—The second principle of treatment, the establishment of free drainage, is a very important part of the treatment of suppurative otitis media. If free drainage is maintained, pressure necrosis is not apt to occur; indeed, if present, it may disappear.

In the early stage of acute otitis media free drainage may be established by incising the membrana tympani, the Eustachian tube being, for the time, inadequate to carry away the excess of secretions. A free incision of the membrana tympani affords an accessory outlet for the secretions, and, in addition, it promotes the reaction of inflammation and relieves the pressure and attending necrosis.

If the obstruction is in the aditus ad antrum, incision of the membrana tympani may fail to establish free drainage, in which case it may be necessary to perform a mastoid operation. In some cases of chronic otorrhea, the obstruction is due to the heads of the malleus and incus, together with the ligamentous bands and adventitious cicatricial tissue resulting from the inflammatory process. In such cases the removal of the malleus and incus establish free drainage. Heath claims that the Eustachian tube is usually adequate to drain the tympanic cavity, even when diseased, but that it is inadequate to also drain the diseased mastoid antrum and cells. He therefore recommends that the secretions from the antrum and mastoid cells be diverted from the aditus ad antrum to the external auditory meatus, as described in the meatomastoid operation.

3. **Removal of Morbid Material.**—Whatever method of treatment is adopted, earnest effort should be made to remove all obstruction to the flow of secretions from the tympanic cavity. In infants and children the removal of the adenoids may accomplish the purpose by unblocking the Eustachian tubes. The removal of aural polypi or granulations may temporarily establish drainage. Incision of the membrana tympani, leeching, hot irrigations, dry heat, etc., may act favorably, but in many cases it will be necessary to resort to a mastoid operation. In simple cases the morbid material consists of the purulent secretions, which are successfully removed by drainage. In the more complicated cases, in which granulations and necrosed bone are present, an operation may be required to accomplish the result.

The Compound Tincture of Benzoin.—During the last ten years the author has used the compound tincture of benzoin in nearly every case of otorrhea treated, with great satisfaction. Its efficacy is in part due to the alcohol in its composition, but not altogether. It is more soothing than plain alcohol, more antiseptic, and more healing. It has proved to be of special value in those cases in which the fetid odor is present. This speedily disappears and the other features of the case also improve.

The compound tincture of benzoin should be dropped into the meatus, the head being inclined toward the opposite side. After such a treatment, if the discharge is not too profuse, the gauze may be allowed to remain in the ear and meatus for two or three days without developing fetor.

The middle ear should be previously cleansed as described above, but after a few applications of the remedy it may be abandoned, as the discharge often rapidly decreases until there is scarcely a drop on the gauze when removed.

It is not to be inferred from what has been said that the otorrhea will not return after the discontinuance of the benzoin, for it is very apt to do so in most cases, no matter what form of local treatment is pursued.

Irrigation.—*The use of the syringe* is not indicated, as it is in acute cases. It may be used to advantage, however, when there is a considerable accumulation of desiccated or tenacious mucus and pus in the *atrium* of the middle ear. The force of the stream loosens and propels the secretions from the middle ear, and thus prepares the tissues for treatment by other methods. Sterile water or normal salt solution should be used as hot as can be comfortably borne by the patient, one-half gallon being the correct amount for each treatment.

Boric Acid Powder Treatment.—This method of treatment is of less value in chronic than in acute inflammations of the middle ear. If the discharge is profuse, it may be used, although other measures afford more relief. If used, the powder should be blown, not poured, into the meatus.

Camphoroxol has recently been highly recommended by Hotz and others in obstinate otorrhea in which other methods of treatment had failed. Hotz reports several cases in which the remedy seemed to give speedy and satisfactory relief. He injects it into the middle ear through the Eustachian tube by means of the Weber-Liel catheter. Further observations along this line are needed, however, before the real value of this remedy can be estimated.

TREATMENT OF SUPPURATION INVOLVING THE ATRIUM AND ATTIC

Under this caption are included those cases in which the attic is chiefly involved, and in which this centre forms the chief source of annoyance and danger. The consideration of the best methods of treatment will therefore hinge upon the structure and arrangement of the parts composing the attic.

The point of chief interest is the lower boundary or floor of the attic, namely, the heads of the malleus and incus, and the ligaments and adventitious fibrous bands uniting them to the walls of the tympanum. Another point of clinical interest is Shrapnell's membrane, or the membrana flaccida. Perforation of this membrane affords one of the most obvious signs of attic suppuration. Irrigation of the attic may be accomplished with a curved cannula inserted through the perforation in Shrapnell's membrane, and local medication and explorations may be carried on through it.

The floor of the attic is of importance because, whereas in health it affords ample drainage for the secretions, it is oftentimes inadequate in chronic otorrhea. The inadequacy may be due to the excessive and heavy secretions, or to a more or less complete obstruction by the adventitious fibrous tissue of the spaces in the floor of the attic. Either condition will cause the secretions to remain in the attic, which may give rise to serious pathological changes, as necrosis and septicemia.

While the principles of treatment remain the same, the motive for treatment increases tenfold.

Free drainage is imperative and should be established by surgical interference. This may be facilitated by enlarging the perforation in Shrapnell's membrane by an incision extending anteriorly and posteriorly. The treatment should be addressed not alone to the attic, but to the atrium also. In other words, the treatment described in the preceding section should be used, and in addition thereto the following measures should be instituted:

The attic should be kept as free of secretions as possible by applying suction to the external auditory meatus with Siegle's otoscope or Delstanche's rarefacteur. The spaces of the attic should be irrigated through the perforation in Shrapnell's membrane, and a 2 to 4 per cent. solution of the nitrate of silver applied with delicate cotton-wound applicators. Should these measures fail, the radical mastoid operation may be performed, special care being taken to remove the external wall of the attic (roof of the meatus near the drumhead). By so doing the attic is fully exposed in the after-treatment.

CHAPTER XLVI

GENERAL PATHOLOGY OF OTITIS MEDIA AND MASTOIDITIS

Microörganisms are the exciting causes of middle-ear and intracranial pyogenic processes. Various organisms are active, either alone or in combination, no special one being characteristic of these processes.

The *free communication* between the epipharynx and the middle ear, and the perforated drumhead makes infection easy if the local conditions are favorable. Such a condition presents itself during the course of one of the exanthematous fevers when the vitality is lowered. Pathological changes occur in the mucosa, microörganisms continue to flourish, and the suppurative process is established. The *cilia* which normally partially cover the tympanic mucosa are destroyed, or their vitality is so impaired that their propelling function is no longer adequate to drive the secretions toward the Eustachian outlet. Accumulation, decomposition, and irritation follow. The mucosa breaks down, the periosteum covering the bone loses its vitality and disintegrates, and the bone depending upon it for nutrition becomes carious. The arteries in the mucosa become thrombosed, and the arterial supply is thus cut off from the membrane and periosteum as well as from the bone. Thus, the process of disintegration proceeds with greater or less activity, oftentimes without serious symptoms being present. The brain may be exposed by the caries of the tegmen tympani and antri, or through various other channels of communication.

It has been said that about two years of chronic suppuration usually precedes bone necrosis in the middle ear and its accessory cavities. This should be taken only as an approximate estimate, as the time varies with the type of the infection, and with the obstruction offered to the discharge of the morbid secretions. If the flow from the mastoid cells and antrum is free and unobstructed, the process may continue for years without bony necrosis. If, on the other hand, marked obstruction occurs early in the suppurative process, bone necrosis may take place before the two years have elapsed. This is often the case in acute primary mastoiditis.

It is of great importance in estimating the gravity of a suppurative process in the tympanum to determine definitely the predominant character of the microbic infection present. To this end cultures and microscopic examinations should be made. While but few physicians are prepared to make either the cultures or microscopic examinations, nearly all know where they can secure culture tubes and have such examinations made. The attending surgeon should smear the secretion from the ear on the contents of the culture tube and send it to a pathologist.

A few places where the above examinations may be made are:

(*a*) The Health Board of the physician's own city or some neighboring city.

(*b*) A neighboring physician.

(*c*) The nearest medical college, or the one from which the physician graduated.

(*d*) A pathological laboratory established for the purpose of accommodating those in need of such work.

The expense of such an examination is small, and the information obtained may be of inestimable value to the patient.

John Funke has reported the results of his observations as to the "Bacteriology of Otitis Media," and his work seems so conclusive and suggestive that an epitome of it is herewith given:

The following conclusions are based on a study of the literature of otitis media and his observations:

1. There is no specific organism of otitis media.

2. Acute otitis media is not invariably monomicrobic, as is commonly held. The pathogenic organism present may be of a single variety, but with it are frequently found a varying number of associated bacteria, which may or may not be influential in determining the outcome of the case.

3. The organisms commonly found, in the order of frequency, are: The pneumococcus, streptococcus, pyogenic staphylococci (albus and aureus), and the bacillus of Friedländer. He is strongly inclined toward the belief in a definite grippal otitis, primarily due to the influenza bacillus, which, however, becomes quickly associated with, or replaced by, other organisms.

4. The Bacillus diphtheriæ is more commonly present in otorrhea than is usually believed; it may be (*a*) the initial infecting agent, or (*b*) it may enter with the streptococcus or pneumococcus, or (*c*) it may be a secondary infection carried to the already infected ear by the fingers of the patient, or otherwise, as held by Babinski.

5. It is reasonable to believe, as Funke's observations show, that it persists for a varying period of time in the discharges, and may constitute a centre of danger, just as has been thoroughly established concerning its prolonged residence in the nasal cavities, pharynx, etc. Its frequent association with the Bacillus pseudodiphtheriæ has here the same significance as elsewhere, a factor not as yet fully determined.

6. The streptococcal infections are more grave and persist longer than pure pneumococcal infections, but both are usually supplanted by the staphylococcal sooner or later.

7. There is a true pneumobacillary otitis, usually acute and quickly converted into a mixed infection. The gravity of the process depends almost exclusively upon the character of the mixed or secondary infection.

8. Chronic suppurative otitis media is practically always a sequence of the acute.

9. Like the acute, it possesses no specific organisms.

10. Unlike the acute, it is almost always polymicrobic.

11. Its polymicrobic character may be evinced in any of three ways: (*a*) A mixed infection of pathogenic organisms; (*b*) one or more recognized pathogenic organisms (usually pyogenic staphylococci), with one or more bacteria usually regarded as saprophytes; (*c*) the usual pyogenic and pathogenic bacteria are absent, and the discharges are maintained through the activity of organisms that commonly lead a saprophytic existence.

12. While anaërobic organisms may play an important part in the pathogenesis of chronic suppurative otitis media, Funke's observations have not established their almost constant presence, as maintained by Rist.

13. The fetor met in the cases reported here can be explained by the presence of Bacillus pyogenes fœtidus without anaërobic organisms.

14. All clinical and collated bacteriological data indicate that otitic inflammations present different bacteriological findings in different localities. According to Moos, during the influenza epidemic of 1890 in Vienna, the otitic complications were due to the pneumococcus (Weichselbaum) and to the streptococcus in Strasburg, Griefswald, and Bonn (Ribbert).

15. Reports gathered from literature establish the existence of a primary tuberculous otitis, but all observers are of one mind as to the almost utter impossibility of the routine demonstration of the bacillus in discharge.

16. For the demonstration of the tubercle bacillus in suspected cases, Funke recommends an examination of tissue obtained by the curette.

Middle-ear Suppuration.—Microscopic Examination of One Hundred Cases, with Special Reference to the Presence of Tubercle Bacilli and Acid-fast Bacilli.—Wyatt Wingrave[1] gives the following analysis: Special care was taken in obtaining the discharge. Carbol-fuchsin was used in staining, with methylene blue as a counterstain:

	Cases.
Squamous and pus cells present together in	41
Pus alone	38
Squamous alone	21

Bacteria.

Staphylococci	41
Diplococci	20
Streptococci	7
Bacillus proteus vulgaris	14
Micrococcus tetragenus	4
Bacillus coli	3
Gonococci	33
Bacillus subtilis	2
Aspergillus niger	1
Leptothrix	1
Diphtheria (Klebs-Loeffler)	1
Yeast	1

[1] Jour. Laryngol., Rhin., and Otol., March, 1903.

Gradle and others, some years ago, called attention to the odor attending chronic otorrhea, claiming its presence or absence was the "most sensitive criterion of the efficacy of the treatment."

So long as the pus of the otorrhea smells fetid, the treatment employed has exerted no curative influence on the disease; and, conversely, "the first sign from any treatment of curative influence is its effect upon the odor of the discharges" (Gradle).

Macewen says: "The virulence of a discharge cannot be measured by its odor. Nearly odorless otorrhea may contain pathogenic micrococci, and some of the most serious intracranial inflammatory lesions ensue in the presence of odorless otitis media. It is well, therefore, in estimating the gravity of an otorrhea that pus from the middle ear should be stained and examined microscopically and by cultivations."

He states, further, that intracranial complications often arise in the course of fetid otorrhea, but that the pathogenic germ is not the one causing the odor, it usually being a non-pathogenic microörganism.

These views, while they seem to be diametrically opposed to each other, are really not so opposite as they appear. The first is fallacious, in that it leads to the inference that with the disappearance of the odor the patient's condition becomes safe; whereas, the second view tells us the absence of fetor is no criterion as to the non-virulence of the infection. Gradle's views lead, by inference, to the conclusion that absence of fetor is a guide to the mildness of the infection; whereas, Macewen says the absence of fetor gives no information whatever as to the virulence of the infection. He goes still farther and says some of the most virulent intracranial infections have occurred in connection with odorless otorrhea.

The author is inclined to agree with Macewen on this point, although he readily admits Gradle's major proposition, that the disappearance of the odor under the syringe, etc., usually heralds an improved drainage and ventilation. The improvement, however, is not due to the removal of the odor or the germs producing it, but to the removal of the saprophytic bacteria and the establishment of free drainage by the removal of the desiccated secretions. The disappearance of the odor is incidental, and signifies that other and more virulent organisms may have been removed also.

When the true nature of chronic otorrhea is explained to patients, many of them reply that they have had the discharge off and on for many years with no untoward result, and that they do not fear serious complications in the future. They express a belief that is often too prevalent among physicians, namely, that chronicity of otorrhea is a guarantee of its innocent nature. The process of disintegration has been going on, and may continue to do so as long as the otorrhea lasts. Fresh invasions of germs, or the encroachment upon a new area, or a lowered vitality of the patient, may give rise to sudden and alarming symptoms.

It may be said that *the more chronic the otorrhea the greater the danger of intracranial or other extension of the infective process.*

Acute primary otitis media suppurativa rarely extends to the brain or

meninges, as the process does not continue long enough to break down the mucous membrane, bone, and other tissues enveloping it.

In infants this protection is not so complete, as the various parts of the temporal bone are not yet united by ossification. The vascular and cartilaginous lines of union afford less resistance to the transmission of microörganisms to the cranial cavity; hence, intracranial involvement is more common in infants in the course of, or subsequent to, an acute primary suppurative otitis media.

In addition to the infection and consequent ulceration, thrombosis, and necrosis, there are other pathological conditions which are incidental to the suppurative process. *Adhesive bands* often form in the course of this disease, and the ossicles become bound to each other and to the tympanic walls. The handle of the malleus is retracted and may become adherent to the promontory.

The writer has a case under observation, aged forty years, with adhesion of the handle of the malleus to the promontory. When a young child she had suppuration of the middle ear, following scarlet fever. There have been occasional discharges since then. When she came under observation there was a *perforation of Shrapnell's membrane.* This healed under applications of the nitrate of silver. Examination with Siegle's otoscope shows the malleus to be adherent to the promontory. The anterior half of the drumhead is also adherent in places, while the posterior half is perfectly free. In other cases the adhesions have been severed with great improvement of the hearing.

Calcareous salts may be deposited in the drumhead and in the tympanic mucosa. The articulations of the ossicles may become ankylosed. The foot plate of the stapes is sometimes ankylosed from the deposit of lime salts in the fibrous ring which unites it to the margin of the oval window (fenestra of vestibule). This condition may be mistaken for hyperostosis of the bony capsule of the labyrinth (spongifying), though in the latter condition the drumhead and Eustachian tube are normal.

Granulations (aural polypi) are common, especially in old cases, in which the mucosa and periosteum are ulcerated and bone necrosis is present. They are the expression of Nature's effort to repair the tissues.

CHAPTER XLVII

INTRACRANIAL AND JUGULAR PYOGENIC DISEASES OF OTITIC ORIGIN

General Considerations.—Infection and inflammation of the middle ear, mastoid cells, and labyrinth are not, *per se*, usually a serious menace to life. The real danger is in the extension of the infection to the contents of the cranium or to the jugular vein, and thence to the important viscera, as the lungs, spleen, liver, heart, and kidneys, or a general dissemination throughout the body (general septicemia). Pneumonia, splenitis, hepatitis, endocarditis, and nephritis of otitic origin have been observed. The infection more often extends to the intracranial sinuses (veins) and to the jugular vein.

Of the intracranial pyogenic infections, thrombosis of the sigmoid portion of the lateral sinus, and the various types of meningitis, are most often observed. As the symptoms are not always characteristic of the type and field of invasion, the differential diagnosis is often difficult to make. There are, however, certain general characteristic phenomena, especially after the process is well advanced, which usually enable the aural surgeon to diagnosticate the condition present. When, for example, there is a chill, followed by a rapid and excessive rise of temperature, the evidence is conclusive that the system has been invaded by a numerous pyogenic host from some source. The most probable source of such an invasion is a disintegrating thrombus. The thrombus, being infected, finally undergoes disintegration, and the pathogenic bacteria are thrown in great numbers into the general circulation. As the sigmoid portion of the lateral sinus is in intimate anatomical relation to the mastoid process, the natural inference to be drawn from the chill and rapid rise of temperature is that sigmoid sinus thrombosis is present. If after the lapse of twenty-four hours a similar symptom complex recurs, the diagnosis may be more surely made. The thrombus may, however, be either the lateral, the superior, or inferior petrosal, longitudinal, or the cavernous sinus. These sinuses are, however, usually involved secondarily to the sigmoid sinus. The symptoms of cavernous thrombosis are so characteristic that, when involved, the diagnosis is easy.

Diffused purulent meningitis also presents certain characteristic symptoms which render the diagnosis comparatively easy. The temperature remains more or less constantly elevated, whereas in thrombosis there are distinct chills followed by a sudden and marked rise in the temperature, and a recession to nearly normal within from six to ten hours. Extradural abscess and brain abscess may be attended with a

moderate elevation of temperature or none at all, though there are frequent exceptions to this rule.

Lumbar Puncture.—Lumbar puncture for the diagnosis of meningitis should be made between the third and fourth lumbar vertebræ. A tapeline or cord passing around the body on a level with the crest of the ilia passes over the spine of the fourth lumbar vertebra; the spine just above is the third lumbar vertebra, and at a point midway between the two spines is the location for making the puncture. The needle should be introduced at a point a little to one side of the median line, and should be five or six inches long and 1 mm. in diameter. The spinal fluid will escape spontaneously when the point of the needle reaches the space in the cord. The increased tension may be estimated by the force and rapidity with which the fluid escapes. If normal, it drips rather freely from the needle, whereas in meningitis it escapes more rapidly. In some cases, however, the tension is not much elevated.

In infants and young children a simple acute otitis media may give rise to symptoms simulating cerebral complications, as headache, nausea, vomiting, and excessive elevation of temperature (Gradle). If meningitis is suspected, the diagnosis may be cleared by making a lumbar puncture and subjecting the removed spinal fluid to microscopic examination. If purulent meningitis is present, the fluid is turbid and loaded with pus cells and pathogenic bacteria, especially streptococci. If the fluid escapes under high pressure, and is clear and contains only a few leukocytes and no demonstrable bacteria, serous meningitis is present, and a mastoid operation should effect a cure without resorting to an exposure of the cranial contents other than at the atrium of infection, the tegmen tympani or antri. Lumbar puncture is negative in reference to the other intracranial infections.

These and other clinical phenomena usually enable the aural surgeon to differentiate the various extensions of the infection from the ear and mastoid cells to the cranial cavity. In the following presentation to the intracranial and jugular infections only the more typical clinical phenomena will be given.

MENINGITIS SEROSA

This disease is of otitic origin and is characterized by a serous infiltration of the pia mater and an increase in the cerebrospinal fluid in the subarachnoid space and in the ventricles of the brain.

Etiology.—(*a*) It is more often a complication of chronic otitis media and mastoiditis. (*b*) The channels of invasion may be through the tegmen tympani and antri, or through the labyrinth.

Symptoms.—Headache, dizziness, nystagmus, nausea, vomiting, restlessness, ataxia, torticollis, disturbances of vision, etc., may be present, though not all of them at one time. The symptoms are not different from those in the suppurative form of meningitis, and it is, therefore, difficult to make a diagnosis before operation. If there is

a spontaneous cessation of the meningeal symptoms, or if they cease after a mastoid operation, the disease is probably serous in character, the purulent forms rarely being thus favorably affected. Lumbar puncture is negative.

There are two chief differential points between diffuse suppurative meningitis (leptomeningitis) and serous meningitis, namely: (1) in suppurative meningitis lumbar puncture shows spinal fluid charged with pus and bacteria, whereas in serous meningitis the spinal fluid contains neither pus nor bacteria; (2) Practically all cases of diffuse suppurative meningitis end fatally, whereas practically all cases of serous meningitis recover. As the symptoms of both diseases are otherwise about the same, it is usually difficult to differentiate the two diseases. Lumbar puncture should, therefore, be performed in all cases of suspected meningitis.

S. J. Kopetsky has recently shown that (*a*) in meningitis and meningeal edema the cerebrospinal fluid is acid in varying degrees, whereas it is normally alkaline. When the cerebrospinal fluid is acid in reaction, it is strong presumptive evidence of meningeal suppurative infection or meningitis. (*b*) The dextrose, which is normally present in the cerebrospinal fluid, disappears when pus and bacteria appear, hence the absence of dextrose, the copper reducing agent, is strongly indicative of suppurative meningitis.

ACID REAGENT

℞—Five per cent. ferrichloride solution . . . . . 1 part.
One per cent. carbolic acid solution . . . . . 5 parts.

M.—Sig.—Add the cerebrospinal fluid drop by drop until the first yellowish tinge occurs. When the yellowish tinge occurs from the addition of from one to five drops of cerebrospinal fluid it is indicative of meningitis.

When the cerebrospinal fluid fails to reduce the copper in Fehling's solution it is strongly indicative of meningitis.

Treatment.—The surgical treatment of serous meningitis is essentially that of decompression. Unless the symptoms point to cerebellar involvement, the temporal lobe should be exposed by first removing the tegmen tympani et antri, and then removing the contiguous bone of the squamous plate of the temporal bone as shown in Fig. 518. In mild cases, and more especially in children, this will afford prompt relief. In severe cases it may be necessary to incise the dura in one or more places, or it may be necessary to puncture the lateral ventricle, or to perform repeated lumbar punctures. When the patient becomes delirious and his vital forces become greatly depressed, several incisions in the exposed dura should be promptly made, as simple decompression will not relieve the intracranial pressure. Relief should be expected within twenty-four hours.

Puncture of the lateral ventricle should be reserved for very severe cases, though its performance need not be attended by serious consequences. It may be done with either a hollow needle, a long slender scalpel, or with a special dull-pointed brain knife.

Seromeningo-encephalitis as described by Köner, is serous meningitis plus an edema of the underlying cortex of the brain. The treatment consists of multiple incisions through the dura, as in simple serous meningitis, and in extending the incisions about one-half inch into the substance of the brain.

Sterilized gauze should be loosely applied to the exposed dura after either of the above procedures, but should not be inserted beneath the dura.

EXTRADURAL ABSCESS; PACHYMENINGITIS EXTERNA CIRCUMSCRIPTA

Definition.—An extradural abscess is a localized or circumscribed pachymeningitis. The thin plate of bone between the attic and the dura, or between the antrum and the dura, undergoes carious and necrotic degeneration, and the dura over this area becomes inflamed, throws out a plastic exudate, and is firmly attached to the bone it covers. After a time the bone is destroyed and the purulent secretion burrows between the dura and the bone, but is prevented from extending over a large area by the plastic exudate. It is generally located in the middle fossa.

Etiology.—The abscess usually occurs in chronic otorrhea with acute exacerbations of mastoiditis. It also occurs in cholesteatoma with suppuration. The cholesteatomatous mass in the attic or antrum causes pressure necrosis of the tegmen tympani and antri, and thus exposes the dura of the middle fossa to infection. Acute suppurative otitis media, especially of influenzal origin, may also cause it, as the bacillus of influenza is very destructive to bone tissue. An infected embolus or a thrombus from one of the veins or its tributaries may cause an extradural abscess without bone necrosis.

Symptoms.—The signs of this condition are not well marked, a severe headache with a slight rise in temperature being the most reliable. The headache is continuous and is referred to the affected side. When, however, there is a sudden profuse discharge of pus from the ear, the headache and the temperature are relieved or disappear altogether. If the membrana tympani is observed by reflected light, and the pus pulsates in the perforation, it may be inferred that it has its origin in the middle fossa of the skull. That is, the pus comes from a cavity surrounded or partly surrounded by a resilient tissue; the dura is such a tissue, hence the inference. If the pus comes from a bony cavity, no such pulsation is present, unless an artery is exposed by the necrotic process. The internal carotid artery passes close to the anterior portion of the cochlea, and if there is a labyrinthine suppuration, and the artery is exposed, there may be a pulsation of the escaping pus.

If during a mastoid operation there is a profuse discharge of pus which pulsates synchronously with the heart beat, there is in all probability an

extradural abscess, which may be evacuated and cured by removing the tegmen tympani and tegmen antri.

Localizing motor symptoms are absent, as the motor tract of the brain is not involved (Fig. 424).

The abscess is not always located in the middle fossa. Necrosis of the cells posterior to the labyrinth may occur, and thus communicate with the cerebellar fossa back of the pyramid of the temporal bone. Hence, vomiting and vertigo may be the prominent symptoms. The headache in these cases is referred to the region of the occiput on the affected side. The temperature is about the same as in extradural abscess of the middle fossa. As the disease progresses, mental dulness and coma develop from the increased intracranial pressure, due to the effusion into the ventricles.

In a case recently operated on by the author, the patient rapidly developed coma during the course of an otitis media and an acute exacerbation of mastoiditis on the right side. The surgeon who was in attendance had placed the patient in a hospital for observation, and had recommended an operation for mastoiditis. This was refused. During the absence of the surgeon from the city the coma developed. When seen by the author, the patient was comatose. The nurse stated that he had been complaining of pain in the back of the head, but did not know to which side he referred it; a radical mastoid operation was performed upon the right side, and, as a cerebellar abscess was suspected, the operation was extended in the usual way to this region, but without locating the abscess. At the postmortem an extradural abscess containing about 2 drams of thin yellow pus was found on the opposite side on the posterior inferior aspect of the cerebellum. The left ear was not affected.

Prognosis.—If the abscess becomes latent, and acute exacerbations of the otitic and mastoid inflammation do not occur, the patient's life may not be placed in jeopardy for a long time. If, on the contrary, the abscess occurs during an acute exacerbation, or following an acute attack of influenza, it may break its bounds and penetrate the substance of the brain and lead to a fatal issue.

If the abscess is recognized, located, and successfully operated on, the patient usually recovers. Spontaneous evacuation into the ear or through the outer table of the skull may result in recovery. Knapp reports two such cases which evacuated near the occipital protuberance, both of which recovered. Dench reports 25 cases of extradural abscess, 23 of which recovered and 2 died. Of 12 cases occurring in the author's practice, 10 recovered and 2 died.

Treatment.—The treatment is surgical; alcoholic stimulants may be given if sepsis is present.

The surgical treatment of an extradural abscess consists in removing the plate of bone underneath which the abscess rests and evacuating its contents. If the abscess is in the middle fossa, it can be generally reached through the tegmen tympani and antri, which have already been exposed by the radical mastoid operation. A carious opening usually exists, and this should be enlarged until the plastic adhesion to the bone

is reached. This should not be disturbed, as to do so opens the avenues of infection to the healthy dura beyond it. A curved probe introduced through the fistulous opening in the roof of the attic or antrum will enable the operator to define the outlines of the abscess cavity, and he can thereby judge the area of bone to be removed. It will often be necessary to make an opening through the squamous portion of the temporal bone, especially in those cases due to a thrombus or an embolus, in which case the skull on the affected side should be trephined. If there is a point of tenderness, this may be utilized as a tentative means of locating the abscess. If after making the opening healthy dura is found, introduce a probe between the dura and the bone and pass it in various directions in an endeavor to locate the abscess. If the abscess is chronic and walled off, do not rupture the plastic barrier if it is possible to reach it by making an opening directly over it, as to do so may set up a diffused meningitis. If, however, the abscess is not directly accessible through an external opening, the plastic wall may be broken down and the pus evacuated through the opening already made by lifting the dura with a heavy probe or spatula and allowing it to escape. The dura should then be irrigated with warm bichloride solution, 1 to 5000.

If the abscess is between the posterior wall of the pyramid and the dura, it may be reached through the mastoid wound by extending the bony wound from the posterior wall of the antrum backward and to the inner aspect of the sigmoid groove of the lateral sinus. If the sinus is large and well forward, this route is not available. The skull should then be trephined as shown in Fig. 518

INTRADURAL ABSCESS; PACHYMENINGITIS INTERIOR CIRCUMSCRIPTA

This condition is quite similar to extradural abscess, except that the dura is perforated and the plastic exudate exists between the dura and the arachnoid, thus walling off the purulent accumulation from the brain. The symptoms are the same as in extradural abscess. The prognosis is more grave, as the brain is in great danger of infection. The treatment is the same, though the probing must be more carefully prosecuted, as the arachnoid is more delicate than the dura.

LEPTOMENINGITIS DIFFUSA PURULENTA OF OTITIC ORIGIN

Leptomeningitis may arise in the course of an otitis media or mastoiditis from a perforation through the tegmen tympani and antri, the carotid canal, the labyrinth [(*a*) meatus auditorius internus; (*b*) aquæductus vestibuli, (*c*) aquæductus cochleæ], and through the sheaths of the anastomotic bloodvessel in influenza. Ethmoiditis and sphenoiditis may also give rise to it. Abscess of the brain in its later stages is often complicated by diffuse purulent meningitis.

Symptoms.—When the infection reaches the posterior fossa *via* the labyrinth and internal auditory meatus, the onset of the disease is sudden and is accompanied by spontaneous nystagmus directed toward the affected ear. Previous to this (premeningeal stage) the nystagmus, if happily it was observed, was toward the sound ear. In all middle-ear inflammations attended by giddiness, staggering gait, nystagmus, facial paralysis, and great tenderness over the mastoid emissary vein, the closest watch should be kept of the patient for spontaneous nystagmus, and if found directed toward the sound ear, labyrinth involvement only may be diagnosticated. If it reverses its direction while under observation, it marks the onset of meningitis. If the reversal is not observed, but the nystagmus is toward the diseased ear, a tentative diagnosis of meningitis in the posterior fossa may be made.

Headache, at first remittent and later constant, is characteristic of this disease. The temperature is elevated and face flushed. The pulse ranges from 120 to 140, and the respirations are rapid, the latter assuming the Cheyne-Stokes type as a fatal issue is approached. Persistent vomiting of mucus and bile is present. Mental excitement, such as irritability, delirium, and extreme restlessness are marked symptoms; as the disease progresses, somnolence and loss of memory develop. Rigors are present, but not so marked as in sinus thrombosis.

The pupil on the side affected is often contracted, and occasionally the one on the opposite side. Choked disk, papillitis, and optic neuritis, while not always present, are often present.

The muscles of the face and extremities become drawn or contracted, but these phenomena finally centre in the muscles of the neck, and the head is retracted. The muscles of the abdomen are drawn in and the abdomen flat. The motor oculi, trochlear, and abducens nerves become paralyzed.

Spinal involvement is shown by Westphal's symptoms, viz., increased tendon reflexes and paresthesia and hyperesthesia of the extremities.

By Quincke's *lumbar puncture*, the increased pressure increased coagulability and the presence of bacteria may be determined. The virulence of the bacteria may be tested by inoculating a guinea-pig with them. Coma occurs a few hours before death. In the early stage lumbar puncture may furnish the only reliable data for a positive diagnosis. The presence of pus and bacteria without other diagnostic symptoms would warrant a diagnosis of meningitis. An early Haynes operation upon the cisterna magnum might effect a cure. (See Lumbar Puncture.)

Prognosis.—Death occurs in nearly every case. Operative interference is not warranted, unless, indeed, Haynes' operation upon the cisterna magnum is resorted to.

Treatment.—The treatment of purulent meningitis is essentially surgical, though the results of such treatment have been almost uniformly unsuccessful. Several cases thus treated have been reported as cured, but in all probability they were serous in character. Nevertheless, surgical treatment appears to be the only rational method at our command. The chief difficulty is anatomical, namely, the purulent

exudate penetrates the subdural and subarachnoid spaces, where it becomes more or less organized, and as a consequence does not drain away, even when the whole area is exposed by the removal of the bone and numerous incisions are made in the dura. The same difficulty attends Haynes' operation upon the cisterna magnum. (See Haynes' Operation.) As I have already said, all operative measures have thus far, with a few possible exceptions, proved futile, insofar as a cure is concerned. Many cases are, however, relieved of the delirium and coma, and spend their last hours in comparative comfort, with a reasonably clear intellect, a point of considerable sentimental and medicolegal importance.

The treatment consists in exposing the dura over the affected area, and removing the tegmen or the squamous plate of the temporal bone as in serous meningitis. (See Treatment of Serous Meningitis.) If the cerebellum is involved a plate of bone should be removed as shown in Fig. 518. When the dura has been thus uncovered several incisions should be made in it. Loose dressings of gauze should be applied daily under the strictest antiseptic precautions.

Haynes' Operation.—This operation is based upon the theory that in meningitis there is a constant high intracranial pressure, and that the natural drainage pathways are blocked. He proposes by opening the cisterna magnum to relieve the high pressure, and at the same time unblock the arachnoid pathways for drainage. His operation undoubtedly does both of these things, but unfortunately the purulent exudate is coagulated and entangled in the subdural and subarachnoid spaces, and is physically unfit to drain away. As a consequence the operation has thus far proved very disappointing, except in cases that were probably serous in character. Dr. Ewing W. Day has reported nine cases, all of which ended in death; Dr. J. C. Beck, seven cases, all but one of which died; Dr. Emerson, one case, which recovered, and which he now regards as serous meningitis. Haynes, Kopetsky, Phillips, and others have reported cases with similar results. This operation seems to offer little or no advantage over the exposure of the dura in the temporal and cerebellar areas. It does, however, afford a better chance for drainage, and is therefore to be preferred to the other operations. Subsequent experience and improved technique, which can only be gained by continuing to do the operation, may show better results.

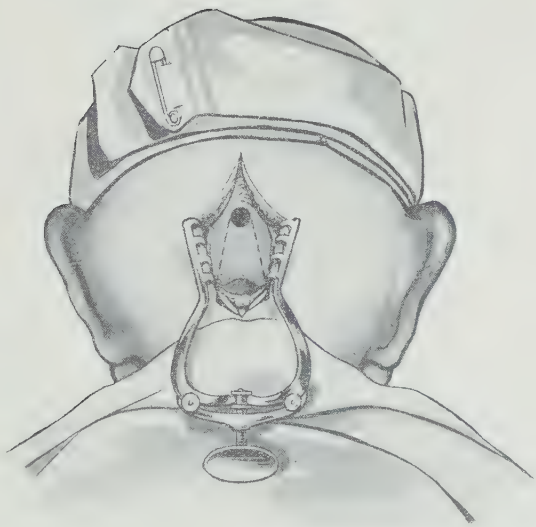
Technique.—1. The preparation of the patient consists in shaving the entire head and sterilizing it, the neck, and contiguous areas of skin. The patient is then placed face downward on the operating table, with the head flexed upon the sternum over sand bags or the end of the table.

2. The anesthetic preferred by Haynes is ether administered through a nasal tube, by the warm spray method, beneath a sterile sheet covering the patient. The variations of the pulse and respirations are constantly watched during the entire operation by an assistant especially detailed for this purpose. A sphygmomanometer is applied, and its fluctuations carefully noted by the assistant.

Fig. 420

The incision in Haynes' operation.

Fig. 421

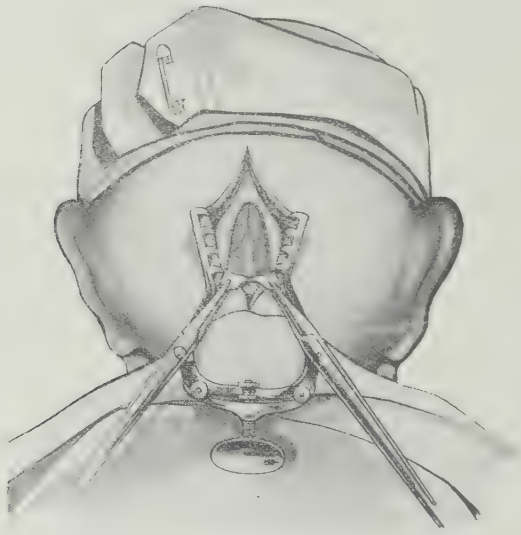


The skin and periosteum elevated, and an opening trephined through the bone at the upper angle of the wound.

3. *The skin incision* extends from the occipital protuberance to the spinous process of the axis (Fig. 420), and is rapidly carried down to the occipital bone and posterior arch of the atlas. Bleeding arteries and veins are tied as they are encountered. The periosteum and overlying muscles are elevated on either side of the incision, thus making, when retracted, a field about one and one-half to two inches long, and one inch wide (Fig. 421), the widest portion being at the posterior margin of the foramen magnum.

4. *Opening the skull* is accomplished with a De Vilbiss trephine three-eighths of an inch in diameter (Fig. 421). The trephine should be located about one inch behind the posterior margin of the foramen magnum, and the button of bone removed. This opening should be enlarged forward to the foramen magnum with a De Vilbiss bone forceps, and should be made wider and wider as it approaches the foramen, thus forming a V-shaped opening as shown in Figs. 421 and 422.

FIG. 422



Incision through the dura.

5. *The incision of the dura* should only be done after locating the occipital sinus, or sinuses, as the case may be. In some cases it is single, while in others it is double. The incision should include only the dura, and should be made as long as possible without injuring the occipital sinus (Fig. 422).

6. *The arachnoid* should now be punctured with the point of the bistoury, the fluid escaping under high pressure, often rises several inches into the air. Some of the fluid should be caught in a test-tube for examination. The arachnoid incision should now be made

coextensive with the dural incision (Fig. 423). A sphygmomanometer should be applied throughout the operation, and when the fluid escapes the change in blood pressure should be noted.

Fig. 423

The arachnoid incised and the cisterna magnum opened.

The dressing advised by Haynes is made of accordion pleated rubber tissue, which is inserted one-quarter of an inch into the cisterna magnum (Fig. 423). One or two sutures are used to bring the soft parts together. The outer dressings are changed daily, or as often as they become saturated.

ABSCESS OF THE BRAIN[1]

General Considerations.—Brain abscess following middle-ear or mastoid disease, may present a typical train of symptoms of an infective process combined with a tumor formation within the cranial cavity in which definite localizing symptoms are present, consequently rendering the diagnosis an easy and simple matter. In other cases of chronic encapsulated abscess there may be an entire absence of any localizing symptoms, and the general symptoms may be so indefinite that the surgeon may not suspect the real nature of the trouble, hence making a definite diagnosis impossible; however, taking into consideration that there is a history of middle-ear disease, combined with some general symptoms of toxemia, such as prolonged exhaustion

[1] Revised and largely rewritten by Dr. Howard Charles Ballenger.

or emaciation with no explainable cause, the surgeon may suspect an intracranial complication, although he may be unable to localize it.

Etiology.—Abscess within the cranial cavity may be due to a number of conditions, of these only one will be considered in this chapter, namely, those abscesses secondary to middle-ear or mastoid disease. Of all etiological factors concerned in producing brain abscess, disease of the tympanum antrum and labyrinth is the most common.

The extension of the middle-ear diseases to the cranial cavity depends upon the vulnerability of the natural barriers separating the middle ear from the cranial cavity, namely, the bone and its lining mucous membrane of the middle ear and antrum. Hence it is in those long-continued middle-ear suppurations that the vitality and protective ability of these natural barriers are more apt to be impaired or destroyed and an easy avenue of entrance to the cranial cavity established. It has been shown by the statistics of Neumann, Okada, Grunert, and others that approximately 85 per cent. of brain abscesses come from the chronic form of middle-ear suppuration.

The most susceptible age of attack usually falls within the limit of the tenth to thirtieth years of life.

Men are more frequently affected than women. In Neumann's 188 collected cases, 127 were men and 61 were women. Of Koerner's 204 collected cases, 136 were men and 68 were women.

By far the greatest number of otitic brain abscesses are found in the temporosphenoidal lobe, the cerebellum being second in the frequency of attack, the pons and crura cerebri being a comparatively rare location for abscess. According to the statistics (Kerrison) as given below we find out of a total of 1400 cases in which the abscess in the rarer situations, such as pons and crura cerebri are left out, that 930 or, approximately, 70 per cent. involve the cerebrum, and that 470 or, approximately, 30 per cent. involve the cerebellum.

	Total number cases.	Temporal lobe.	Cerebellum.
Barr	68	55 (81 per cent.)	13 (19 per cent.)
Koerner	119	79 (67 per cent.)	40 (32 per cent.)
Hermann	581	395 (68 per cent.)	186 (32 per cent.)
H. Todd	100	65 (65 per cent.)	35 (35 per cent.)
Neumann	532	336 (63 per cent.)	196 (37 per cent.)

Various investigators have shown that a streptococcic infection of the middle ear is more apt to result in an intracranial infection than is any other one microörganism.

Ruttin has called attention to Streptococcus mucosa capsulatus as an exceedingly virulent organism which if left unchecked will go on in many cases to intracranial complications.

Pathology.—The spread of infection from the diseased ear to the brain may occur in various ways; probably the most frequent avenue of entrance is by way of the tegmen tympani, which usually results in an abscess located in the temporosphenoidal lobe. A long-continued suppurative process within the middle ear may result in an

erosion and perforation of the tegmen tympani and a consequent localized inflammation of the dura mater. According to Macewen the visceral surface of the dura has, at a point corresponding to the osseous erosion, a raised projection of granulation cells and plastic effusion, generally somewhat conical in shape, with its obtuse extremity pointing upward, and forming an indentation on the surface of the brain, the pia mater usually being adherent to it. This conical mass of granulation tissue and inflammatory exudate later forming a direct pathway from the middle ear to the brain substance.

As a result of the extension of the infection to the pia mater and brain, the veins and lymphatics of the former may become directly affected and in some cases result in thrombosis of the terminal cerebral vessels; the infected thrombus later giving rise to a cerebral abscess, located at some distance from the point of entrance into the cranial cavity.

The cerebellar fossa is most prone to attack from a suppurative process in the mastoid by way of the sigmoid groove, the majority of these cerebellar abscesses remaining in contact with the sigmoid sinus.

Frequently a thrombus forms in the sigmoid sinus corresponding to the site of the bony erosion, due to an associated inflammatory condition of the inner surface of the sinus wall.

Infection may gain entrance into the labyrinth through necrosis of the external semicircular canal, through the fenestra ovalis, through the promontory or the fenestra rotunda; and from the labyrinth the infection may pass to the meninges by way of the meatus auditorius internus, aquæductus vestibuli, or aquæductus cochleæ. The increasing attention which is now being paid to suppurative labyrinthitis and its recognition may result in establishing a much greater frequency than formerly supposed, of the role of the labyrinth in producing abscess of the brain, especially of the cerebellum. Neumann says that in two-thirds of all abscesses of the cerebellum the labyrinth is involved.

A brain abscess may vary in size from that of a pea to one capable of displacing an entire lobe of the brain.

An abscess usually has an encapsulating membrane due to the more or less inflammatory process of its development, although an entire absence of any fibrous retaining wall has been noted (Neumann).

Symptoms.—Macewen divides the symptoms and the course of the disease into three stages, and this classification will, in a general way, be followed.

First or Initial Stage.—In this stage the predominant features are those of *toxemia* rather than of a tumor formation, and when seen by the surgeon, an abscess formation may not be anticipated. The patient usually gives the history of a long-continued, purulent discharge from the ear. He may have become ill from the abscess rather suddenly. *Pain* is nearly always complained of. It is usually confined to the region of the abscess, although it may be referred to the frontal or occipital regions irrespective of the location of the abscess. The pain is usually of an excruciating character or may be of the neuralgic type.

Vomiting frequently occurs during this stage and bears no relation to the taking of food. It may be unaccompanied by nausea.

A chill is one of the fairly constant early symptoms. It may vary in intensity from a slight sense of coldness to a most violent shaking, and may endure from a few moments to an hour or so.

The temperature is somewhat elevated and the *pulse faster* than normal.

The *otorrhea* may cease or become less in quantity.

In a few hours or several days the symptoms of the acute stage usually abate and a period of *quiescence* or the *latent* stage as some authors classify it, may ensue. The patient may apparently be in normal health and be able to pursue his usual vocation. This latent period may last only a few days or may extend over a period of weeks or months, but sooner or later signs of increasing intracranial pressure will become manifest.

Second Stage or Stage of Increasing Intracranial Pressure.—As the abscess takes form and gradually increases in size, the pressure within the cranium is more and more increased.

The patient may complain of *a dull, persistent headache* in the region of the affected lobe, or he may have a slight pain referred to the frontal or occipital region. Percussion of the affected side may elicit some tenderness, but the pain and headache may not be present, the patient appearing to be in a fairly comfortable condition.

Mental Lethargy.—As the abscess increases in size, and consequently increases intracranial pressure, there is a retardation of cerebration in which the patient will consume several times the usual length of time in any simple mental process, such as answering questions and the like. His attention will not be sustained for any length of time. He appears drowsy and easily drops into a somnolent condition, although unable to get any refreshing sleep.

Loss of Motor Will Power.—The patient may give the impression of a profound weakness during this stage which is not based upon a lack of muscular strength, but is rather due to a lack of motor will power.

Temperature during the second stage in uncomplicated cases is normal or slightly subnormal, in marked contrast to other intracranial complications.

The *pulse* is slow, usually fifty to sixty per minute, and is of good volume.

Respirations are slow and usually regular.

Vomiting usually does not occur during the second stage, although it may be present, and when associated with a persistent headache is very suggestive of intracranial disease (Macewen). Vomiting associated with a persistent headache is more frequently a later symptom of tumor and meningitis, than of abscess, in which latter condition these symptoms more often occur early in the initial stage of the disease. The remittent type of vomiting is more apt to result from cerebellar than from cerebral abscess.

Emaciation of a profound degree frequently occurs in brain abscess

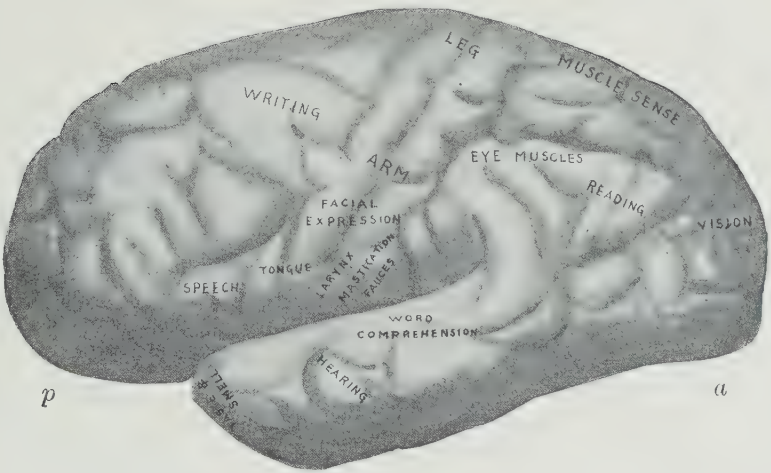
during the latter part of the second stage. The presence of an unexplained loss of flesh and strength combined with an entire absence of any fever, especially when bradycardia is present, is very suggestive of a cerebral abscess.

Constipation is the rule and may be very obstinate

The *urine* frequently is retained, and occasionally shows albumin. Sugar is absent unless the location of the abscess is in the pons or the medulla oblongata, in which case sugar is frequently present.

Localizing Symptoms.—*Convulsions* seldom occur in abscess of the brain, and when they are present are probably due to a leakage of the abscess with a consequent localized meningeal irritation. When a localized spasticity of some muscle or group of muscles is present, it affords valuable aid to diagnosing the location of the abscess; however, it should always be taken into consideration that the irritation of the motor centres involved may lie at some distance from the abscess.

FIG. 424



The cortical centres of the cerebrum, to be used in localizing lesions within the skull.

Paralysis when present is of much aid in diagnosing the location of the abscess, as it usually depends upon direct pressure of the motor centres or pathways involved, or upon the more or less destructive process produced in the immediate inflammatory zone surrounding the abscess. *Hemiplegia* on the side opposite to the lesion has been noted in large temporosphenoidal abscess. Frequently the abscess is located near the arm or leg centre, with the resulting paralysis of one or both of those members (Fig. 424). *Contralateral facial paralysis* may occur in certain temporosphenoidal abscesses, although it is possible for the paralysis to be homolateral from direct involvement of the peripheral nerve itself. Paralysis of the third nerve and adductor muscles of the eyes on the same side may occur, although this paralysis is rarely complete. The *pupil* may be either myotic or mydriatic,

that is, contracted or dilated; usually a small abscess with some cerebral irritation, producing a more or less contracted and sluggish pupil, and a large abscess with the consequent increased pressure resulting in mydriasis.

Aphasia may be looked for in certain cases in which the cortical speech centre of the left temporosphenoidal lobe is involved. The aphasia may be either sensory or motor. The most common speech defect is that form of sensory aphasia known as word deafness, in which the patient recognizes the form of the object and its uses, but is unable to recall its name.

Optic neuritis, papillitis, and choked disk are sometimes found in the latter part of the second stage of large abscess of a more or less chronic nature, especially those abscesses involving the cerebellum. These eye-ground changes are not so frequent in brain abscess as in certain other intracranial affections such as meningitis and slow-growing tumor.

Terminal Stage.—If the disease is allowed to pursue its own course it ends, as a rule, in death. This may occur in one of at least two ways.

(*a*) By a gradually deepening stupor and coma followed by death.

(*b*) By rupture of the abscess into the ventricles and subarachnoid spaces producing a more or less diffuse suppurative leptomeningitis, from which death results within one or two days.

Diagnosis.—Bacon emphasizes the significance of a firm, dense, mastoid process in the cases operated, in which such symptoms as high fever, rapid pulse, etc., do not abate after the operation. He thinks it points to cerebral complications, and should lead the operator to explore the cranial cavity without further delay. If the pus and debris are removed and drainage is established, the symptoms should at once become better, and they should remain so. If, on the other hand, only the outer pus pocket (mastoid antrum) is evacuated, while the inner pus pocket (brain abscess) remains closed, the septic symptoms will continue. The needlessness of delay in operating, or doing secondary operations upon the cranial cavity, when the septic symptoms continue without abatement cannot be too strongly impressed. The dangers attending the exploration of the cranial cavity are small compared with those of delay.

When, after a mastoid operation, the fever and pain continue and the examination of the fundi of the eyes is negative, the surgeon should not be misled by the negative findings, as many cases are reported in which the subsequent history showed brain involvement to have been present.

J. F. McKernon writes that when the occipital pain is not relieved by the primary mastoid operation, the aural surgeon should go deeper and explore the cerebellar area, in order, if possible, to determine the cause of the pain. He recommends a grooved director for exploring the brain substance in place of an aspirating needle, as it allows the thick pus to escape, whereas an aspirating needle does not.

McKernon formulates the following indications for exploring the cranial cavity when an otitic abscess is suspected:

1. That a chronic otorrhea is or has been present.
2. Persistent headaches, general or localized.
3. Restlessness and irritability of temper.
4. Tenderness of the affected side on percussion.
5. Vomiting, and vertigo.
6. An almost persistently low temperature.
7. A slow pulse, later stupor. Optic neuritis may or may not be present, when present it may aid materially in arriving at a diagnosis as may also aphasia and motor disturbances.

He believes head pain (2) is the most significant symptom.

"In the great majority of cases, other than traumatic or pyemic, the patient has had a chronic purulent discharge from the middle ear, often dating from an attack of one of the exanthematous fevers of childhood, or he has had a chronic ulceration about the nose or mouth." (Macewen.)

The otorrhea may have given little trouble, and its long continuance without apparent harmful result may have lulled the initial fear, until the ear disease is regarded as of no importance.

A person thus affected may suddenly become seriously ill after unusual exposure or injury to the head, or even without any known cause. Persistent headache develops without any increase in the pus discharge. Other symptoms follow, and the patient applies to his physician for relief.

There may be a fistula in the tegmen tympani, which has existed for years without infection of the meninges. The granulations fill the opening and effectually guard the intracranial contents from infection. Such a favorable result is not always to be expected. In removing the granulations from the attic through the external meatus, great care should be exercised lest a perforation in the tegmen be thereby opened and septic infection transmitted to the meninges.

Differential Diagnosis.—In acute cases one must distinguish between abscess, encephalitis, meningitis, and septic sinus thrombosis. The chronic cases may be mistaken for tumors, since the pressure symptoms are similar in both. The evidence of brain pressure, stupor, slow pulse, and subnormal temperature are more frequently associated with an intracranial abscess than with meningitis or encephalitis. Cervical rigidity and Kernig's sign, are more characteristic of meningitis than abscess. In meningitis the cerebrospinal fluid obtained by a lumbar puncture even if bacteria are absent, shows an increased number of leukocytes (Starr). This is not true of abscess unless it be complicated by meningitis. In septic thrombosis of the sigmoid sinus the high temperature with its marked remission, rapid, weak pulse, recurrent rigors, followed by profuse sweating, and the pain in the submastoid region and down the course of the affected jugular should enable the surgeon to make a differential diagnosis.

Sometimes it is necessary to differentiate between abscess and

tumor of the brain. The absence of a history of a suppurative process in the temporal bone, the slow progress of the symptoms, the progressing involvement of the cranial nerves, the absence of rigors, and the intense degree of optic neuritis should all point to tumor.

Prognosis.—The natural termination is death. Surgical interference often arrests this if done in the first or second stage. Koerner reported 92 cases of brain abscess operated upon with 51 recoveries. The prognosis depends somewhat upon the bacteria which may be present. If the examination of the pus from the abscess reveals a streptococcic or anërobic bacteria, the prognosis is bad, if diplococcic bacteria are present the prognosis is better.

The prognosis also varies according to the stage in which the operation is performed. If seen and operated in the first stage the death rate should be small, perhaps less than 10 per cent., if in the early part of the second stage it should not exceed 50 per cent. According to Macewen, "an uncomplicated cerebral abscess, whose position is clearly localized, if surgical measures are adopted for its relief at a sufficiently early period, is one of the most hopeful of all cerebral affections."

Treatment (see Surgery of the Temporal Bone).—The most important of all therapeutic measures is prophylaxis, hence, the early radical treatment of extracranial suppurative processes is the best preventative for these intracranial complications.

However, the general condition of the patient should receive careful attention such as prevention of constipation and coughing, as the straining incident to these two conditions may produce a prolapse of the brain.

CEREBELLAR ABSCESS

Cerebellar abscess in the early stage, or when the abscess is small, gives rise to few localizing symptoms, and, hence, is very difficult of diagnosis. When the abscess is large, or when there is an associated meningitis of the cerebellar fossa, there is a more characteristic train of symptoms. Abscess in the region of Bechterew's nucleus and of the third and sixth oculomotor centres, may give rise to characteristic symptoms such as vomiting, giddiness, nystagmus, ataxia, and oculomotor paralysis.

Symptoms.—General Symptoms.—The general symptoms are similar to those described under Brain Abscess in the preceding pages, and only a brief résumé will be given.

Pain and headache is usually found in the occipital region on the side of the abscess or in some cases referred to the frontal region. It is usually more persistent and more severe than is found in temporosphenoidal abscess.

Neumann says the pain is usually frontal or four finger-breadths back of the ear.

Vomiting is especially common in cerebellar abscess, and when it persists throughout the second stage special attention should be directed to the cerebellum.

The temperature after the initial rise, becomes normal in uncomplicated cases.

The pulse is abnormally slow.

The respiration is normal or slower than normal.

Prostration and depression with no assignable cause is a predominant feature of cerebellar abscess.

Yawning frequently occurs as the pressure of the cerebellar abscess is increased.

Focal Symptoms.—*Retraction* of the head and neck forward and to to the diseased side occurs when there is some associated meningeal inflammation (Neumann).

Facial paralysis may occur on the same side as the abscess, due to the direct involvement of the peripheral nerve.

Changes in the eye-ground, papillitis, optic neuritis, and choked disk, are more frequently associated with cerebellar than with cerebral abscess.

Subjective vertigo is a fairly constant symptom of cerebellar involvement. This vertigo, unlike that of labyrinthine disease, bears no relation to the nystagmus (see chapter on the Labyrinth). The dizziness or vertigo is usually very persistent and may be present when nystagmus has disappeared.

The *nystagmus* in cerebellar abscess is usually rotatory and may be directed to either side, that is, the quick component may be directed to the diseased side, or may be to the normal side. (See Plate XVII.) It is usually to the diseased side. It may change its direction and be first to one side and then to the other, for instance, with the eyes turned toward the right side the nystagmus will be to the right, and conversely, if the eyes look toward the left side the nystagmus will change and also be to the left. This changing nystagmus when present, being in marked contrast to the nystagmus of labyrinthine disease, which does not change in direction when the eyes are voluntarily turned except in some cases of circumscribed labyrinthitis. Cerebellar nystagmus does not show any tendency to diminish, but rather to increase in severity as the disease progresses, giving another valuable aid in differentiating a cerebellar nystagmus from that produced by a disease of the labyrinth, in which latter condition the nystagmus tends to gradually subside after loss of function, and usually disappears in from a few hours to three weeks. The nystagmus may not be manifest until opaque glasses are applied to the eyes.

Disturbance of equilibrium is sometimes very marked in abscess of the cerebellum, indeed to such a degree that the patient is unable to remain in the upright position without falling. Other cases may show a comparatively small impairment of the sense of equilibrium, the only noticeable evidence being a slight spreading of the legs or a turning out of the toes in order to gain a broader base of support. There

is a tendency to fall backward or laterally in the direction of the side of the lesion, according to whether the abscess is situated in the vermis or in a hemisphere.

The *loss of the sense of position*, or of the *arthrodial sense*, may be demonstrated in some cases. This is done by having the patient passively move an arm or leg corresponding to the side of lesion, while blindfolded or with eyes closed, and then arresting the arm or leg in some unusual position. If the arthrodial sense is impaired he will be unable to assume the same position with the opposite arm or leg.

The Pointing Test.—Barany, of Vienna, has shown that if a normal person is blindfolded, or closes his eyes, after having previously determined the location of some object held in a stationary position, he is able to lower or raise his arm and touch the object. If the patient harbors a cerebellar abscess with focal symptoms, the hand corresponding to the side of the cerebellar lesion frequently deviates to one or the other side, while the other hand or arm corresponding to the normal side is able to point fairly correctly. With vestibular disturbance both hands deviate in the same direction, *i. e.*, in the direction of the slow component of the nystagmus.

Disturbance of motility is frequently present in cerebellar abscess. This may be noticed in the affected parts as awkwardness, tremors, incoördination, etc., or the disturbance of motility may take the form of "excessive movements." If, for example, the patient carries the point of his index finger toward the end of his nose it does not stop when the nose is reached but passes over it and violently strikes the jaw.

Babinski has drawn attention to the disturbances of *diadokokinesis* or the ability to execute rapidly successive volitional movements. *Adiadokokinesis* is the loss of this faculty. For example, if the patient be directed to rapidly supinate and pronate both hands and forearms, the hand corresponding to the cerebellar lesion will be greatly retarded, or if he is asked alternately to flex and extend the forearm upon the arm, movements of the arm corresponding to the cerebellar

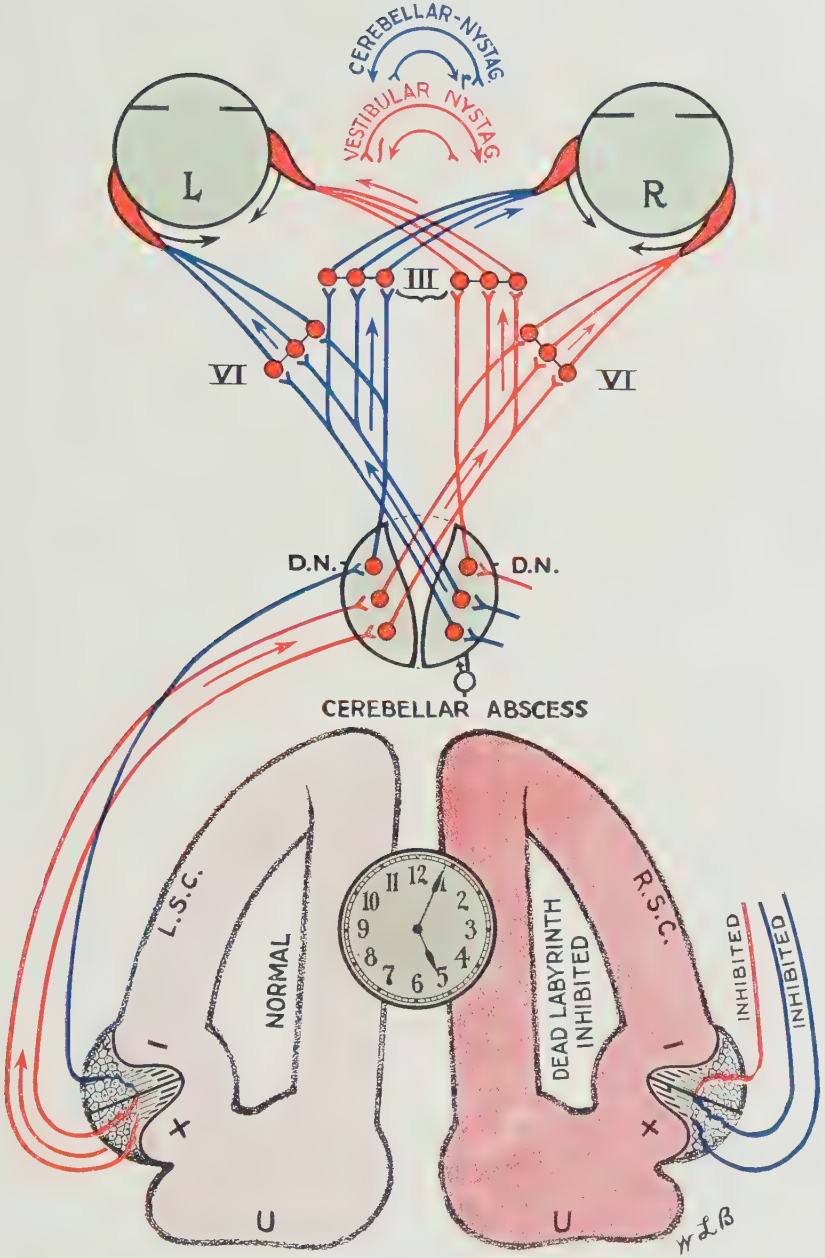
PLATE XVII.

Showing Spontaneous Nystagmus of (A) Vestibular Origin, and (B) of Central or Cerebellar Origin.

(*A*) The serous labyrinthitis affecting the right ear inhibits the labyrinth upon that side and leaves a preponderance of tonus in the left labyrinth. This causes a slow conjugate movement of both eyes to the right (slow component); the cortical reflex immediately turns the eyes to the left (quick component) and the nystagmus thus produced is spontaneous rotatory vestibular nystagmus to the left. The red arrows indicate the course of the vestibular impulses which give rise to the slow component of the nystagmus.

(*B*) The cerebellar abscess stimulates the right Deiters' nucleus (D. N.), and thereby increases the tonus on the right side. The increased tonus causes a conjugate movement of both eyes to the left (slow component) and the cortical corrective impulse immediately turns the eyes to the right (quick component) and the nystagmus is spontaneous rotatory cerebellar nystagmus to the right or side of disease. The blue arrows indicate the course of cerebellar impulses which give rise to the slow component of the nystagmus.

PLATE XVII



(*A*) Vestibular Nystagmus; (*B*) Cerebellar Nystagmus.

lesion will be slower and less regular, although the muscular force is preserved and sensibility is intact.

Disturbance of speech is sometimes present in abscess of the cerebellum. The voice may be nasal and the words emitted with suddenness or the articulation may be slow and thick.

Treatment (see Surgery of the Temporal Bone).

THROMBOSIS

A thrombus is a mass formed in the heart or peripheral vessels, the component parts of which are derived from the blood (Frazier). They are arterial, venous, capillary, or cardiac in origin, and, according to to their composition, are white, red, and mixed thrombi.

The following four factors enter into the pathogenesis of a thrombus:

1. Infective microörganisms.
2. Structural changes in the intima of the vessel or organ.
3. Disturbances of the blood current.
4. Chemical changes in the blood.

1. In the non-infective thrombus the microörganisms are absent. It is the infective type, however, with which the otologist has to deal. "The primary causative factor is a pyogenic organism, a primitive lesion, a phlebitis, and the terminal process a thrombosis or a thrombophlebitis. Thrombophlebitis, associated with such general septic processes as pyemia and septicemia, was the first to be recognized as of infective origin; subsequently, however, the infective nature of thrombophlebitis has been admitted and recognized in other diseases of infectious origin, as in the various so-called infectious diseases" (Frazier). Streptococci are the most frequent cause of this disease. A negative bacteriological finding does not necessarily preclude an infectious origin, the toxin remaining being the exciting inflammatory agent.

2. The structural changes in the intima are due to the irritation by the toxins of the bacteria. The intima becomes rough and adhesive. The injured cells of the intima liberate a fibrin ferment which favors thrombus formation. The roughened projections of the intima into the lumen of the vessel interfere with the velocity of the blood current and thereby favor thrombus formation.

3. The slowing of the blood current cannot alone cause thrombosis. If associated with changes in the intima and the presence of microorganisms, it predisposes to thrombus formation. The slowing of the blood current is attended with a rearrangement of the constituents of the blood. The white blood corpuscles incline to the periphery of the current and are admixed with a few platelets. As the current becomes slower, the white corpuscles diminish and the platelets increase in number. In some instances a projection from the intima causes a whirling motion of the current, which still further favors thrombus formation.

4. The chemical changes in the blood, while not yet demonstrated, seem to be a factor in thrombosis. A fibrin ferment is probably liberated in the infected thrombus, and it may influence the production of the platelets.

Pathology.—The thrombus is composed of the constituents of the blood in varying proportions, and are white, red, or mixed, according to whether they are formed in circulating or stagnant blood. If in circulating blood, they are white or mixed; whereas, if in stagnant blood, they are red, and have no clinical significance. Blood platelets form the nucleus of the white and mixed variety, though in the later stages they may have disappeared.

According to Frazier, the thrombus, at first composed of the normal constituents of the blood, undergoes various changes, which become an element of considerable danger. The leukocytes undergo fatty degeneration and necrosis; the red corpuscles are decolorized, irregular in shape, and pigmented. The platelets disappear and are replaced by fibrinous deposits. Softening or liquefaction occurs, and the creamy substance contains granular debris, pus cells, and microörganisms. It is in the septic variety of softening that fragments become separated from the thrombus, and, as infected emboli, are carried off by the circulation and deposited in the internal organs, usually the liver, kidneys, and lungs, where they give rise to secondary or embolic abscesses.

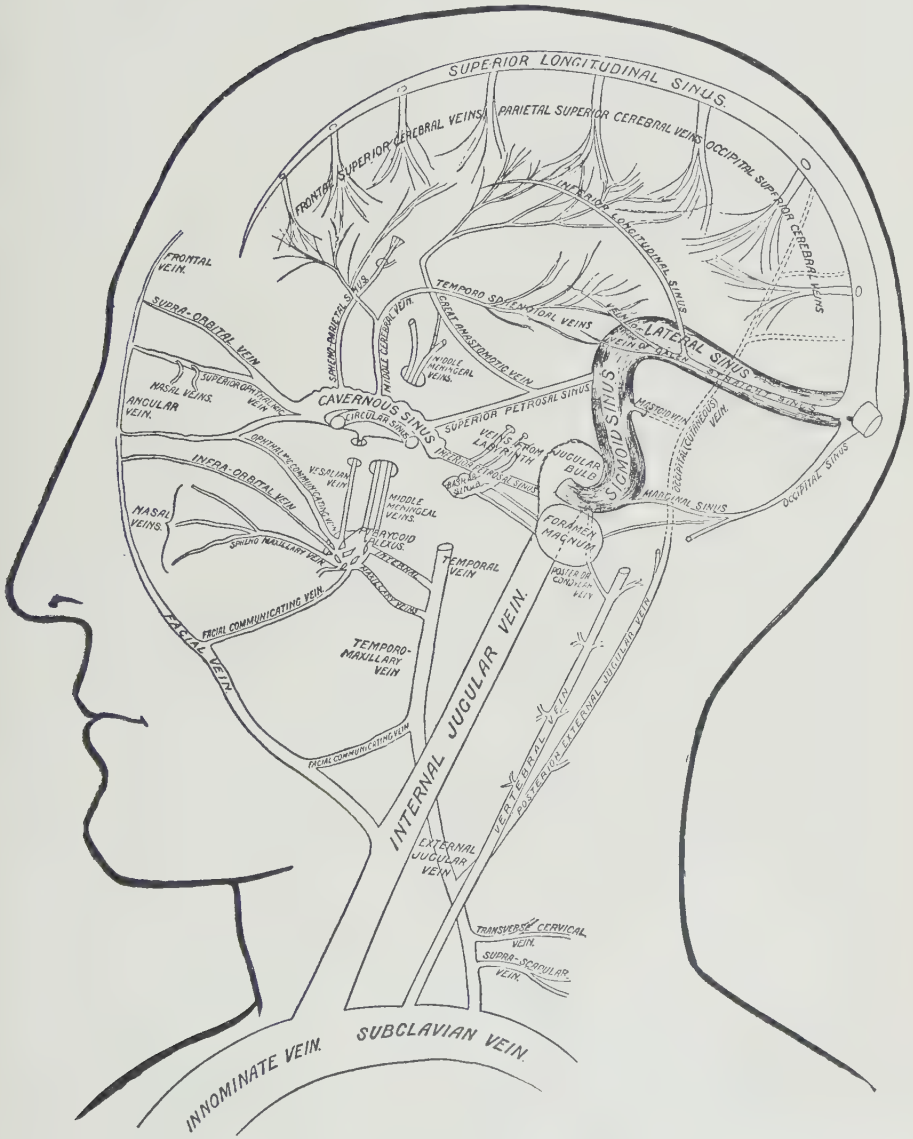
The terminal stage of a thrombus is organization, or rather a disappearance of the thrombic material and the deposit of fibrous material. At the beginning of organization the thrombus becomes infiltrated with leukocytes, and following this there is a proliferation of fixed connective-tissue cells derived from the endothelium and the other fixed cells of the intima. Bloodvessels penetrate the clot and form anastomoses with each other and with the vessels above and below the thrombus. The thrombus is absorbed, and is replaced by embryonic connective tissue rich in bloodvessels. The fibrous mass becomes firm, contracts, and may completely or partially occlude the vessels. In rare instances the fibrous tissue disappears and leaves the lumen of the vessel unimpaired.

Venous thrombi extend toward the heart or with the blood current. In thrombosis of the sigmoid or petrosal sinuses, the thrombus may extend to the jugular vein and completely occupy its lumen.

LATERAL SINUS THROMBOSIS

Etiology.—The causes of infective thrombosis of the sigmoid portion of the lateral sinus are chiefly to be found in the loss of integrity of the intima of the membranous sinus from the extension of the destructive process in suppurative mastoid or labyrinthine inflammation. So long as the intima is healthy it inhibits the coagulation of the blood in contact with it, but where its vitality is impaired by a necrosing mastoiditis its inhibitory power is lost and the blood fibrin coagulates on the affected area, and a thrombus is thus established. The thrombus may or may not occlude the lumen of the vessel. At the beginning it is limited to the external or bony aspect of the sinus, as this is the part first involved by the necrosis of the bone. The necrosis may extend from the mastoid cells of the process or from the labyrinth (in labyrinthine suppuration)

Fig. 425



Schema showing venous sinuses of the head. (After Macewen.)

to the cells lying between the labyrinth and the antrum, and thence to the antrum and mastoid cells, from whence it involves the sinus.

At the beginning the thrombus is not infected. It is only after the wall of the membranous sinus has undergone marked deterioration that the infective microörganisms penetrate it and lodge in the thrombus. There is food for thought in this fact. That is, if the condition is diagnosticated before infection of the thrombus occurs, the infection and its evil consequences could be thwarted by an exposure of the sinus and the removal of the diseased bone surrounding it without opening the sinus itself. Unfortunately, the diagnosis of thrombosis at this early stage is extremely difficult to make, and is rarely made except during a mastoid operation.

Symptoms.—The symptoms of lateral sinus thrombosis may be divided into three stages, based upon the pathological changes so minutely described by Macewen in his masterly work on *The Pyogenic Diseases of the Brain and Spinal Cord.*

First Stage.—The thrombus, partial or complete; disintegration not established.

(*a*) Slight fever.

(*b*) Rigors, usually present. Slight rigors exceptional

(*c*) Headache, slight or severe, limited to the affected side.

(*d*) Slight tenderness over the region of the mastoid emissary vein.

(*e*) Slight edema and tenderness below the tip of the mastoid in the posterior triangle of the neck.

(*f*) Leukocytosis with increased polymorphonuclear count.

Second Stage.—The thrombosis, partial or complete; disintegration and systemic absorption established.

(*a*) Temperature always above normal and distinctly fluctuating.

(*b*) Frequent rigors.

(*c*) Headache and tenderness over the mastoid emissary vein.

(*d*) Edema and tenderness below the tip of the mastoid in the posterior triangle of the neck.

(*e*) Increased leukocytosis and polymorphonuclear count.

Third Stage.—The thrombosis, partial or complete; disintegration and excessive systemic absorption.

(*a*) A chill or rigor followed by great and marked fluctuations of temperature; sometimes subnormal, and then rapidly rising to 104° or 106° F.

(*b*) Headache, severe, often excruciating.

(*c*) Marked tenderness over mastoid emissary vein and the posterior triangle of the neck. The internal jugular vein may be tender on pressure.

(*d*) Metastatic pneumonia, enteritis, or meningitis may be present, with characteristic symptoms.

(*e*) Still greater leukocytosis and polymorphonuclear count.

NOTE.—The leukocytosis and polymorphonuclear count is greater in sinus thrombosis than in simple mastoiditis.

(*f*) Coma as the fatal issue approaches.

Early Diagnosis.—If diagnosticated in the first stage, and operated

at once, nearly all cases recover. If diagnosticated and promptly operated in the second stage, before metastatic extension to the brain, lungs, bowels, spleen, etc., fully 50 per cent. will recover; whereas, if diagnosticated and operated in the third stage, the mortality rate is very high.

In view of the foregoing facts, it is evident that all cases of suppurative otitis media, especially if there is a secondary acute manifestation, should be critically studied to detect the earliest sign of sinus involvement. Such observations cannot be made unless the patient is placed in bed, with a trained nurse in attendance, and the temperature, pulse, and respirations recorded every three hours. Inquiry as to the presence of a unilateral headache, not necessarily severe, should be made two or three times daily. The surgeon should examine for tenderness over the mastoid emissary vein and the posterior triangle of the neck. The occurrence of a rigor, even if slight, should excite suspicion, and lead to most careful inquiry as to all the other symptoms.

If a diagnosis is not positively made before a mastoid operation is performed, the sigmoid portion of the sinus should be exposed and its membranous wall examined. Infective perisinus abscess may be present, without involvement of the intima of the sinus. Sometimes the external surface of the membranous sinus is velvety and granular in appearance, the smooth surface and pearly gray color normal to the sinus being absent. I have seen cases like this recover after exposing the membranous sinus. They recovered because the intima (lining) of the sinus was not yet involved. The drainage of the perisinus abscess checked the inward extension of the infective process, and thus thwarted the formation of a thrombus in the sinus.

In one case, observed by the author, in which perisinus abscess was present and the lumen of the sinus open, there afterward developed thrombosis of the lateral and the cavernous sinuses. The question as to the advisability of opening such a sinus is of considerable importance. The author believes it should be done, and done thoroughly, the sinus being walled off after exploration and packed with iodoform gauze.

A partial thrombosis of the sigmoid sinus may sometimes be demonstrated by compressing the sinus with the finger and noting the uneven or nodular surface when collapsed. The use of a hypodermic needle is useless for diagnostic purposes, as it may penetrate beyond the thrombus, and withdraw blood from the normal blood current.

In complete thrombosis of the sinus, palpation with the finger gives the sense of a doughy resistance. After full exposure of the sinus, it should be palpated to determine, as far as possible, the probable extent of the thrombus. If it is doughy over the full area of the exposure, the clot probably extends to or above the knee, and below to the jugular bulb.

The knowledge thus gained may determine the advisability of a still further exposure of the jugular bulb. (See Thrombosis of the Jugular Bulb.) In complete thrombosis there is no flow of blood upon incising the sinus, nor will the hypodermic needle draw fresh blood.

The Diagnostic Value of Blood Cultures in Otitic Disease, Especially in Sinus Thrombosis.—In articles first published by E. Libman,

and later by Libman and Celler, and still later by Seymour Oppenheimer, the conclusions are reached that from the number of cases which have been studied as regards the phenomena of bacteriemia in relative otitic suppuration, it can be definitely stated, that (*a*) the findings of bacteriemia in the presence of a suppurative disease of the middle ear and its adjoining osseous spaces is proof evident that there is an infective thrombosis of the sigmoid sinus, and such a finding is warrant for immediate operation. (*b*) Furthermore, that if, following the sinus operation, streptococci are found in the blood after the first day, it indicates that the internal jugular vein is also involved in the thrombosis and measures for its operative relief should be at once carried out.

If Oppenheimer's conclusions are sustained by further observations and clinical experience, we will have at our command a means for the early diagnosis of sinus thrombosis that has hitherto been denied us. An early diagnosis before characteristic symptoms appear will enable the otologists to carry out surgical measures that will avert great dangers to this class of patients. As he so well states, the great problem of sinus thrombosis is not so much the etiology or the operative technique, but the early recognition of infection of the sinus, as the danger increases greatly as each day passes.

Unfortunately, thrombosis of the lateral sinus (the sinus nearest the antrum and cells, and in consequence most often infected) is not always characterized by distinct and well-defined symptoms. Symptoms such as chills, high fever, sweats, rapid pulse, etc., followed by a rapid recession of the temperature to or below normal are not always present, especially when the infection is of the streptococcus type and there is a significant differential blood count. In some cases the chill is not well marked, and might escape the attention of the surgeon, unless a very careful watch of the patient is maintained, which might elicit the fact that the patient experienced slightly creepy or chilly sensations. Sweating may or may not follow these minor chills. These chilly sensations may be complained of especially following the mastoid operation, and they are symptoms of the greatest importance, particularly if the case is of a typical type.

Oppenheimer also calls attention to the fact that it has been considered in many cases that a blood count, especially an increase in the polynuclear percentage to over eighty, indicates the presence of a focus of pus from which the general circulation is being infected, and in many typical cases this is essentially true, and such a count is of value in arriving at a diagnosis. In many cases, however, the blood count is of little or no value in interpreting the condition present, as the polynuclear percentage may be high and yet no sinus disease exist, or the reverse may be the case.

It appears, therefore, that many cases of sinus thrombosis do not present clear and well-defined symptoms, and that the disease often progresses to a dangerous stage before the physician suspects its presence, and even though operative measures are adopted, the mortality

rate is extremely high. If, however, as Oppenheimer leads us to hope, the use of blood cultures in estimating the bacteriemia present will enable the otologist to make an early diagnosis, many lives will be saved.

Oppenheimer cites the investigations of Leutert, Lehart, Neurenberg, Kobrak, and Hasslauer as showing that an early and accurate diagnostic sign of sinus thrombosis may be elicited by means of blood cultures, to estimate the bacteriemia present. With this aid, the otologist could by an early operation, before the general system became the seat of serious pyemic infection, avert a fatal issue.

The investigations thus far show (with the exception of that done by Duel and Wright) (*a*) that bacteriemia has not been demonstrated in simple otitis media, simple mastoid disease, extradural abscess, or in brain abscess; and (*b*) positive cultures have been demonstrated in meningitis and sinus thrombosis. The earlier the culture is taken the more often and positive will the results be.

Duel and Wright have not corroborated the findings of the other observers, as they also found bacteriemia in disease of otitic origin, other than meningitis and sinus thrombosis. All other investigators, as reported by Oppenheimer (including himself), have found bacteriemia to be a reliable sign of sinus thrombosis, provided other foci of infection, as meningitis, endocarditis, phlegmonous pharyngitis, etc., are excluded.

The differences in the findings of Duel and Wright, and Libman and Oppenheimer may perhaps find explanation when the experimental work of Stenger is taken into account. The necessity of preventing the interpretation of a finding of "primary bacteriemia"—a condition common to most systemic pyogenic invasions—and the really significant "secondary bacteriemia" must be borne in mind.

As the technique of blood cultures for determining bacteriemia is a technical laboratory process, I will not describe it. I will only state (*a*) that blood or fluid should be withdrawn from the lateral sinus at the time of the mastoid operation, also (*b*) 15 c.c. of blood from a peripheral vein of the arm, and both submitted to a laboratory expert for cultural purposes.

If an infective thrombosis is in the sinus, numerous colonies of streptococci will be found in the cultures from the sinus, while they will not be so numerous in the cultures from the blood withdrawn from the peripheral vein.

If in a suspected case of sinus thrombosis the first culture is negative, new blood should be withdrawn the next day for another culture, when the result may be found positive. A thrombus may be present and the needle fail to reach it at the first attempt, or it may be localized at a point not reached by the needle, or the general system may not as yet be invaded. So long as suspicious symptoms continue after operation, make daily cultures until a positive result is obtained or the patient's condition improves.

In the event of a positive result, open the sinus and evacuate its contents. If the operation upon the sinus is extensive enough, the bacteriemia as shown by the culture of blood from the peripheral vein will

rapidly progress toward sterility. If, however, after the lapse of three or four days the blood is still bacteriemic, it signifies infective thrombosis of the jugular vein (a focus unreached by the sinus operation), and it should be resected at once. If, after this, the blood cultures from the peripheral vein continue to show bacteriemia, metastatic culture foci are present elsewhere in the body, and they should be sought for and eliminated if possible. (For treatment, see Surgery of the Temporal Bone, Chapter XLVIII, and Vaccine Therapy at close of Chapter X.)

Prognosis.—The prognosis depends chiefly upon the stage in which diagnosis and operative procedures are made. If made in the first stage, nearly all will recover. If in the second, about one-half will recover. If in the third, the mortality rate is high. If not operated, nearly all cases terminate fatally.

FIG. 426

One of the author's cases of cavernous sinus thrombosis of otitic origin. The drawing shows the case in the early stage before thrombus had extended to the left side through the circular sinus.

Here is a field in which an early diagnosis and an early operation are the means of saving life; whereas a late diagnosis, even with operative interference, will in a majority of subjects result in death.

Thrombosis of the Jugular Bulb.—Whiting has formulated the following test: Compress the membranous sinus as near the bulb as possible,

and draw the finger upward to empty it; the compression is then removed, and if the vessel fills from below, it is assumed that the bulb is not thrombosed. Allport believes this procedure is dangerous, as it may liberate infective clots and disseminate the infection to other parts of the body. Such occurrences have not been reported.

Grunert exposes the jugular bulb by opening the mastoid, exposing the sinus, and ligating the jugular. The retro-auricular and cervical (jugular) incisions are then united and the tip of the mastoid process is resected. The soft parts are then pulled forward and loosened as far as the jugular foramen. The bone should be removed until the jugular bulb is exposed. (See Surgery of the Temporal Bone.)

Cavernous Sinus Thrombosis.—Thrombosis of the cavernous sinuses is rare. Two cases of otitic origin and one from an abscessed tooth have occurred in the author's practice, though this is probably an exceptional experience, as many aurists of equally large experience have reported no cases.

When of otitic origin, it usually extends from the superior or inferior petrosal sinus to the cavernous sinus. When it complicates inflammation of the nasal accessory sinuses, it extends from the secondarily infected eye through the ophthalmic vein to the cavernous sinuses.

The general symptoms are similar to those present in thrombosis of the lateral sinus. The characteristic symptoms are the marked edema of the peri-ocular tissues and the protrusion of the eyeball, as shown in Fig. 426, which illustrates one of the three cases just mentioned.

The first case occurred in a girl, aged twelve years, seven years after an attack of scarlet fever, at which time she had an acute otitis media purulenta. During the interim (except the last week of her life) she was said to have had no ear discharge. The mastoid symptoms and otorrhea developed rapidly. When the author saw her on the third day she was greatly prostrated and septic, one eye slightly protruding. The first chill and rigor occurred on the fourth day. The lateral sinus was exposed, but was apparently not thrombosed. Death occurred three days later.

In the second case, a young woman, the cavernous sinus was thrombosed secondarily to the lateral sinus. The lateral sinus was exposed, and the thrombus removed as high and as low as possible without establishing a flow of blood. The patient gradually became stupid, finally comatose, and died one week after the lateral sinus was exenterated.

The third case occurred in Mrs. W., aged thirty-three years, who for four weeks had suffered from an abscessed wisdom tooth on the right side. Upon examination the nasal accessory sinuses and the ears were normal. The right eyeball protruded, as in Fig. 426. Two days prior to the time I saw her the temperature remained steadily at 101° F. The next day it remained at 102° F. And on the next, the day I saw her, it stood at 103° F. She complained of slight chilly sensations, but of no distinct chill. There were no remissions in the temperature.

The edema of the tissues of the orbital cavity was so characteristic that I unhesitatingly pronounced the disease to be thrombosis of the

cavernous sinus, though the attending rhinologist had made a tentative diagnosis of ethmoidal and sphenoidal empyema in spite of the absence of suppuration. The family physician had diagnosticated typhoid fever. Two days later the postmortem examination corroborated my diagnosis of cavernous sinus thrombosis.

Symptoms.—The symptoms depend on whether one or both sinuses are affected. It usually begins in one and spreads to the other through the circular sinus. The symptoms shift from one eye to the other, a differential point between thrombosis of the cavernous sinus and inflammations confined to the orbital cavity.

(*a*) Pain may be occipital, supra- and infra-orbital, and in the vertex.

(*b*) Exophthalmos and edema of the eyelids and side of the nose are characteristic symptoms due to venous obstruction.

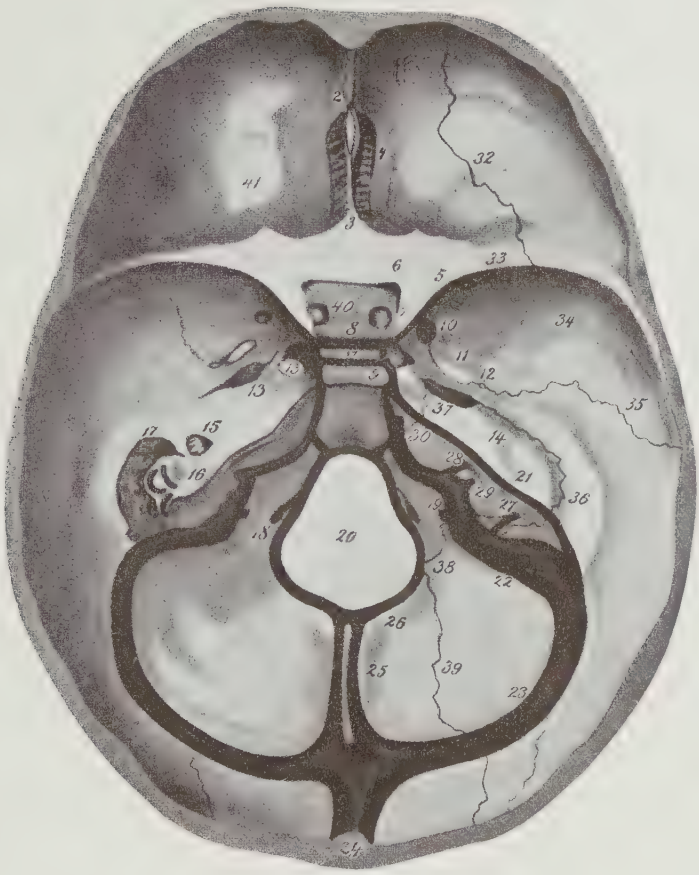
(*c*) Drooping of the eyelids (ptosis), strabismus, and pupillary reactions due to pressure on the third nerve are also present.

(*d*) Edema of the pharynx and tonsil on the same side is occasionally present.

The nerves involved are the second, third, fourth, and sixth, and the first division of the fifth. The third is the most constantly involved, as is evidenced by the ptosis. The duration of the disease varies from a few days to several months, generally only a few days. The death rate is extremely high.

Treatment.—The treatment is chiefly palliative. When tension of the conjunctiva is extreme, it may be slit or punctured. The eyeball may be removed, together with the thrombosed vessels, with a view of affording some relief from the pain and distress. Such interference should be undertaken only in extreme cases, as there is no hope of effecting a cure by this procedure. Attempts to operate upon the sinus have generally failed, though favorable reports have been made. (See Surgery of the Temporal Bone.)

PLATE XVIII



Base of the Skull: Left Labyrinth Exposed on the Right Side, the Grooves in the Base of the Skull are Shown, also the Sinuses of the Dura Mater. Two-thirds Lifesize. (After Bruhl-Politzer.)

1, crista frontalis (on the left, beginning of the superior longitudinal sinus); 2, foramen cecum (emissarium Santorini); 3, crista galli; 4, lamina cribrosa (olfactory nerve); 5, lesser wing of sphenoid; 6, optic foramen (optic nerve, ophthalmic artery); 7, anterior clinoid process; 8, sella turcica, flanked by the median clinoid process; 9, dorsum ephippii, with posterior clinoid process; 10, foramen rotundum (second division of fifth nerve); 11, foramen ovale (third division of fifth nerve); 12, foramen spinosum (middle meningeal artery and recurrent branch of fifth nerve); 13, carotid canal and foramen lacerum anterius (great and lesser superficial petrosal nerves, Eustachian tube, and tensor tympani muscles); 14, anterosuperior surface of pyramid; 15, cochlea; 16, semicircular canals; 17, tegmen tympani and roof of antrum laid open; 18, anterior condyloid foramen (twelfth nerve); 19, posterior condyloid foramen (emissarium Santorini); 20, foramen magnum; 21, superior petrosal sinus; 22, sigmoid sinus (descending portion); 23, lateral sinus (horizontal portion); 24, superior longitudinal sinus and torcular Herophili (confluence of the sinuses); 25, occipital sinus; 26, occipital sinus; 27, vein of aquæductus vestibuli (emerging at the external aperture of aquæductus vestibuli); 28, internal auditory vein (emerging from the internal auditory meatus); 29, vein of aquæductus cochleæ (emerging at the external aperture of aquæductus cochleæ); 30, inferior petrosal sinus draining the cavernous sinus; 31, circular sinus (Ridley); 32, groove traversing anterior fossa of skull; 33, sinus of lesser wing of sphenoid; 34, groove of meningeal artery; 35, transverse groove through middle fossa of the skull; 36, longitudinal groove through petrous portion of temporal bone (tegmen tympani); 37, groove through apex of pyramid; 38, transverse fissure (between posterior condyloid foramen and foramen magnum); 39, longitudinal groove through posterior fossa of skull; 40, impressio carotica (corresponding to the bend in the internal carotid artery); 41, juga cerebralia and impressiones digitatæ.

CHAPTER XLVIII

SURGERY OF THE TEMPORAL BONE

Treatment of the surgical diseases and complications included in this chapter are: (1) acute mastoiditis; (2) chronic mastoiditis; (3) Bezold's mastoiditis; (4) necrosis of the semicircular canals; (5) necrosis and suppuration of the semicircular canals and vestibules; (6) necrosis and infection of the cochlea and semicircular canals; (7) thrombosis of the lateral sinus; (8) thrombosis of the jugular vein; (9) thrombosis of the jugular bulb; (10) extradural abscess in the middle fossa of the skull; (11) serous meningitis; (12) abscess of the cerebrum; (13) abscess of the cerebellum; (14) facial paralysis; and (15) postauricular fistula.

Ossiculectomy.—The removal of the malleus and the incus for the relief and cure of chronic suppurative otitis media has fallen into disuse since Macewen's work on *The Pyogenic Diseases of the Brain and Spinal Cord* appeared in 1893. His presentation of the efficacy of the radical mastoid operation for this purpose was so convincing that it has been almost universally adopted by otologists throughout the world. There is now a reactionary tendency to differentiate the cases, and to adopt various surgical procedures, according to the characteristics of each case. In some instances the radical mastoid operation is elected as the best method of procedure; in others, the meatomastoid operation is preferred; and in still others, the otologist is content to remove the granulation tissue and secretions through the external meatus by means of small curettes, the syringe (Figs. 427 and 428), and inflation and irrigation through the Eustachian tube by means of a Weber-Liel catheter.

Technique.—*The Anesthetic.*—Ossiculectomy may be performed under local anesthesia, though it is usually quite painful. In the author's experience the most reliable anesthetic mixture is composed of equal parts of cocaine, carbolic acid, and menthol. Instil a few drops of this mixture into the meatus, and at the end of twenty minutes its full anesthetic effect is obtained.

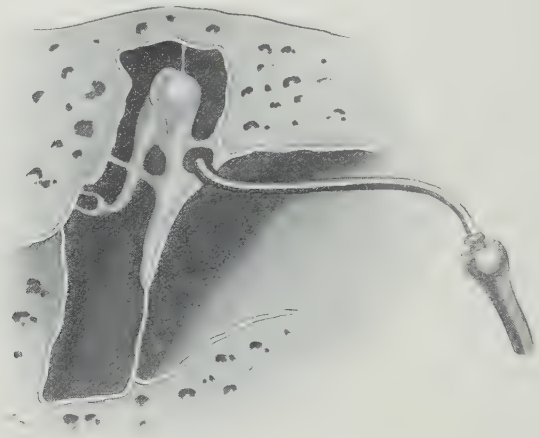
It is usually preferable, however, to administer a general anesthetic, as this insures a painless operation.

Preparation of the Ear.—The auricle and external meatus should be scrubbed with soap and water, followed by an alcohol bath. A cotton-wound toothpick or applicator may be used for the purpose. If a general anesthetic is to be given, the patient should be placed in a hospital the day before the operation, and the bowels and diet regulated as for the mastoid operation.

Incision of the Membrana Tympani.—The incision may begin at the margin, at the junction of the anteroinferior and the anterosuperior

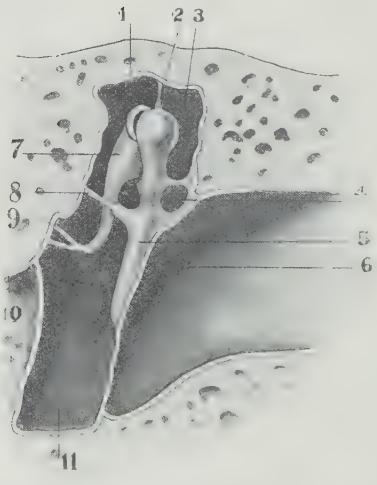
quadrants of the membrane (Fig. 429), and be extended upward to the malleus, thence downward along the anterior border of the handle to its

FIG. 427



Irrigation of the attic through a perforation in the membrana flaccida.

FIG. 428



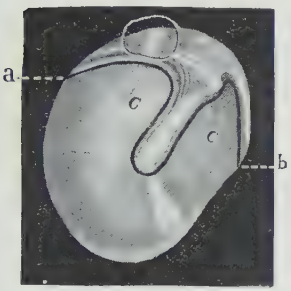
1, the attic; 2, suspensory ligament of the malleus; 3, external space of the attic; 4, Prussack's space; 5, malleus; 6, external meatus; 7, incus; 8, ligament attaching malleus to inner wall of the tympanic cavity; 9, stapes; 10, promontory; 11, cavum tympani.

umbo, or lower extremity, thence upward along its posterior border to the upper limit of the membrane, and thence downward along the posterior margin of the membrane to the junction of the postsuperior and postinferior quadrants of the membrane, as shown in Fig. 429. This incision makes two flaps of the membrana tympani, which drop downward and expose the tympanic cavity (Fig. 429). This operation preserves a large portion of the membrana tympani and favors speedy regeneration in the process of repair. The great objection to it is that the lower half of the membrane interferes with the drainage of the tympanic cavity.

Instead of the above incision, the entire membrane, or the fragments of it, if it is largely destroyed, may be removed by making an incision around its entire margin and along both borders of the handle of the malleus. This provides for drainage during the after-treatment.

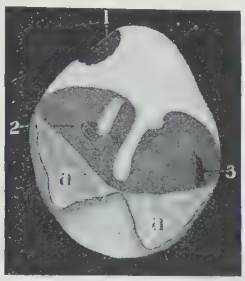
Removal of the Malleus and Incus.—The malleus should first be removed and then the incus. The attachments of the tensor tympani muscle and the tendinous attachments of the malleus to the tympanic wall should be severed. Various instruments have been devised for this purpose, the best of which are Sexton's small angular blades (Fig. 431), which should be passed behind the handle of the malleus and carried

Fig. 429



The right membrana tympani with a perforation at the margin of the postsuperior quadrant over the lenticular process of the incus, indicating necrosis of the incus and of the mastoid antrum. The line *a b* is the line of incision preliminary to the removal of the malleus and incus. The flaps of membrane thus made drop down and expose the upper half of the tympanic cavity to view (Fig. 430).

Fig. 430



The incision and flaps preliminary to ossiculectomy. 1, perforation in the membrana flaccida; 2, stapes in the oval window; 3, tympanic orifice of the Eustachian tube; *a a*, the membrana tympani—flaps turned downward.

upward to the tendinous attachment of the tensor tympani muscle. It should then be introduced through the space occupied by the membrana (pars) flaccida, to sever the ligamentous attachment to the outer wall of the tympanic cavity.

Delstanche's ring knife (Fig. 432) may also be used to remove the malleus. Its ring blade should be insinuated around the handle of the

Fig. 431

Sexton's ossiculectomy knives.

Fig. 432

Ring currettes for removing the malleus.

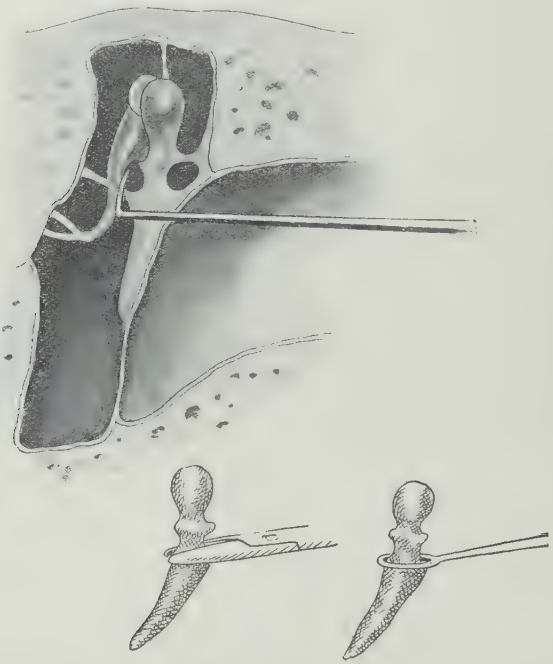
malleus and passed upward as far as possible, cutting the attachment of the tensor tympani muscle.

Having thus severed some of the attachments of the malleus, it should be removed either with the ring knife or with forceps (Fig. 433).

The ring knife, or dull ring, should encircle the handle of the malleus as high as possible, and then, with a rocking or side-to-side motion,

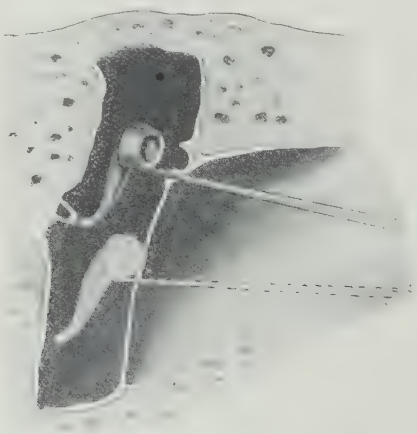
combined with a downward pull, the malleus is dislodged and removed through the external meatus.

FIG. 433



Showing the severance of the ligamentous attachments of the malleus. After this is done the malleus is grasped with the forceps or a ring curette, and drawn downward until its head is disengaged from the attic. It is then removed through the external auditory meatus.

FIG. 434



Removal of the incus with the incus hook, after the removal of the malleus. The hook should be introduced posterior to the incus, the incus pushed forward and downward. If it is pushed backward it is apt to become lodged in the aditus ad antrum.

If the forceps are used, the handle of the malleus should be seized as high as possible and removed in the same manner as with the ring knife (Fig. 433).

The incus is not so easily dislodged from its position, as its long process is often beyond the grasp of the forceps, and even when it can be seized it is so fragile that it is apt to break. The incus hook (Fig. 434) is the best instrument for its removal. Another difficulty encountered is the liability to dislocate it backward into the aditus ad antrum. To obviate this mishap, the incus hook should be introduced behind the body of the incus and passed upward and forward over its body. The hook should then be pressed downward and slightly forward, thus dislodging the incus and bringing it into the lower portion of the tympanic cavity, where it may be removed with the forceps.

The stapes is never removed in the operation, as to do so would subject the labyrinth to infection and would cause pronounced deafness.

Hemorrhage.—Bleeding may be controlled by mopping the tympanic cavity with adrenalin or with a hot 1 to 2000 bichloride of mercury solution.

Dressings and After-treatment.—The best dressing is a loosely applied strip of sterile gauze extending from the tympanic cavity to the auricle. The cavity of the auricle should be loosely filled with gauze and cotton and the whole covered with an ethereal solution of collodion, which holds in place as effectually as a large and cumbersome bandage.

The after-treatment consists in applying similar dressings and the cleansing of the tympanic cavity with cotton-wound applicators, inflation through the Eustachian tube, and the reduction of granulations with carbolic acid or dehydrated crystals of chromic acid, for a period of about one month, or until the ear is dry.

If the operation is unsuccessful, either the radical or the meatomastoid operation may be performed. The percentage of cures (chronic otitis media purulenta) is very small.

ACUTE PRIMARY MASTOIDITIS

Indications for Surgical Intervention.—It is taken for granted that the usual abortive therapeutic measures, as (*a*) the application of leeches (or the artificial leech) over the mastoid process and in front of the tragus, (*b*) the instillation of a 12 per cent. solution of carbolic acid in glycerin into the auditory meatus, (*c*) free incision of the membrana tympani, (*d*) ice over the mastoid process, (*e*) heat, cathartics, etc., have been used without success.

1. These and perhaps other therapeutic measures having failed to abort the infectious and destructive process in the cavum tympani and mastoid antrum and cells, the disease tends to become chronic, a fact which may constitute a sufficient reason for performing a simple exenteration of the mastoid antrum and cells. To wait for other and more definite indications might develop the necessity for a much more radical

operation, or even lead to serious intracranial complications, and endanger the life of the patient. Intervention, when threatened chronicity is imminent, requires a comparatively simple surgical procedure, which almost always results in a permanent cure, often with but little or no impairment of the functions of the ear.

2. Bulging or sagging of the posterior superior wall of the external auditory meatus near the membrana tympani is due to the involvement of the mastoid cells below and anterior to the antrum (cells of Kirchner), and is a positive indication for the mastoid operation.

3. Pain over the mastoid antrum and tip which is not relieved by the application of ice (one to four hours), alternated with heat, over a period of from twenty-four to forty-eight hours, is an indication for the simple mastoid operation. The pain signifies congestion, edema, or granulations which block the drainage of the secretions. Pressure necrosis and toxemia rapidly develop under such conditions, and if the pain persists, the mastoid antrum and cells should be opened.

4. Edema and redness of the mastoid region signify blocking of the secretions, and, if the condition is not relieved by leeching, ice, heat, etc., constitute another indication for surgical intervention.

5. The presence of a subperiosteal abscess over the mastoid process, especially in adults, having its origin through a fistulous opening in the mastoid cortex, is an indication for the operation. In infants and children such a condition often has its origin beneath the periosteum of the meatus, the mastoid cortex being intact, hence a subperiosteal abscess and the infection of the ear and mastoid antrum may be cured by an incision (Wilde's) through the skin over the mastoid process.

6. Meningeal irritation (complicating acute mastoiditis), as evidenced by convulsions (in infants and children), delirium, intense headache, etc., may call for the mastoid operation.

7. Other and more serious intracranial complications, as circumscribed meningitis (epidural abscess), serous meningitis, thrombosis of the lateral sinus, etc., constitute positive indications for the mastoid operation.

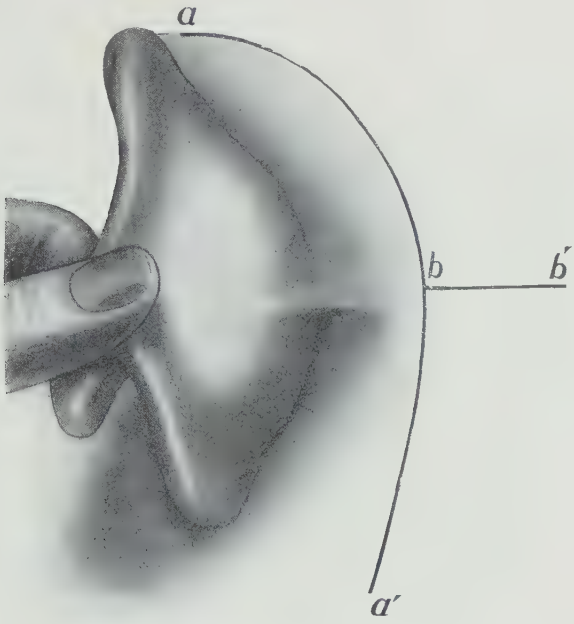
The Simple Mastoid Operation in Acute Mastoiditis.—The Technique.—The preparation of the patient and anesthesia will not be discussed farther than to say that the head should be shaved, scrubbed, etc., over an area extending at least three inches from the attachment of the auricle, both above and behind it. Otherwise the patient should be prepared and anesthetized as for any other major surgical operation.

The incision back of the auricle should be so extended as to fully expose the entire field of the operation. In adults, the primary incision (Fig. 435, *a*, *a'*) should begin at the mastoid tip one-half inch posterior to the attachment of the lobule of the auricle, and extend upward behind the auricle, gradually approaching its attachment, until, when near the superior attachment, it should be about one-fourth of an inch posterior to it. It should then be extended anteriorly to a point immediately above the superior attachment of the auricle (Fig. 435, *a*). If upon retracting the posterior flap the operative field (posteriorly) is not fully

exposed, a secondary incision (Fig. 435, b, b') should be made at right angles to the primary one. It should begin on a level with the external auditory meatus and be extended backward for a distance of one inch (Whiting). In those cases in which the mastoid cells extend well back toward the occiput, it will be necessary to extend the secondary incision accordingly.

The primary incision (Fig. 435 a, a') should be first superficially outlined with the scalpel to ensure clean-cut edges, proper curve, and extension. It should then be carried through the entire substance of the skin, subcutaneous tissue, and the periosteum.

Fig. 435



The postauricular mastoid incision. a, a', the primary incision; b, b', the secondary incision.

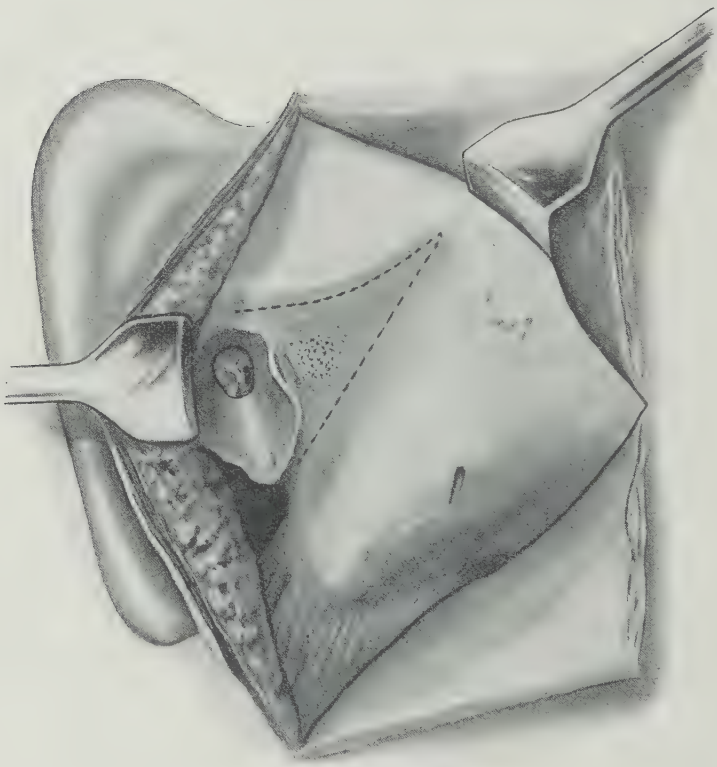
The Elevation of the Cutaneous Periosteal Flaps.—The skin and periosteum should be elevated together. Great care should be taken to preserve the periosteum, as the subsequent repair of the wound will depend somewhat upon the integrity of this structure. With this object in view, the author devised the periosteal elevator shown in Fig. 436. The periosteal blades are at right angles to the axis of the handle of the instrument. Experience has shown that this angle is best adapted to the clean elevation of the mastoid periosteum. The instrument is provided with two right-angle elevators, one elevating on the pull, and the other on the push. But little difficulty will be experienced in elevating the upper two-thirds of the anterior and posterior flaps; whereas, the lower third will be elevated with difficulty, as the tendinous fibers of the sterno-

mastoid muscle pierce it. The tendinous bands of this muscle should be cut with short, blunt scissors from the external cortex of the mastoid tip before elevation of the periosteum is attempted. If this is not done, long muscle fibers may be pulled from the stenomastoid muscle, thus opening avenues of infection in its substance (Whiting).

Fig. 436

The author's mastoid periosteal elevator.

Fig. 437

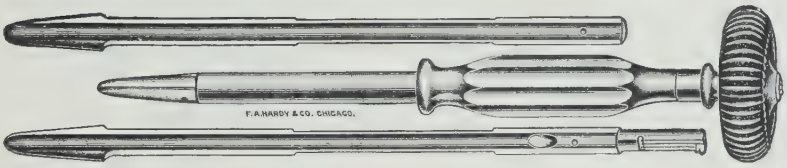


The anatomical landmarks for opening the mastoid antrum. The suprameatal triangle, the spine of Henle, and sieve-like depression.

The Anatomical Landmarks.—Having elevated the cutaneoperiosteal flaps, the external characteristics of the mastoid process and auditory meatus should be noted. To experienced surgeons, this requires but a few seconds of time. The first concern should be to determine the location of the mastoid antrum, as it forms the deeper landmark of the

mastoid process. It is usually located at a depth of about one-half inch beneath the mastoid cortex and a little above and behind the cavum tympani. There are four more or less constant external landmarks which will guide the surgeon to the mastoid antrum. The one most constantly present is the area of sieve-like perforations in the mastoid cortex just behind the external opening of the meatus (Fig. 437). These small openings contain minute vessels which bleed after the periosteum is elevated. The surface of the bone should be mopped dry, and in a moment the bleeding-points will appear. Another landmark usually present is the suprameatal spine, or the spine of Henle (Fig. 437). It is a small triangle or diamond-shaped bony lip projecting outward and forward from the posterior margin of the external auditory meatus. The point for entering the antrum is immediately behind the spine. The third landmark for locating the mastoid antrum is the suprameatal triangle (Fig. 437). The upper boundary of the triangle is formed by the lower border of the posterior ridge or root of the zygomatic process; the posterior inferior boundary is formed by an imaginary line extending from the posterior end of the root of the zygoma to the inferior portion

Fig. 438



The Russian perforator.

of the spine of Henle, or, if this is not present, to the posterior inferior margin of the auditory meatus. An opening made in the anterior portion of this triangle near the auditory meatus will enter the mastoid antrum. The fourth landmark to the antrum is the direction of the posterior superior wall of the bony portion of the auditory meatus. This is ascertained by introducing a straight probe into the meatus along its posterior superior aspect and noting the angle of inclination in relation to the general surface of the mastoid cortex. Having noted the foregoing anatomical landmarks, the exenteration to expose the antrum should be begun at the point indicated by the first three landmarks described and extended inward in a direction parallel with the probe, as suggested in the description of the fourth landmark. The usual direction of the posterior superior wall of the bony meatus is markedly inward, and slightly downward and forward. After excavating for a depth of one-half inch (sometimes more, rarely less), the outer extension of the mastoid antrum may be looked for. The sigmoid sinus is sometimes near the surface. Should the mastoid cortex be carious, the fistulous tract may be followed to its origin in the antrum or cells without regard to the external landmarks.

Opening the Mastoid Antrum.—The Russian perforator (Fig. 438) or a gouge may be used to expose the mastoid antrum. If the Russian perforator is used, its tip should be placed in the suprameatal triangle (Fig. 437), with the long axis of the instrument parallel with the probe placed against the posterior superior wall of the meatus, as described under External Landmarks. The mastoid cortex is then perforated with a boring movement of the perforator, the bone shavings passing into the hollow chamber of the instrument. The instrument should be removed from time to time to examine the bottom of the bony wound, to see when a pneumatic space is uncovered. When this occurs, a dark spot will be found in the bottom of the wound. When the mastoid cortex is carious the tissue may be excavated with a curette, the anatomical landmarks being disregarded. A curved silver probe

Fig. 439

Allport's mastoid mallet.

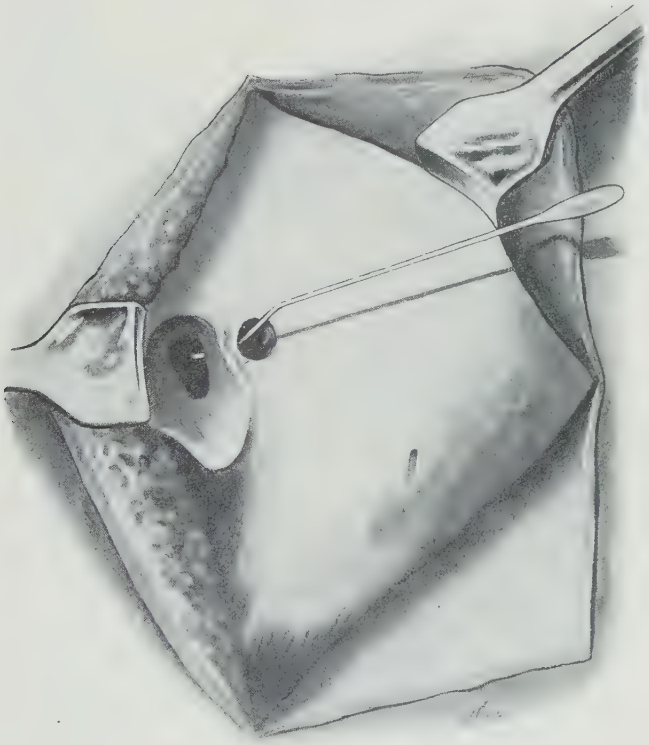
should be introduced into the pneumatic space, the curved tip being directed anteriorly. If the pneumatic space is the mastoid antrum, the tip of the probe will pass forward through the aditus ad antrum into the cavum tympani, as shown in Fig. 440. If the pneumatic space is a mastoid cell, the probe will not pass forward through the aditus ad antrum. If the sigmoid portion of the lateral sinus is located anteriorly against the posterior wall of the auditory meatus, the perforator will uncover it, but will not injure its membranous covering. Herein is another reason for preferring the Russian perforator to the gouge.

I no longer use the Russian perforator, though it is a good instrument for beginners in otology. I use the Alexander gouges and first remove the cortex, and then open the antrum with a No. 6 gouge. The mastoid cortex is outlined with the No. 14 gouge and mallet, the gouge being so directed as to only include the cortex. After the cortex is thus separated around its circumference the large chip of bony cortex is freed from its attachment to the underlying cells and removed *en masse*. The mastoid cells are then removed with a rongeur forceps, or if the bone is soft or necrotic, with a large spoon curette. The antrum

is then opened with a No. 6 Alexander gouge, the gouge being directed toward the bridge of the nose.

As Whiting has so well shown, the external conformation of the mastoid process will show the position of the sigmoid portion of the lateral sinus. The sinus, being a large vessel, requires space; hence, the area of greatest external bulging or convexity of the mastoid cortex may be taken as a guide to the location of the sinus. When the convexity is at

FIG. 440



The opening into the mastoid antrum made with the Russian perforator. The fact that the silver probe passes forward through the aditus ad antrum into the cavum tympani is proof that the pneumatic space at the bottom of the wound is the antrum and not a mastoid cell.

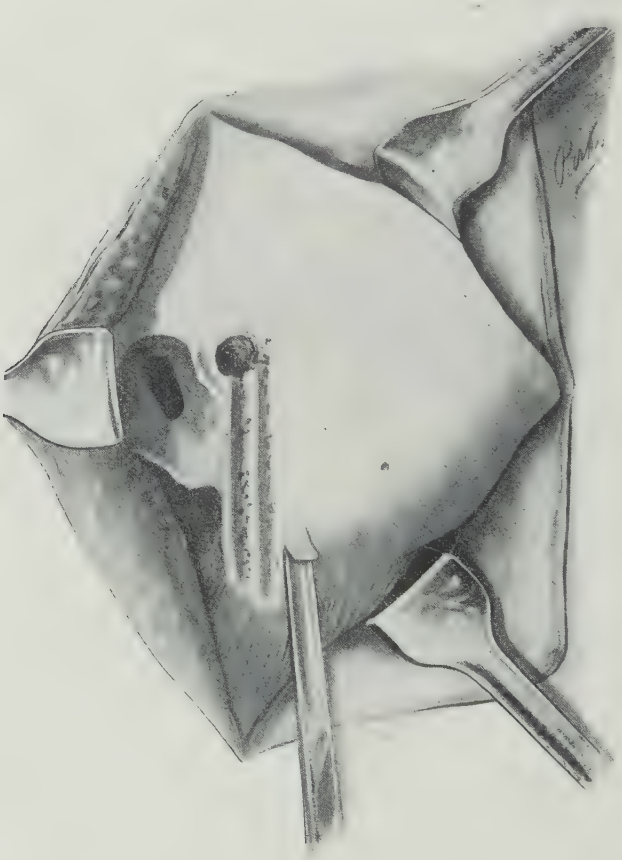
the middle portion of the mastoid cortex, it is well out of the way in opening the antrum. When, however, the anterior portion of the mastoid cortex is elevated, and the posterior wall of the meatus drops at right angles from it, the sinus is located anteriorly, and will be exposed in opening the antrum. In such subjects, it may be necessary to open the antrum by removing the posterior wall of the meatus after the method of Stacke.

Having exposed the mastoid antrum, its dimensions and extensions should be determined with a bent probe introduced through the bony

wound. The whole outer wall of the antrum should then be removed with a gouge and mallet or the rongeur forceps.

The Removal of the Mastoid Cortex.—The mastoid cortex may be removed in parallel shavings (Fig. 441), as recommended by Whiting. From three to four grooves are made, exposing the superficial cells. The gouge may be applied at either the mastoid tip, as shown in the drawing, or at the level of the mastoid antrum. Care should be exercised to avoid injuring the mastoid emissary vein shown at the posterior

Fig. 441

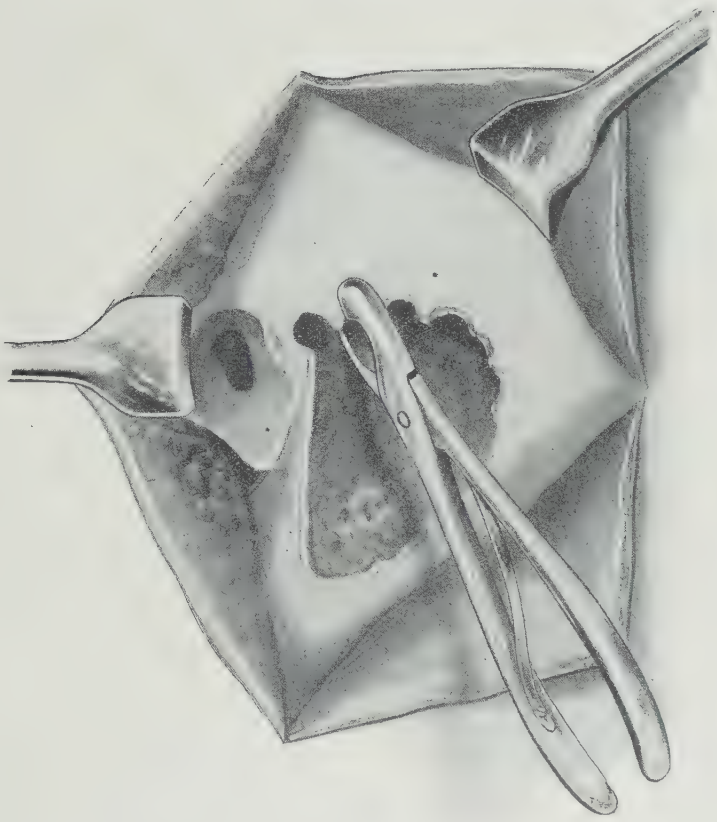


The removal of the cortex of the mastoid process.

portion of the mastoid process (Fig. 441). This vein opens into the sigmoid portion of the lateral sinus, and, when injured, bleeds profusely and persistently. It may be readily closed by placing the tip of some blunt instrument against the opening of its bony canal and tapping it smartly with the mallet. (See preceding paragraph for another method of removing the mastoid cortex.)

The Exenteration of the Mastoid Cells.—After the cortex is removed the cells should be broken down and removed with the curette and the rongeur forceps. If the intercellular walls are soft or necrosed, they may be removed with a curette. If they are firm, the rongeur forceps is better for the purpose. The overhanging edges of the mastoid cortex should be removed with the rongeur forceps (Fig. 442) until all cells are completely exposed and accessible to curettement. Large mastoid cells are often found in the tip of the process. These may be the focal centre of the infection and the only place where pus is found. The cells should

FIG. 442



The completion of the removal of the mastoid cortex with the rongeur forceps. The cells may also be removed with the same instrument.

therefore be exposed to the tip in all cases, as otherwise the focal centre of infection may not be exposed and the operation fail of its purpose. All cells should be opened, but not completely obliterated, as the mucous membrane is essential to the rapid healing of the wounds.

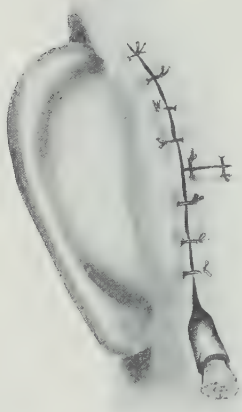
The Irrigation of the Wound.—As the infective microörganisms in acute mastoiditis are usually quite active and virulent, and it being

almost impossible to prevent them coming in contact with the soft tissues, it is a wise precaution to irrigate the wound with a 1 to 4000 bichloride solution at about 110° F. The external auditory meatus should also be scrubbed and irrigated with the same solution, care being exercised to avoid injuring the membrana tympani and dislocating the ossicles. Tincture of iodine may also be applied to the wound.

The Closure of the Cutaneous Wound.—As drainage must be maintained for several days, and the cavum tympani is not exposed by the operation, it is necessary to provide for drainage through the posterior wound.[1]

The cutaneous wound is not, therefore, completely closed at the time of the operation. The upper two-thirds is sutured, as shown in Fig. 443, while the remaining lower third is left open for the introduction of the drainage tube and gauze.

Fig. 443



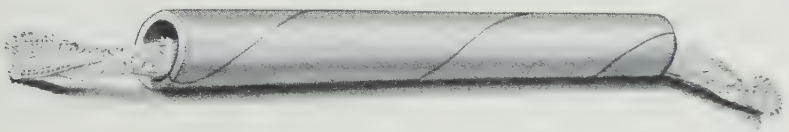
Method of closing the mastoid incision after the simple mastoid operation in acute mastoiditis. The spiral rubber tube and gauze drain in the lower angle of the incision prevent disfigurement.

The Dressing.—The object of the dressing is twofold—namely, to promote drainage and protect the wound from further infection while the process of repair is in progress. In order to accomplish the first object, the dressing should be so applied as to insure free drainage. According to the author's experience, only so much gauze should be introduced into the depth of the bony wound as to carry off the secretions to the outer absorbent dressing. To pack the wound with gauze is poor practice, as the gauze becomes saturated with the secretions, retains them in the wound, where they bathe its walls and retard the reparative process. On the other hand, if only a small wick of gauze is carried to the bottom of the bony wound, the secretions are carried out as fast as formed, and the healing process progresses uninterruptedly and rapidly to recovery. A spirally cut soft rubber tube, with a small wick of gauze placed loosely in its lumen (Fig. 444), should be introduced into the mastoid wound. A small wick of gauze is also placed in the external auditory meatus. The outer absorbent and protective dressing consists of gauze pads, 5 x 6 inches, placed over the auricle and mastoid wound, and held in position with a bandage applied in a fan-shaped figure (Fig. 465). The bandage should not be applied under the chin or around the neck, as it is uncomfortable and unnecessary.

[1] In performing the simple mastoid operation for acute mastoiditis it is unnecessary to expose the external auditory meatus, as is shown in the drawings. The drawings are thus made to show the anatomical landmarks for teaching purposes, and for reference in describing the radical and the meatomastoid operations for chronic mastoiditis.

The After-treatment.—The first dressing should be removed at the expiration of three days, the wound cavity gently mopped dry with a cotton-wound applicator, and another spiral tube dressing introduced. The meatus should also be mopped until freed of secretions, a fresh gauze wick applied, and the whole covered with gauze pads, as in the first dressing. The sutures should be inspected before redressing the wound, and if stitch abscesses are present, the sutures should be removed.

FIG. 444



A spirally cut rubber tube with a small wick of gauze in its lumen constitutes one of the best drainage dressings after mastoid operation.

If the wound is healthy, they may be left in position until the fourth or fifth day. The wound should be dressed daily as described, until the secretion diminishes to a small amount, after which the tube should be omitted and only a small wick of gauze introduced. The cavity will then rapidly fill in from the bottom with healthy granulation tissue, and at the end of from three to six weeks be entirely healed, with a slight depression at the lower angle of the wound.

In exceptional cases infection of the labyrinth, sinus thrombosis, etc., may develop subsequent to the operation and modify the course of the reparative process, or even necessitate the adoption of other surgical procedures hereinafter described.

CHRONIC MASTOIDITIS

Chronic mastoiditis is one of those diseases which resists simple methods of treatment, and for the last fifteen years the radical mastoid operation has been the only treatment that insured any real success. Two years ago, however, Charles J. Heath, of London, called attention to the brilliant results obtained by a less radical procedure, whereby the hearing was greatly improved and the disease apparently cured. Körner, Stacke, and others previously described an operation somewhat similar to that described by Heath. Since then the author has performed forty-five operations with a modified technique, with good results. The difference between the methods is that the author makes a complete exenteration of all the pneumatic cells of the temporal bone and uses a modified Ballance plastic meatal flap, as in the radical operation. To this new operation he has given the name *meatomastoid*. The (*a*) radical and the (*b*) meatomastoid operations will therefore be described as remedial measures for the cure of chronic mastoiditis.

The Radical Mastoid Operation.—Technique.—*The Removal of the Cortex and the Exenteration of the Mastoid Cells.*—The patient is prepared as for the simple mastoid operation in acute mastoiditis. The

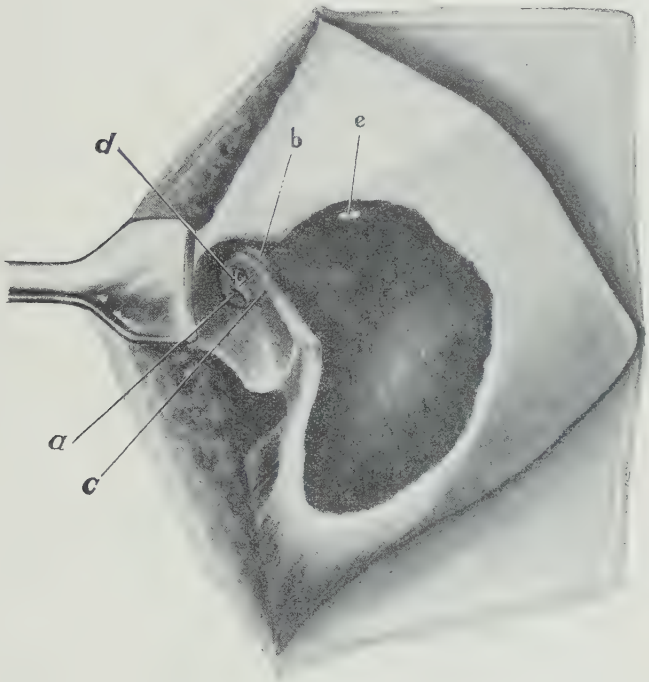
mastoid antrum and cells are exenterated as in the simple operation in acute mastoiditis (see Simple Mastoid Operation, Figs. 440, 441, and 442), with this difference: In the simple mastoid operation there is no necessity for making a complete exenteration; whereas in the radical operation all pneumatic spaces in the mastoid process and zygomatic root, as well as those in the posterior wall of the pyramid of the petrous portion of the temporal bone (Jansen), are removed. It is not enough to expose the cells to view, they must be totally exenterated. To fail in this respect may lead to the necessity of performing a secondary operation. It has been claimed by some operators, who do not completely remove these cells, that it was impossible to tell when all of them had been removed. They also claim that 25 per cent. of the radical mastoid operations had to be followed by secondary operations. While it is true that the operator cannot positively state that *all* the cells have been removed, he can at least endeavor to remove them, and in the vast majority of cases he will be successful. It has been the author's earnest endeavor during a period of ten years to remove all the pneumatic cells, whether in the mastoid process, zygomatic root, or in the posterior wall of the pyramid, with the result that only 1 per cent. of all cases have required a secondary operation. The good results obtained were partially due to the painstaking removal of all the pneumatic cells in the temporal bone and to certain points of improved technique to be narrated in subsequent paragraphs of this chapter.

The Removal of the Posterior Wall of the Bony Meatus.—Having completed the exenteration of the mastoid antrum and cells, the posterior wall of the bony meatus is removed with a chisel, as shown in Fig. 445. In the removal of this wall there are certain anatomical structures which may be injured if due care is not exercised to avoid them. These structures are the facial nerve, the external or horizontal semicircular canal (Fig. 445, *b*), and the dura of the middle fossa of the skull (Fig. 445, *e*). The facial nerve emerges from the petrous portion of the temporal bone and passes backward along the superior margin of the inner wall of the cavum tympani just above the oval window (Fig. 445). It then courses downward across the inner and inferior wall of the aditus ad antrum, immediately below the upper and deeper portion of the bony wall of the meatus (Fig. 445, *c*). From thence it passes downward, deeply buried in the plate of bone forming the posterior wall of the auditory meatus, and emerges just posterior to the styloid process. The nerve is most liable to injury in removing the deep portion of the posterior meatal wall directly over the aditus ad antrum, as it is only protected in this area by a thin but dense bony covering. Should the chisel by any mischance cross the space of the aditus ad antrum (the channel of communication between the cavum tympani and the mastoid antrum) to its inner and inferior wall, across which the facial nerve passes, facial paralysis may follow. In the removal of the posterior wall of the meatus the more superficial parts may be removed without fear of damaging the facial nerve, while the deeper portion should be removed with due care to avoid this danger.

After the facial nerve crosses the floor of the aditus ad antrum it turns sharply downward and emerges near the styloid process. As it makes

the bend (Fig. 445, *c*) it rises almost to the level of the posterior portion of the annulus tympanicus, to which the membrana tympani is attached. It is obvious, therefore, that the lower portion of the posterior wall of the meatus cannot be removed deeper than the annulus tympanicus without injuring the nerve.

FIG. 445



Anatomical landmarks after the complete exenteration of the mastoid process and cavum tympani: *a*, the round window; *b*, ridge of horizontal, semicircular canal; *c*, the facial ridge; *d*, the stapes in the oval window; *e*, the dura of the middle fossa exposed through a perforation in the tegmen antri.

To recapitulate: The upper portion (patient in erect position) of the posterior wall of the meatus may be removed in its entirety, or down to the aditus ad antrum, whereas the lower portion should only be removed down to the level of the annulus tympanicus or posterior segment of the drum-head. The complete removal of the wall, insofar as it is compatible with the integrity of the facial nerve, is shown in Fig. 445. In the meatomastoid operation the removal does not include the annulus tympanicus. When completely removed, the upper bony wound extends inward at almost right angles to the lateral plane of the head, whereas the inferior bony wound extends inward and upward at an acute angle to this same plane.

Another important anatomical structure in the immediate vicinity of the facial nerve as it crosses the floor of the aditus ad antrum is the external or horizontal semicircular canal (Fig. 445, *b*). It is located a little above and behind, and more superficially, than the facial nerve at

this point. The precautions taken to avoid injuring the nerve will at the same time protect the semicircular canal. Indiscriminate curettage of the inner wall of the cavum tympani (middle ear) may injure either the facial nerve, the semicircular canal, or the stapes and oval windows (Fig. 445).

All these structures should be constantly held in mind during the removal of the posterior bony wall of the meatus. The dura of the middle fossa (Fig. 445, *e*) is in but slight danger of exposure, and even when exposed the probability of infection is slight, as the pathogenic microorganisms of chronic infection are but moderately virulent. One of the greatest objections to the radical mastoid operation is that the hearing is often impaired, especially after a period of one year. The impairment of the hearing is due to two factors, namely: (*a*) To the displacement of the foot-plate of the stapes in the oval window (Fig. 445, *b*) at the time of the operation, and (*b*) to the gradual displacement and fixation of the foot plate of the stapes by cicatrices and contraction subsequent to the operation. On the other hand, it is claimed that the radical operation is justified, because in many cases it is the only known procedure that will cure the chronic otorrhea and protect the patient from the dangers incident to such a pathogenic process in the temporal bone. Life insurance companies have justly refused policies to persons affected with chronic otorrhea, and have granted them when an aural surgeon of repute has made a written statement that they were cured by the radical operation.

With these facts in mind, the radical mastoid operation has been and is still a justifiable procedure in properly selected cases. It is important, however, that the surgeon should take every precaution in the performance of the operation, consistent with safety to the life and health of the patient, to preserve the hearing as much as possible. In order to do this, the stapes and the oval window must be protected and extraction of the stapes from the oval window most carefully avoided. Should the latter occur, it opens an avenue of infection to the labyrinth, which means the almost certain loss of hearing. Fortunately, infection has rarely occurred when this accident has happened in the course of the radical operation, as the infective bacteria are usually of low virulency.

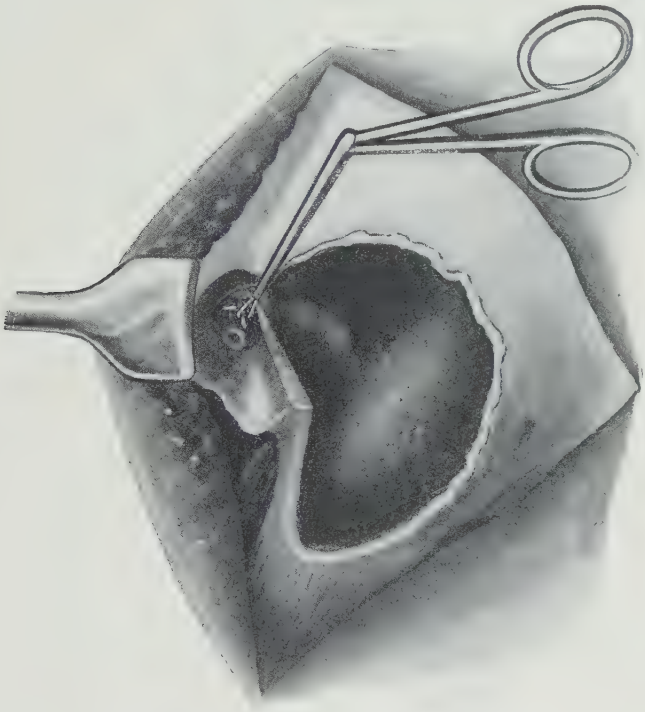
The removal of the posterior bony wall of the meatus converts the cavum tympani, mastoid antrum, and the mastoid cells into one large irregular cavity (Fig. 445), which is easily drained, and, if the plastic surgery of the meatal skin flaps is properly executed, results in a cure of the disease in more than 95 per cent. of the cases.

The Removal of the Malleus and Incus.—The removal of the malleus and incus, or their necrotic fragments, is an essential part of the radical operation, as it has been held that if they are left in position the attic of the middle-ear cavity will not be sufficiently drained. This is true to a degree, as the bodies of these bones partially form the floor of the attic, and their presence interferes somewhat with the exit of the secretions from the attic or upper portion of the cavum tympani. Furthermore, the complete removal of the bony partition involves the fracture and removal of a portion of the annulus tympanicus, to which the membrana

tympani is attached. In addition to this the incus, the long process of which projects backward into a sulcus of the bone forming the wall of the aditus ad antrum, would, in many instances, be dislocated and thus rendered useless as a functionating mechanism of the ear.

The *technique* of the removal of the malleus and incus is comparatively simple if the skin incision or incisions have been sufficiently extended to allow the complete exposure of the auditory meatus and cavum tympani. The primary skin incision (Fig. 435, *a*, *a'*) should, at its upper limit, extend one-half inch anterior to the upper attachment of the auricle. This will allow the auricle to be retracted far enough forward to expose the meatus and cavum tympani.

Fig. 446



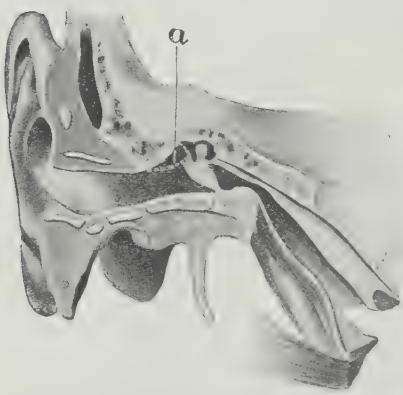
The removal of the malleus and incus in the radical mastoid operation.

When the posterior bony wall of the meatus is removed, the middle-ear cavity should be packed with cotton saturated with a 1 to 2000 solution of adrenalin chloride to check the hemorrhage. After the lapse of five minutes it should be removed and the contents of the cavum tympani inspected. The manubrium or handle of the malleus should then be seized with small alligator forceps, dislocated downward, and removed. The incus should be likewise removed. Instead of the alligator forceps a small curette may be used, though the danger of dislocating and extracting the stapes is thereby increased (Fig. 446).

The Removal of the Outer Wall of the Attic and Atrium.—The outer wall of the attic (superior wall of the external bony meatus) should be removed to fully expose the tegmen tympani to inspection and curettement. This procedure also gives the surgeon direct access to this region during the after-treatments. This is accomplished with a chisel or gouge, as shown in Fig. 447, *a*.

The outer wall of the atrium (inferior wall of the meatus) should also be removed. This may be done by curetting the anterior and posterior margins of the annulus tympanicus, and chiselling away the deeper portion of the floor of the external meatus (Fig. 447). The failure to observe these points of technique may defeat the object of the radical operation and necessitate the performance of a secondary operation.

FIG. 447



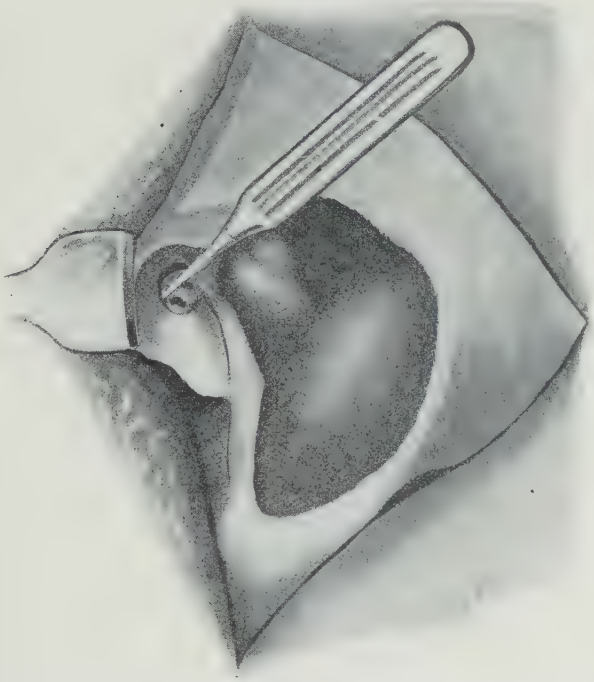
Schema showing the removal of the outer wall of the attic (*a*) (upper deep wall of the meatus) in the radical mastoid operation, to expose the attic in the after-treatments.

The Removal of Necrosed Bone from the Cavum Tympani.—Necrosis of the tegmen tympani (roof of the attic) is present in a majority of the subjects of chronic mastoiditis, a fact which gives color to the claim that the radical operation should always, or at least usually, be performed in these cases. This phase of the subject will be more fully discussed under the meatomastoid operation in chronic mastoiditis. All necrosed tissue in the tegmen tympani, or elsewhere in the walls of the cavum tympani, should be carefully but thoroughly removed with a small, sharp curette. The region of the oval window and the promontory, as well as the external semicircular canal, should be inspected, under adrenalin ischemia, with a strong reflected light for necrosed bone and granulation tissue, and, if found, the proper surgical procedures should be instituted to improve the conditions of the labyrinth which the necrosis and granulations indicate are present.

Curettage of the Eustachian Tube.—Many failures attending the radical mastoid operation are attributed to the infected and purulent discharge from the tympanic end of the Eustachian tube into the cavum tympani, subsequent to the operation. With this fact in view, it has been recommended that the tympanic end of the tube should be curetted, or burred out, to promote its closure by granulation tissue and cicatricial contraction (Fig. 448). The author has repeatedly performed this procedure, with an almost unbroken record of failures. He attributes the failures to the fact that in nearly every instance the suppuration within the tube had its origin either in a chronic epipharyngitis or a chronic ethmoidal and sphenoidal infection, to which the salpingitis is often

due. Epipharyngitis may also be caused by the enlargement of the posterior ends of the turbinated bodies, and to the presence of adenoids. If either of these conditions is present, it should be surgically corrected. The failure of the tube to close may also be due to the fact that too large a burr was used. To be successful, the burr should be small enough to reach to the isthmus of the Eustachian tube. If the sinus disease and epipharyngitis are corrected, the curettage of the Eustachian tube would almost invariably result in its permanent closure.

Fig. 448



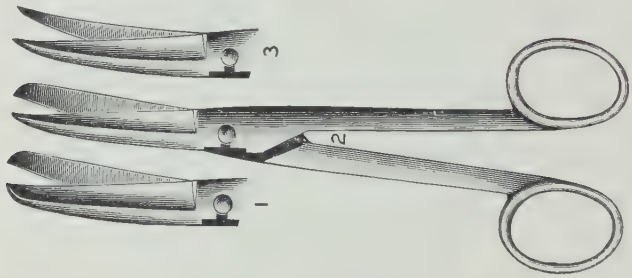
Curettage of the tympanic end of the Eustacian tube to cause it to close. A small burr or curette should be used to reach the isthmus of the tube.

Inspection of the Bony Wound.—Having completed the surgery of the bone, the wound should be dried with small gauze tampons and the application of adrenalin. Fistula of the external semicircular canal should be especially searched for. If present, it is indicated by a small granular area just posterior and above the facial ridge in the region of the aditus ad antrum. If found it should not be probed or otherwise disturbed, as this would break down the wall of granulation tissue deposited there, and might give rise to an acute labyrinthine inflammation and cause death. If anything is done at all it should be freely opened, as shown in the surgery of the labyrinth. As a matter of fact, most of these cases

will recover without an operation other than the radical mastoid operation, as this establishes free drainage and checks the necrotic process.

The Plastic Surgery of the Cutaneous Meatus.—The success of the radical mastoid operation often largely depends upon the proper use of the skin of the auditory meatus in lining the bony cavity of the mastoid wound. The bone of the mastoid process is frequently sclerosed, and affords scant soil for the formation of granulation tissue with an epidermis covering. The granulation tissue in such subjects is poorly nourished, as the blood supply from the underlying bone is scant, and infection, therefore, often occurs. The reparative process is thus hindered, and the after-treatment may be extended over a period of several months. This deplorable state of affairs may be largely overcome by the proper disposition of the meatal skin flaps against the bony walls of the mastoid wound. The plastic flaps thus reflected become adherent to the walls of the mastoid wound, and immediately cover a large portion of the bone which would otherwise have to depend upon the reparative granulation tissue, springing from the bone. In addition to this, the full blood

Fig. 449



Curved flat scissors.

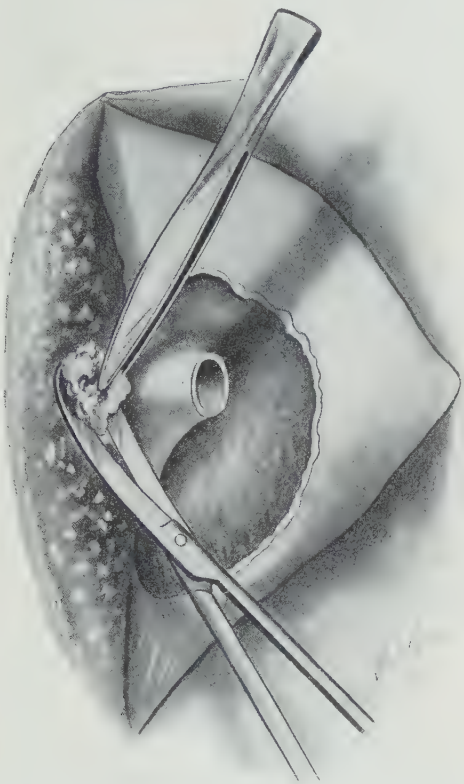
supply of the meatal flaps insures the rapid extension of the granulation tissue from their edges. The scant blood supply from the sclerotic bone of the mastoid process is thus complemented by that of the meatal skin flaps, and a speedy regeneration and epidermization of the entire mastoid wound may be confidently expected. In exceptional cases it will be necessary to resort to plastic skin flaps from the margins of the mastoid wound, or upon Thiersch grafts, as recommended by Charles Ballance. (See Thiersch Grafts.)

The *technique* of the formation and application of the plastic flaps of the meatus to be described is after the method recommended by Ballance. The form of the flaps is after Ballance. The suturing to hold them in position is, so far as known, original with the author.

Before making the incision in the meatus all the tissue on the posterior surface of the cutaneous meatus should be removed with short, stout, curved scissors (Fig. 449). This should be carried to the extent shown in Fig. 451, which shows the whole of the meatus and a portion of the concha divested of all tissue except the cartilage of the concha. The

skin of the concha is included in the upper plastic flaps. This extensive removal of all the tissues, as shown, is essential, because by so denuding them the meatal flaps can be more perfectly and extensively applied to the bony walls of the mastoid wound. It is obvious that the meatal flaps, with the thick, tendinous, fibrous, muscular, and cartilaginous tissues attached to them, could not be properly reflected and adapted to the walls of the mastoid wound.

FIG. 450

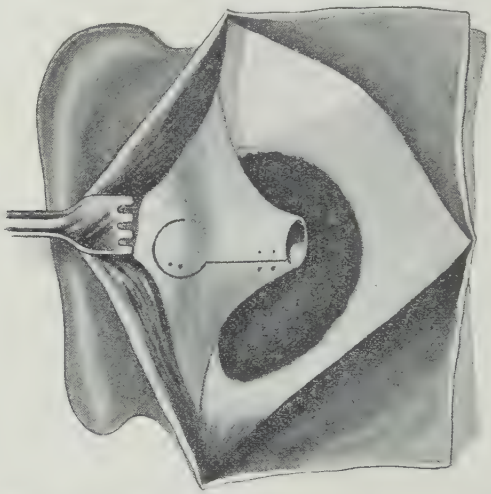


Removing the fibrous and muscular tissue from the posterior surface of the cutaneous meatus and concha, preparatory to making the plastic meatal flaps.

Having prepared the meatus and concha as described in the preceding paragraphs, and as shown in Fig. 451, the Ballance incision, sometimes referred to as the "shepherd's crook" incision, should be made. While it is by no means as easy as might be inferred from the schematic drawings, it is nevertheless comparatively so if the superfluous tissue is removed as recommended. The blades of Allport's divulsion forceps (Fig. 452) should be introduced into the meatus with the tips at the inner end of the meatal tube. They should then be spread, to put the meatal tube upon a slight tension, and should be placed so that the open space between them is at the posterior inferior segment of the tube, in order

that the straight incision may be made through this portion of the meatus, while the curved portion is made from the anterior surface of the auricle, as shown in Fig. 453. If the cartilage of the conchal portion of the upper

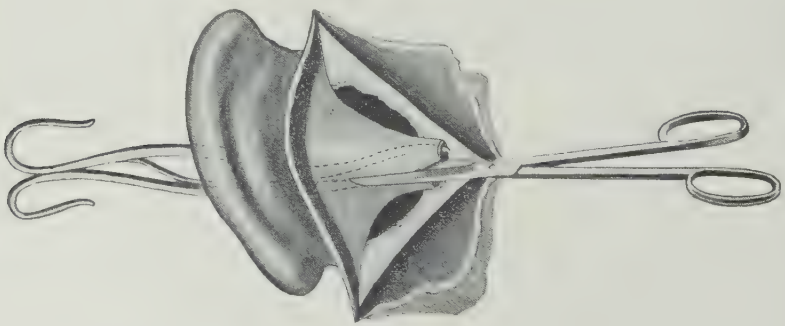
Fig. 451



The Ballance incision. The straight portion is made in the posterior inferior portion of the meatus, and the curved portion in the concha. The curved portion should be made from the anterior aspect of the concha (Fig. 453).

flap has not already been removed, it should be done at this time, as it will otherwise interfere with the placement and attachment of the flap to the bony wall of the mastoid wound.

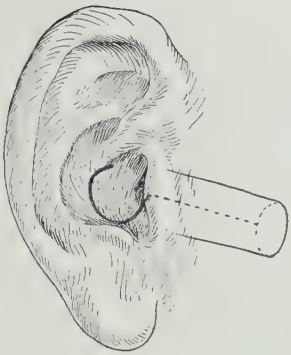
Fig. 452



Showing the method of splitting the posterior wall of the skin meatus with Allport's meatus divulsor in position to convert it into flaps for reflecting into the upper and lower portions of the mastoid bone cavity.

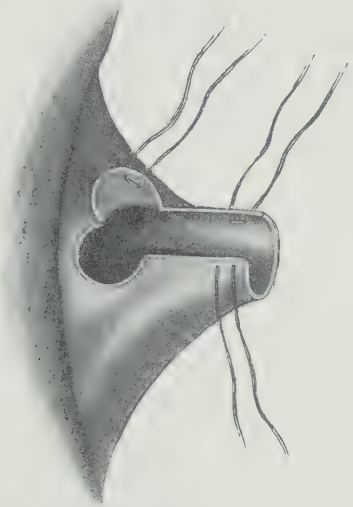
Ballance stitches the flaps to the posterior fleshy surface of the anterior or auricular mastoid flap. According to the author's method, the plastic meatal flaps are anchored to the posterior mastoid flaps, as shown

FIG. 453



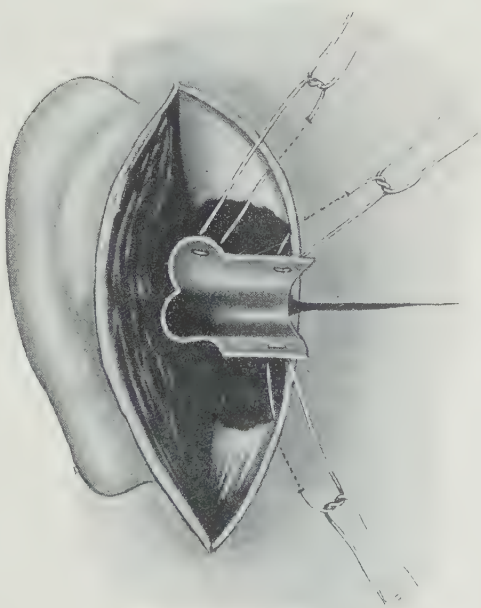
The Ballance plastic meatal incision. The incision begins in the posterior wall of the meatus (straight dotted line) and extends into the concha in the form of a shepherd's crook.

FIG. 454



The plastic flaps slightly retracted with the anchor sutures in position.

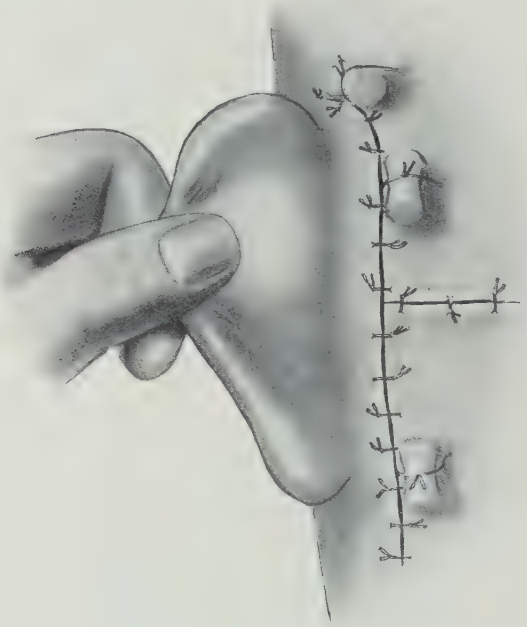
FIG. 455



The plastic meatal flaps with the anchor sutures in position. When the auricle is placed in its proper position and the anchor stitches are drawn over the rolls of gauze (Figs. 456 and 457) the plastic meatal flaps will partially line the mastoid wound.

in Figs. 454, 455, and 456. Two sutures are used in the superior meatal flap, one in the conchal portion, one in the meatal portion, and but one in the abbreviated inferior meatal flap (Fig. 451). One thread of each suture is introduced beneath the skin and subcutaneous tissue of the posterior mastoid flap for a distance of three-quarters of an inch, and then through these tissues to the surface of the skin. The other thread of each suture is placed in the primary mastoid incision (Figs. 454, 455, and 456). Before piercing the mastoid skin with the sutures, the auricle and mastoid flaps should be placed in their proper relations.

Fig. 456



The postauricular incisions closed and the anchor sutures tied over small rolls of gauze. The anchor sutures retract the plastic meatal flaps into the mastoid wound, when they become adherent and partially cover the bony surface with true skin. The whole surface is finally covered by extension from the borders of the plastic flaps.

to the head, and traction should be made upon each suture until the flaps assume the proper position in the mastoid wound. The conchal suture should be thus tested and its location determined. The meatal suture of the superior meatal flap should next be tested, and, finally, the inferior meatal suture. The flaps should be properly located and stitches in the posterior mastoid flap placed accordingly. The ends of the sutures should then be secured with artery forceps until the mastoid incision is completely closed by sutures. The anchor sutures should then be tied over small rolls of gauze (Figs. 456 and 457), beginning with the upper, and thence to the lower ones, until the flaps

assume the desired positions in the mastoid wound. The upper flap is drawn against the roof of the mastoid wound, while the lower is drawn

FIG. 457

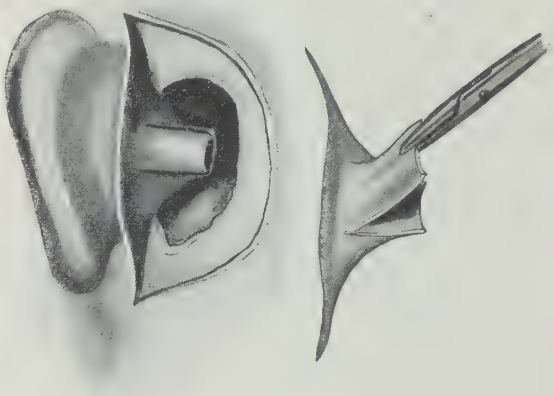
The drainage dressing consists of a spirally cut soft rubber tube with a small wick of gauze in its lumen.

FIG. 458

The Siebermann Y-plastic incision of the concha and skin meatus. Three flaps are formed by it, an upper and a lower meatal flap and a V-shaped conchal flap. The cartilage should be removed from the V-shaped conchal flap, and each should be drawn backward into the mastoid wound by sutures and fixed in position.

over the facial bridge. The bony walls being removed, and the cutaneous flaps reflected into the mastoid cavity, and permanent free drainage and ventilation of the middle ear and mastoid cavities thereby assured, the dressings may be applied *via* the external auditory meatus, as shown in Fig. 457. Other methods of making the plastic meatal flaps are shown in Figs. 458 to 463.

Fig. 459



Showing the Troutmann tongue flap, which should be reflected into the mastoid wound and held in apposition to its posterior surface by small pledgets of gauze packed over cargile membrane. Remove the gauze in forty-eight hours.

Fig. 460

The Panse plastic incision of the meatal skin.

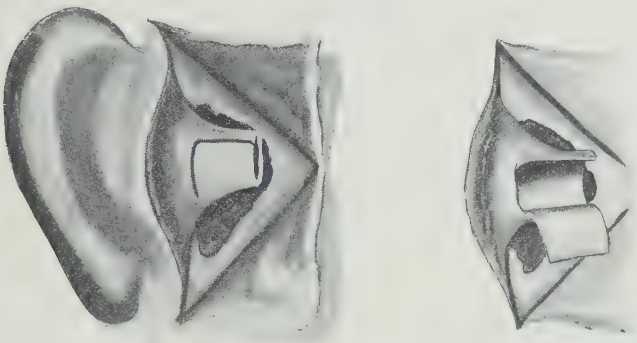
Fig. 461

The Jansen-Stacke plastic incision. This flap should be used when the sigmoid sinus and jugular bulb are exposed. The flap is turned downward and backward and thus covers these areas.

After-treatment.—The primary dressing is identical with that for acute mastoiditis, with the single exception that the spiral tube and gauze are inserted through the enlarged meatal opening in the concha (Fig. 457) instead of through the postauricular wound. The distal end of the tube is placed into the deepest portion of the mastoid wound.

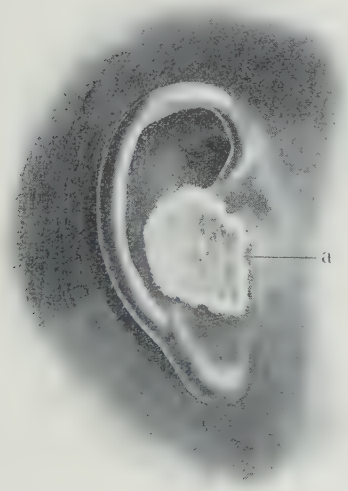
This should be removed on the fifth day, or earlier if the temperature persistently remains above 102° F., or if severe pain develops and persists. The wound should be mopped dry with a cotton-wound appli-

Fig. 462



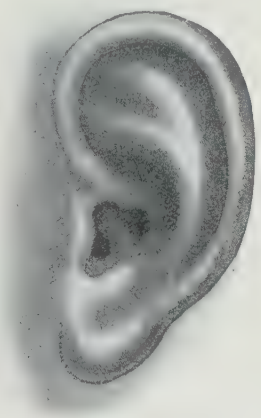
Showing the method of making the Jansen modification of the Stacke plastic flap of the skin meatus. The inferior large flap should be reflected into the lower portion of the mastoid wound and held in place by anchor stitches. The upper short flap should be reflected into the upper portion of the mastoid wound and held in place by an anchor stitch.

Fig. 463



A collodion dressing used in the after-treatment of operative mastoiditis. A loose wick of gauze is inserted into the mastoid wound through the external meatus and covered with a film of cotton, which is then saturated with an ether solution of collodion to seal it.

Fig. 464



The appearance of the concha and the external auditory meatus, after healing is complete.

cator, inspected for exuberant granulations, and a fresh sterilized tube and gauze inserted. If exuberant granulations are present, they should be reduced by painting them with 95 per cent. carbolic acid, and, after the lapse of one minute, with alcohol, to check the action of the acid. This method of treatment should be continued daily for ten days after the operation. After this the tube may be abandoned and a small wick of gauze inserted into the wound at its most dependent portion and extended to the concha. Small gauze pads should be placed in the concha of the auricle to catch the secretions drawn out by the gauze wick. Large pads are placed over the auricle and mastoid region and secured with the fan-shaped bandage (Fig. 465). After the tenth day the large gauze pad and bandage may be omitted and the dressing applied in the cavity of the auricle instead. This should be secured by placing a thin film of cotton over it (Fig. 463) and painting it with an ethereal solution of collodion (Pierce). The mastoid wound usually becomes covered with squamous epithelium in from three weeks to two months, though the process may cover a longer period of time. Various factors may cause a prolongation of the period of repair, chief of which are suppurative inflammation of the epipharynx, ethmoiditis, sphenoiditis, and an infection of the Eustachian tube. Certain constitutional dyscrasias, as syphilis, tuberculosis, and struma, may also lower the vitality of the tissues and prolong the reparative process.

Fig. 465

Method of applying a bandage over the ear and mastoid process.

The disfigurement following the Ballance plastic meatal flaps is slight (Fig. 464). It should be said, however, that chondritis of the auricle with marked shrinkage and deformity may follow any of the plastic operations which include the cartilage of the concha. Every effort should be made to prevent the infection of the wound either during or after the operation. The edges of the conchal wound should be touched with carbolic acid to seal up the lymph spaces.

Author's Modified Radical Operation.—This operation may be called a modified radical mastoid operation, though it does not include the exposure of the middle ear. It does, however, include the plastic meatal flaps and the removal of the posterior bony wall of the meatus down to the annulus tympanicus. The postauricular wound is closed as in the radical operation, and the dressings are applied through the conchomeatal wound.

The advantages claimed for this operation over the radical operation in chronic mastoiditis are: (*a*) The preservation of the function of the middle-ear contents, and of the membrana tympani; (*b*) an improve-

ment in the hearing, whereas in the radical operation the hearing is either unchanged or impaired; (*c*) the closure of the perforation in the membrana tympani which often takes place after the necrosis and granulations have disappeared; (*d*) the drainage of the secretions from the antrum and mastoid cells into the auditory meatus through the opening in the posterior wall of the meatus, thus relieving the Eustachian tube of the excess of secretions.

The principle upon which the operation is based is that *if ample drainage is provided the infectious process tends to subside and the diseased tissue to heal.* The removal of the posterior wall of the bony auditory meatus and the retraction of the plastic meatal skin flaps into the mastoid wound provide for the drainage of the mastoid antrum and cells, and thus remove the stress from the Eustachian tube. The Eustachian tube, being relieved, is usually ample to drain the cavum tympani, even when chronically infected. As a result, the resistance of the diseased membrane, periosteum, and bone is increased, and the infection gradually subsides. The mucous membrane, periosteum, and bone become healthy and "heal out."

The removal of the fragments of the malleus and incus often disturbs the relation of the stapes to the fenestra vestibuli (oval window), and thus impairs the hearing; that is, the stapedius muscle pulls the stapes backward and displaces the foot-plate of the stapes in the window. This could be obviated in the radical operation by severing the tendon of the stapedius muscle.

Technique.—(*a*) Prepare the patient as for the simple and radical mastoid operations. Extend the skin incision well forward above the auricle as in the radical operation, as this allows the external bony meatus and drumhead to be clearly seen during the operation.

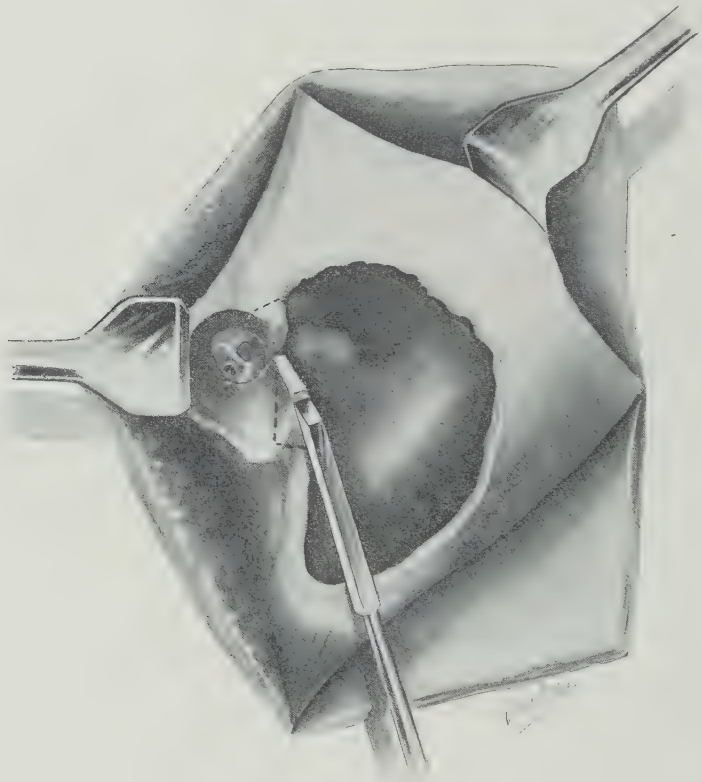
(*b*) Expose the mastoid antrum and cells as in the radical operation.

(*c*) Remove the posterior bony wall of the auditory meatus down to the annulus tympanicus, as shown in Fig. 466. At no time during the operation should the membrana tympani and the ossicles of the cavum tympani be injured by probing or other instrumental procedure. The introduction of a probe into the meatus to determine its depth and direction, as recommended in the radical operation, should be studiously avoided. If this precaution is not observed, the ossicles may be dislocated and the hearing impaired. The posterior wall of the meatus should be removed as widely as possible to provide free drainage and access to the exenterated antrum and cells through the auditory meatus during the after-treatment. It is sometimes necessary to remove some bone from the outer portion of the superior wall of the meatus to fully expose the drumhead to view. Enough should be removed to fully expose the membrana tympani to inspection after the auricle is replaced and sutured in position. The proper prosecution of the after-treatment will largely depend upon the completeness with which this step of the operation is carried out.

(*d*) The plastic meatal flaps should now be formed as in the radical operation. The operator's individual preference may be used, though it

is essential that the skin of the concha be included in the flaps, so as to enlarge the meatal opening and facilitate the application of the dressings to the mastoid wound. This procedure also aids in the inspection of the membrana tympani. The author has found the Ballance incision the most satisfactory for this purpose. The reader is referred to Figs. 450 to 462 for the details of the various plastic meatal flaps, with the suggestion that in applying them to this operation, they should be so utilized as not to obstruct the opening made by the removal of the posterior bony wall of the auditory meatus.

Fig. 466



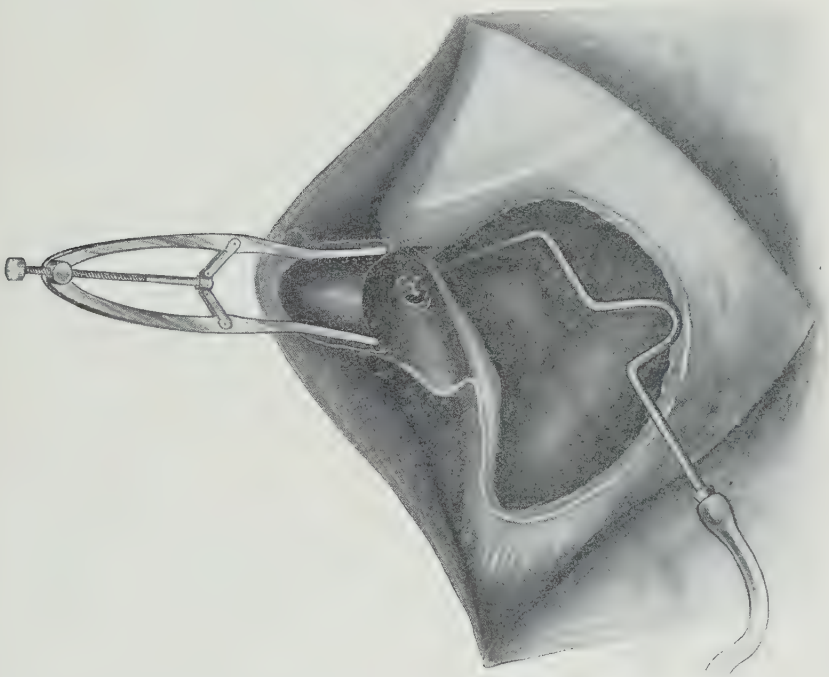
The removal of the posterior wall of the external auditory meatus down to the annulus tympanicus in the meatomastoid operation. Dotted lines indicate the amount to be removed.

(*e*) Retract the meatal plastic skin flaps with the author's retractor to bring the membrana tympani into view, as shown in Fig. 472. This will greatly facilitate the next step in the operation, as it is necessary to see the membrana tympani during its performance. If the meatal retractor is not used the meatal flaps will constantly obstruct the view and hinder the operator in his work.

(*f*) Insert a cannula, as recommended by Heath, into the aditus ad antrum *via* the antrum (Figs. 467 and 468), and with an attached rubber bulb, send blasts of air into the cavum tympani. The secretions and pedunculated granulations within the middle-ear cavity are blown out through the perforation in the membrana tympani into the auditory meatus. The middle ear may also be irrigated with the same apparatus.

(*g*) If granulations or polypi are thus blown through the perforation, they should be grasped by small dressing forceps and removed. If they appear at the perforation, but do not protrude through it, they may be removed by gently pressing the forceps blades (one on either side of the

FIG. 467



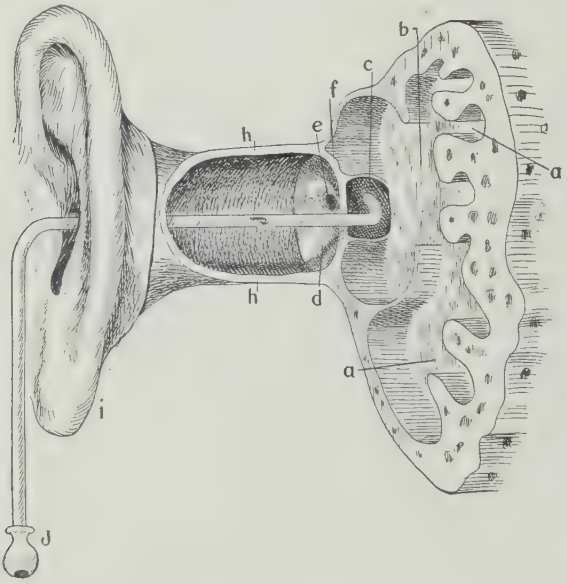
Author's modified Heath operation (bony portion) complete. The curved cannula is inserted into the aditus ad antrum, preparatory to blowing blasts of air through the cavum tympani, to remove the secretions and debris. The author's meatus retractor makes the view of the membrana tympani possible during this procedure.

perforation) against the margins of the perforation, thus bringing them within the grasp of the forceps. The blasts of air should be repeated until all the secretions, polypi, and debris are expelled from the tympanic cavity. Tubes of various sizes should be at hand, so that one may be selected that fits the aditus ad antrum. It may be necessary to modify the shape of the antral aspect of the aditus with a small curette or hand burr, to adapt it to the cannula (Heath). If the tube is too small, it may pass so far into the aditus as to dislocate the incus and thus impair the hearing.

(*h*) Having removed the secretions, polypi, and debris from the tympanic cavity with the air blasts and forceps, place a small wet pad of cotton over the perforation in the membrana tympani, and a small plug of the same material in the antral end of the aditus ad antrum to keep the blood and bone chips from entering the middle ear.

(*i*) Anchor the plastic meatal flaps, as in the radical mastoid operation, with suitable stitches (Figs. 454 to 457).

FIG. 468



Schema of the ear, showing the method of cleansing the tympanic cavity after the Heath operation: *a a*, mastoid cells; *b*, antrum; *c*, aditus ad antrum; *d*, membrana tympani; *e*, perforation in the membrana tympani; *f*, annulus tympanicus; *h*, external meatus, the posterior wall of which is removed; *i*, the auricle; *j*, silver cannula introduced through the opening in the posterior opening in the meatus, and thence forward into the aditus ad antrum; *c*, air pressure applied with a rubber bulb forces the secretions, granulations, etc., from the tympanic cavity through the perforation (*e*) in the membrana tympani into the meatus.

(*k*) Introduce the tube dressing (Fig. 457) through the auditory meatus into the mastoid wound. Do not place it against the membrana tympani, but pass it backward through the opening in the posterior wall of the meatus into the mastoid cavity. If other forms of dressing are preferred, they should be introduced in the same manner. Whatever dressing is employed, it should be loosely placed, not packed, as its primary purpose is to facilitate drainage. Some operators recommend that gauze be firmly packed into the mastoid wound to "keep down" the granulations. If the operation is thoroughly done under aseptic conditions, exuberant granulations will not form; furthermore, good drainage lessens the tendency to their growth. Exuberant granulations are the product of infection, whereas healthy granulation tissue is formed in the

process of repair. Many cases pursue a prolonged process of repair because the dressings are packed in the mastoid wound. If the surgeon grasps the purpose of the wound dressing, namely, to promote drainage (and this alone), he will only insert enough gauze to carry away the secretions. The author uses a one-half to one inch strip of gauze in the rubber tube for this purpose and finds it adequate. If the foregoing technique is observed, exuberant granulations will not form nor will the healing process be prolonged.

The ear should be covered with several large gauze pads, which should be removed in three days, the wound gently dried with a cotton-wound applicator introduced through the auditory meatus, and a new tube dressing applied. This should be changed daily. The sutures should be removed on the fifth day.

The membrana tympani should be inspected daily, especially when the blasts of air are forced through the aditus ad antrum. After the mastoid wound is cleansed with the cotton-wound applicator the curved cannula should be introduced into the aditus *via* the meatus and the opening in the posterior wall of the meatus (Figs. 467 and 468) and blasts of air forced through the tympanic cavity to clear it of secretions and granulations. All granulations or polypi appearing at the perforation in the membrana tympani should be removed with forceps or with caustics. Heath insists upon the value of the blasts of air through the tympanic cavity until the aditus ad antrum becomes closed (eight to fourteen days). The author has followed his method and finds it to be of great value in the after-treatment. By it large quantities of mucus and pus are forced into the external meatus, from which they may be removed with a cotton-wound applicator. The secretions may also be removed by inflation through the Eustachian tube, though this is not as efficacious as Heath's method.

The secretions and granulations from the middle ear gradually subside as the perforation closes. The mastoid cavity becomes lined with epidermis and remains a dry cavity, and the Eustachian tube is no longer burdened with the secretions from this source.

Of the forty-five cases thus operated by the author, in one, complicated by an epidural abscess over the tegmen tympani, it was necessary to convert into a radical operation. The membrana tympani re-formed in twenty-six cases, and the hearing returned to almost the normal in all but one. In this method of operation the mastoid wound is almost filled in the process of repair.

Thiersch Grafts in the Mastoid Wound.—To Reinhard, Jansen, and Ballance belong the credit of applying the Thiersch grafts to the mastoid wound. Ballance has, perhaps, used it more constantly and frequently than anyone else, and his technique is generally followed. Personally the author has had but rare occasion to use it, as his cases usually became covered with epidermis in as short a time as is claimed by Ballance after the use of the Thiersch grafts. In only two cases has it been necessary to apply the grafts, and in these they were successfully applied after sec-

ondary operations. By using the Ballance plastic meatal skin flaps, and fixing them as in Fig. 459, the author's cases have, with rare exceptions, healed with epidermis over the walls of the mastoid wound in from three to ten weeks, rarely longer. This good showing is due to several factors, chief among which are: (*a*) The Ballance plastic meatal flaps applied after the author's method. (*b*) The use of the spiral rubber tubing, with a small wick of gauze in its lumen as the sole drainage dressing. This dressing, as already explained, provides good drainage, which establishes conditions discouraging the formation of unhealthy granulations. (*c*) Another cause of the rapid epidermization of the mastoid wound is the complete exposure and exenteration of the mastoid

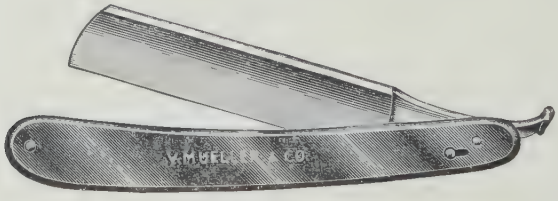
FIG. 469

Hajek's hand burr.

antrum and cells. The cells of Kirschner, between the antrum and meatus, and those in the posterior root of the zygoma and in the posterior wall of the pyramid of the petrous portion of the temporal bone are likewise carefully sought for, and if present removed.

If the surgeon finds that a considerable number of his cases pursue a prolonged course of healing, he should carefully scrutinize his technique, and, if found to be faulty at any point, improve it accordingly. If his cases still refuse to heal properly he may try the Thiersch grafts.

FIG. 470



Thiersch's graft razor.

Technique.—(*a*) The grafts may be applied at the close of the primary operation, ten days after the primary operation, or after a secondary operation. Dench applies the grafts at the close of the primary operation. Ballance ten days after the primary operation. The author only after a secondary operation; that is, only after it is conclusively shown that repair will not follow the primary operation. Since adopting the technique described in the radical mastoid operation, the author has not had more than 1 per cent. of cases requiring a secondary operation, whereas in his earlier practice it was about 10 per cent.

(*b*) The patient's arm or thigh should be shaved and scrubbed twenty-four hours before grafting, a moist carbolized dressing applied, and held in position with a bandage.

(*e*) The patient should be anesthetized for the reason that (1) it prevents the "goose-flesh" contraction of the skin, which so materially interferes with cutting thin Thiersch grafts, and (2) it also prevents the pain incident to securing the grafts and opening the wound for their application. If the grafting is done at the time of the primary operation, the patient is already anesthetized and the arm or thigh prepared when the mastoid region was shaved.

(*d*) Rescrub the skin after the bandage and dressing are removed.

(*e*) With the skin moistened with normal salt solution and drawn tight between the forefinger and thumb, remove the thin cortex by a rapid sawing motion with the broad Thiersch razor (Fig. 470). The razor is flat upon one side, while the other (the upper) is concave. Normal salt solution should be dropped into the concave surface of the razor to float the graft. The size of the graft should be about 2 x 3 cm., or large enough to cover the entire bony wound.

FIG. 471

Thiersch's graft spatula.

(*f*) Float the graft from the razor blade to the large spatula (Fig. 471), using a teasing needle (Fig. 472) in transferring it.

(*g*) The mastoid wound, having been previously opened and freed of all blood and oozing, is made the repository of the graft. With a teasing needle (Fig. 472) the edge of the graft is transfixed to the border of the mastoid wound and the spatula gradually withdrawn. The graft is thus deposited smoothly and evenly over the surface of the wound. If necessary, other grafts are applied.

FIG. 472

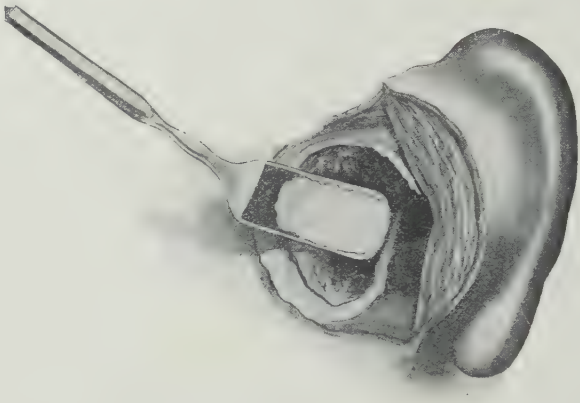
Teasing needle for Thiersch's grafting.

(*h*) The grafts should be pressed against the walls of the wound with a small blunt instrument until they are closely adherent to the uneven surface (Fig. 474). A small glass pipette or medicine dropper may be used to withdraw bubbles of air from beneath the grafts. Some operators prefer to first fill the mastoid cavity with normal salt solution and float the graft upon its surface. The fluid is then gradually withdrawn with a pipette until the graft rests upon the surface of the bony wound. It is not necessary to engraft the entire surface of the wound, as the interspaces soon become covered by extension from the edges of the grafts.

(*i*) Ballance formerly covered the grafts with very thin gold-foil to prevent the small cotton pads adhering to them and dislodging them

when the dressing was removed. He now applies the cotton balls directly to the grafts, with good success. As a matter of fact, the grafts will remain in position, if properly adjusted (evenly and closely applied),

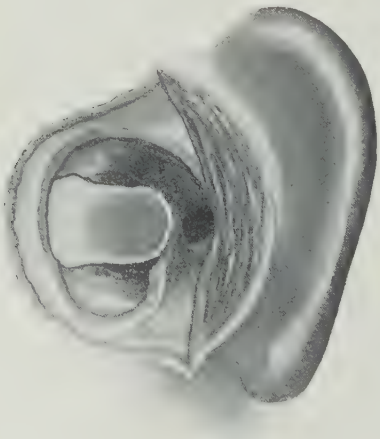
FIG. 473



The Thiersch graft being applied to the mastoid wound.

without either gold-foil or the gauze pads. The postauricular wound should be reclosed with sutures after the grafts are applied and the subsequent dressings applied through the enlarged auditory meatus.

FIG. 474



The Thiersch graft in position. Other grafts are introduced until the entire bony surface is covered.

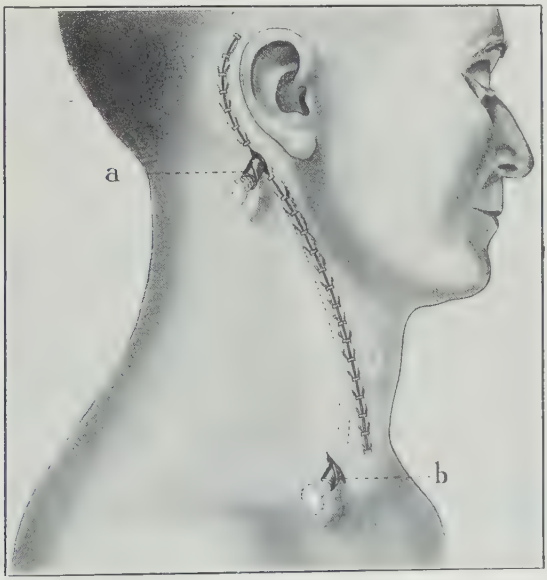
(*j*) The small cotton balls are used to hold the grafts in apposition to the granulating bony wound, and they should be removed on the third day. Portions of the grafts will not "take" or grow, hence necrosis

occurs, giving rise to a horrible stench. The engrafted area should be gently mopped dry with a cotton-wound applicator, the necrosed particles

FIG. 475

Mastoid incision made in infants: *a*, *a*, the proper location of the incision; the lower end of the incision should be about one-half inch posterior to its position in adults, in order to avoid injuring the facial nerve at its exit from the mastoid bone at *b*.

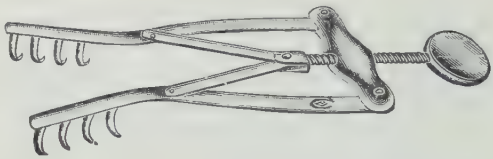
FIG. 476



Bezold's mastoiditis. The wound is closed with Michel's metal clamps. *a*, spiral tube draining the mastoid wound; *b*, spiral tube draining the abscess of the anterior triangle of the neck. An accessory incision is used to drain the abscess, as this will heal quickly after the tube is removed. If the tube makes its exit at the lower portion of the primary incision, healing will be slow and a scar left, as this is in the infected field. The portion of the incision below the mastoid also represents the incision for the excision of the external jugular vein and for the removal of the glands of the neck.

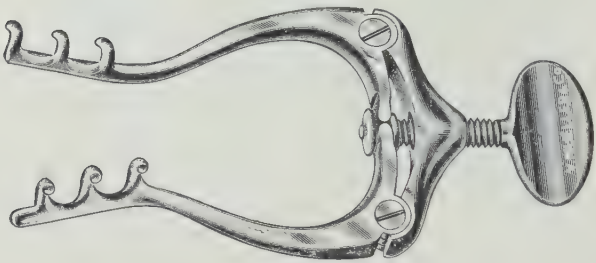
removed, and a fresh dressing applied. The dressing should be renewed daily, as after the mastoid operation.

Fig. 477



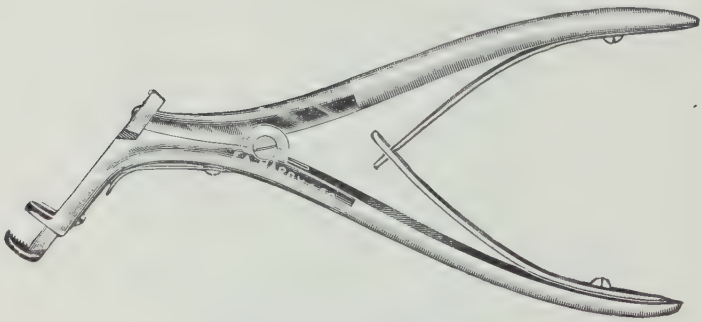
Allport's mastoid retractor.

Fig. 478



Jansen's mastoid retractor.

Fig. 479



Allport's bone-crushing forceps.

Fig. 480

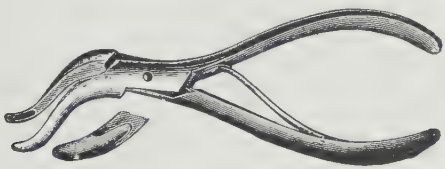
McKernon's rongeur forceps.

It should be borne in mind, however, that Thiersch grafts will rarely be necessary if the cutaneous portion of the external auditory meatus is

properly and intelligently utilized to line the mastoid wound, and if the cells are completely exenterated.

The Mastoid Operation in Infants and Young Children.—As the mastoid tip and cells are but slightly developed before the age of puberty, the technique of the mastoid operation should be somewhat modified. The rudimentary tip of the mastoid process is located much higher and more posteriorly than in adults.

FIG. 481



Jansen's rongeur forceps.

FIG. 482

Reverdin's needle.

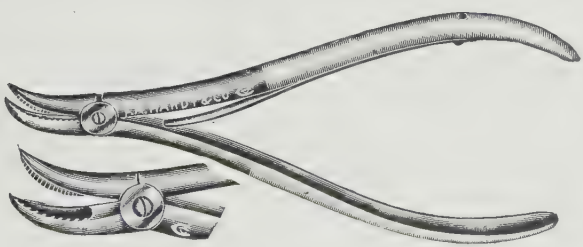
FIG. 483

Scheibel's suture forceps.

FIG. 484

Michel's metal clamp suture.

FIG. 485



Michel's suture clip forceps.

The postauricular incision should, therefore, begin higher and more posteriorly, as shown in Fig. 475. Furthermore, the facial nerve makes its exit from the styloid foramen quite near the surface of the mastoid, and, if the incision is made as in adults, it may be injured and cause facial paralysis. The mastoid antrum is almost or fully developed at birth, and is often the only portion of the mastoid bone involved.

The Surgical Treatment of Bezold's Mastoiditis.—The early surgical treatment is the only procedure that is applicable in this affection. The usual mastoid incision is made with an extension downward beyond

the tip of the mastoid, parallel with the anterior border of the sternomastoid muscle to the lowest portion of the brawny swelling of the neck. The aponeurosis of the sternomastoid muscle is divided and retracted. The mastoid is opened from below upward, toward the antrum. All the mastoid cells are thoroughly curetted until the perforation in its *inner plate* is located. The perforation is followed into the loose tissues of the neck, and the granulations removed with a dull curette. The rough projections of bone are smoothed with a burr or curette and the ragged edges of the muscles are trimmed off with scissors. If the abscess has burrowed into the neck anteriorly or posteriorly, it is necessary to lay it wide open and thoroughly remove all diseased tissue with a curette. The mastoid portion of the incision should then be closed over a spiral tube with gauze in its lumen, the distal end of which is placed in the mastoid wound (Fig. 476). If the abscess extends into the neck, the incision should be closed over another spiral rubber tube, which is allowed to drain through a separate incision back of the lower end of the neck incision, as shown in Fig. 476.

The dangers attending this operation are the wounding of the facial nerve at its exit from the bony canal in the mastoid process, and the spinal accessory nerve going to the trapezius muscle. If this nerve is wounded the shoulder will droop. The lateral sinus is also in close proximity to the perforation, hence great care should be taken in operating in this region.

If the disease is recognized early and prompt and thorough surgical measures are instituted the prognosis is fair, although the recovery may extend over several weeks, as the healing of the wound after such an extensive operation requires considerable time, and not infrequently a secondary abscess forms in the neck because of poor drainage.

CHAPTER XLIX

THE LABYRINTH: ITS PHYSIOLOGY, FUNCTIONAL TESTS AND DISEASE

General Considerations.—Dr. Geo. E. Shambaugh has formulated some of the fundamental problems in reference to nystagmus as follows:

(*a*) Normally the voluntary muscles of the eyes, body, and extremities are under the influence of tonus impulses from the labyrinth.

(*b*) The impulses from the two labyrinths are equal, though antagonistic, and a state of equilibrium is maintained.

(*c*) The *sudden cessation* of the tonus impulses from one labyrinth disturbs the equilibrium, and a spontaneous nystagmus, vertigo, nausea and vomiting, and ataxia result; that is, signs of destruction disharmony occur. In a slow destruction of one labyrinth extralabyrinthine tonus develops as fast as the destruction of the labyrinth tonus occurs, hence the signs of destruction disharmony are absent.

(*d*) After the sudden destruction of one labyrinth, compensation takes place after a shorter or longer period of time.

Physiology of the Semicircular Canals.—1. The hair cells on one side of each crista are stimulated by an endolymph current in one direction, and the hair cells on the opposite side of the crista are stimulated by a current in the opposite direction (Plates XX and XXI).

2. The hair cells on one side of a crista, when stimulated, produce nystagmus in the plane of its canal, and directed toward one side, while the stimulation of the hair cells on the opposite side of the same crista produce nystagmus in the same plane, but directed to the opposite side. The signs of stimulation disharmony occur in each instance.

3. The reactions following the stimulation of the hair cells on the two sides of each crista are unequal and are in about the relation of 2 to 1. This is well illustrated in Plate XXV in which the right labyrinth is totally destroyed. The patient is represented as being turned ten times to the right, with a resulting after-nystagmus of 12 seconds' duration; after turning ten times to the left there is an after-nystagmus of six seconds' duration. This is due to the normal physiological difference in the potentiality of the two halves of the crista stimulated by the respective turnings.

4. In each canal the greater reaction follows the stimulation of these hair cells, impulses from which direct the nystagmus toward the same side (canal half in horizontal and utricular half in superior and posterior canals, Plates XX and XXI).

5. Tonus impulses from the labyrinth have their origin in the hair cells of the cristæ. From each labyrinth, therefore, arise tonus impulses

for the muscles which direct nystagmus to the same side, as well as nystagmus to the opposite side. The stronger tonus impulses from the labyrinth are those which go to the muscles directing the nystagmus to the same side. When the tonus impulses from one labyrinth are suddenly suppressed, as in diffuse manifest suppurative labyrinthitis, the equilibrium between the two labyrinths is disturbed, and the tonus from the normal labyrinth, acting without the restraint of the impulses from the opposite side, produce nystagmus directed toward the normal or opposite side (Plate XXV). (Signs of destruction disharmony.)

Compensation in sudden destruction of the labyrinth occurs (1) by extralabyrinthine compensatory increase in tonus, and (2) by compensatory increase in tonus of the opposite or normal labyrinth.

In some cases compensation may take place entirely independent of the healthy labyrinth, that is, it may be entirely extralabyrinthine.

In very old cases of unilateral destruction it is possible that the two halves of each crista of the remaining healthy labyrinth become equal in tonus impulses, as is suggested by the fact that the rotation in either direction produces nystagmus of equal intensity and duration (Plate XXVIII).

In sudden destruction of one labyrinth there is not only the loss of labyrinthine tonus from this side, but there is a suppression of extralabyrinthine tonus. Monakow calls this diaschisis. The rapid recovery from the disturbed equilibrium (nystagmus, nausea, vomiting, dizziness, ataxic gait, etc.) is due to the restoration of the extralabyrinthine tonus, *i. e.*, the diaschisis rapidly subsides. The establishment of compensatory tonus in the remaining labyrinth is established much more slowly, often requiring years. Indeed, much of the compensatory tonus, other than that which occurs soon after the destruction, is extralabyrinthine in origin, as is shown by the fact that for a long time after compensation under ordinary conditions seems perfect, turning in one direction will produce a longer and more severe nystagmus than turning in the opposite direction. This shows that labyrinthine compensation is not complete (Plate XXV). Those cases of very long standing, as shown by Ruttin, had nystagmus of equal duration and intensity by turning in either direction, though of shorter duration than normal (Plate XXVIII).

After rapid total destruction of one labyrinth the process of recovery of static function is about as follows:

(*a*) The diaschisis or suppression of extralabyrinthine tonus quickly disappears, and the symptoms or signs of destruction disharmony are correspondingly relieved.

(*b*) Compensatory extralabyrinthine tonus gradually develops, though perhaps more rapidly than the compensatory labyrinthine tonus in the healthy labyrinth.

(*c*) Compensatory labyrinthine tonus also develops, until after several years the impulses from two sides of the crista of the healthy labyrinth become equal (but of shorter duration than normal), a fact which

makes it appear probable that the extralabyrinthine compensatory tonus has subsided to its normal plane again.

All afferent impulses affect the static centres of the cerebellum, though those from the labyrinth are probably more defined and effective, as this is a highly specialized organ of special sense.

A sharp distinction should be drawn between induced nystagmus and spontaneous nystagmus. Induced nystagmus is due to an excessive artificial stimulation of a crista ampullaris, and is attended by induced "signs of stimulation disharmony," as a preponderance of nervous impulses emanates from the stimulated labyrinth (Plates XIX and XX). In circumscribed labyrinthitis the nystagmus is spontaneous and may be directed to either side. In diffuse suppurative manifest and in diffuse suppurative latent labyrinthitis and in diffuse serous labyrinthitis, the spontaneous nystagmus is due to the sudden, and total or partial, suppression of nervous impulses from the diseased labyrinth. We should expect, therefore, in diffuse labyrinth disease, to find the "signs of destruction disharmony," while in circumscribed labyrinthitis we may find either the signs of stimulation or of destruction disharmony, or both (Plates XXI, XXII, and XXVIII). When both ears are normal spontaneous nystagmus is absent, but it may be induced.

By reference to Plate XIX we are enabled to explain the various nystagmic phenomena which occur upon artificial stimulation of the crista of the superior canal. That is, this plate illustrates the mechanism of the induced nystagmus (signs of "stimulation disharmony"), by the various physiological tests.

Anatomical Data.—Certain anatomical facts to be taken into consideration in arriving at correct physiological and clinical conclusions are illustrated in Plate XIX as follows:

1. The utricular half of each crista ampullaris of the superior canal emits stronger tonus impulses to Deiters' nucleus than the canal half, and as a consequence the utricular half of the crista exerts more pull or potentiality than the canal half. The ratio is about 2 to 1 (Plate XXV).

2. The right Deiters' nucleus (*D.N.*) sends fibers to the abductors and the adductors of both eyes, but it sends stronger impulses to the abductor of the left eye, and to the adductor of the right eye than it does to the opposing muscles, *i. e.*, the abductor of the right and the adductor of the left; hence, when the right crista ampullaris is stimulated there is a preponderence of pull or potentiality exerted upon the abductor of the left eye, and the adductor of the right eye, with a resultant conjugate movement of both eyes to the left, the slow component of the nystagmus. This movement of the eyes stimulates a nervous impulse in the left cortical centre (*L.C.C.*) which is transmitted to the nuclei, III and VI of the right side, and from thence to the abductor muscles of the right eye, and the adductor muscle of the left eye (Plate XIX). This results in a conjugate movement of both eyes to the right. This movement is more rapid, though of the same amplitude, than the primary slow movement, and is known as the quick component of the

nystagmus. In other words, when there is an excessive stimulus or excess of tonus in the right crista ampullaris, a slow conjugate movement of the eyes to the left occurs, which excites a reflex impulse in the cortical centre (*R.C.C.*) which immediately produces a quick conjugate movement of the eyes in the opposite direction. These two movements constitute nystagmus of vestibular and cerebellar origin. The nystagmus takes its name from the direction of the quick component, hence, when the quick component is to the right the nystagmus is said to be to the right, and *vice versa*. When there is a destruction or inhibition of one labyrinth, from disease, the nervous impulses emanating from the sound labyrinth preponderate over those from the destroyed or inhibited labyrinth and produce spontaneous nystagmus in the opposite direction to that which would be induced by stimulation, provided the destroyed labyrinth could be stimulated. When testing the vestibular apparatus the "signs of stimulation disharmony" are induced, whereas, in acute disease of the labyrinth the "signs of destruction disharmony" are spontaneously manifested. When the acute disease becomes latent (without the signs of destruction disharmony) there are no symptoms referable to the vestibular labyrinth, except upon experimental tests of the labyrinth.

3. The crista ampullaris of each semicircular canal is an end-organ of the vestibular nerve, and plays an important part in maintaining the equilibrium of the body under normal conditions. In labyrinth disease, and under excessive induced stimulations, a disturbed state of equilibrium, and nystagmic movements of the eyes, known as the "signs of destruction disharmony," and the "signs of stimulation disharmony," respectively, are present.

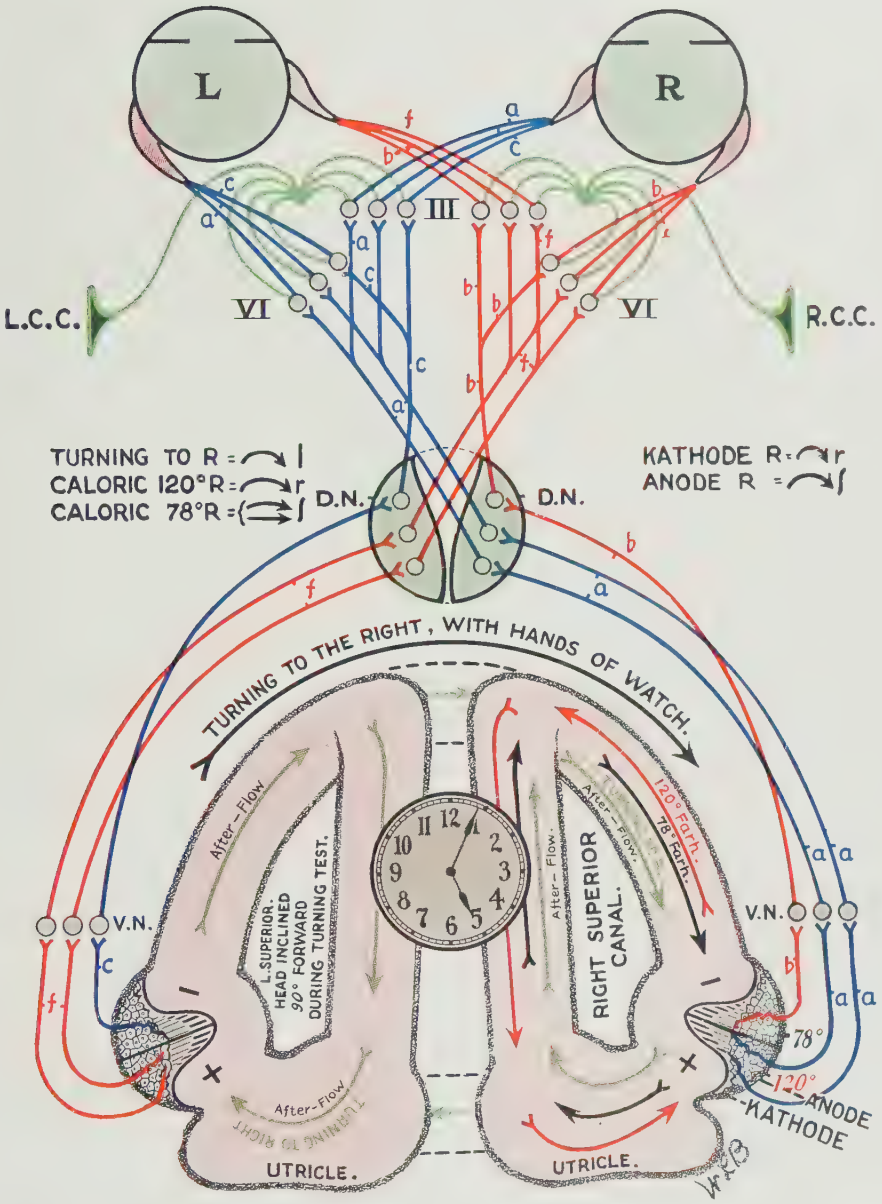
4. The canal side, and the utricular side of the crista are not endowed with the same degree of pull or potentiality, and each acts upon opposing sets of voluntary eye muscles (as well as those of the body). That is, when the utricular side of the crista ampullaris (+) of a superior canal is stimulated (Plate XIX) a nystagmus of a greater degree is produced than when the canal side (−) is stimulated, and produces a

PLATE XIX

Diagrammatic Illustration of the Turning, Caloric, and Galvanic (Induced) Nystagmus.

1. The green arrows indicate the flow of endolymph after turnings to the right have ceased. The head is inclined 90 degrees forward during the turnings. In the right superior canal the impact of the endolymph is against the canal half of the crista ampullaris (—), which gives off a nervous impulse about one-half as strong as is given from the utricular half. In the left superior canal the impact of the endolymph is against the utricular half of the crista, which in consequence, gives off a nervous impulse about twice as strong as is given off from the canal half of the crista. Both impulses are transmitted *via* the red lines (*b* and *f*) to Deiters' nuclei (*D.N.* and *D.N.*) and from thence to the third and sixth nuclei of the right side (*III* and *VI*) to the abductor muscles of the right eye (*R*), and to the adductor muscles of the left eye (*L*). The strength of the impulse arising in the canal half of the right crista and in the utricular half of the left crista is in the ratio of 1 to 2, and they conjointly cause the eyes to turn slowly to the right (slow component of the nystagmus). A corrective impulse is immediately excited in the cortical centre

PLATE XIX



Turning, Caloric, and Galvanic Induced Physiological Nystagmus.

of the left hemisphere of the brain (*L.C.C.*) which is transmitted to the third and sixth nuclei of the left side (*III* and *VI*), and from thence to the abductor muscles of the left eye (*L*), and to the adductor muscles of the right eye (*R*). As a result of this corrective impulse the eyes are quickly turned to the left (quick component of the nystagmus). This reaction is known as induced rotatory after-nystagmus to the left, and is symbolized thus ↷ l. It is referred to as induced because it is artificially produced by unaccustomed turnings, and it is rotatory because it emanates from the superior canals. The plane of the nystagmus is, according to Fleurens' law, always in the plane of the canals stimulated. The plane of the superior canals is frontal, hence the eyes rotate upon their pupillary axes.

2. The black arrows indicate the direction of the downward flow of endolymph in the right superior canal after irrigation of the right ear with cold water (78°) with the head erect. The impact of the endolymph is against the canal half of the crista. This gives rise to a nervous impulse which is transmitted through the path shown as a red line (*b*) to Deiters' nucleus (*D.N.*), and from thence to the third and sixth oculomotor nuclei of the right side (*III* and *VI*), whence it is conveyed to the abductor muscles of the right eye (*R*) and to the adductor muscles of the left eye, thus inducing a slow conjugate movement of both eyes to the right (slow component of nystagmus). A corrective impulse is thereby stimulated in the left cortical centre (*L.C.C.*) which is transmitted to the third and sixth oculomotor nuclei of the left side, from whence it is conveyed to the adductor muscles of the right eye (*R*) and to the abductor muscles of the left eye, thereby causing a quick rotatory movement of both eyes to the left (quick component of the nystagmus), which is symbolized thus, ↷ l. Cold irrigation (78°) therefore induces rotatory nystagmus to the opposite side.

3. When warm irrigation (120°) is used the flow of endolymph is upward (the reverse of what it was with cold irrigation 78°), hence the impact of the endolymph, as indicated by the red arrows, is against the utricular half of the crista (+), which sends a nervous impulse through the paths indicated by the blue lines (*a*, *a*) to Deiters' nucleus (*D.N.*). From thence it is transmitted to the third and sixth (*III*, *VI*) oculomotor nuclei of the opposite (left) side, and thence to the adductor muscles of the right eye (*R*) and to the abductor muscles of the left eye, thereby causing a conjugate slow movement of both eyes to the left (slow component). A cortical correction impulse immediately arises in the right cortical centre (*R.C.C.*), and acting through the third and sixth oculomotor nuclei of the right side, produces a quick conjugate movement of both eyes to the right (quick component of the nystagmus). The nystagmus is induced, rotatory and to the right, and is symbolized thus: ↷ r. Warm irrigation (120°) induces rotatory nystagmus to the same side.

4. When the kathode or negative pole of a galvanic battery is applied to the right ear a nervous impulse is transmitted through the paths indicated by the blue lines (*a*, *a*) to Deiters' nucleus (*D.N.*), and thence to the third and sixth oculomotor nuclei (*III*, *VI*) of the left side, and thence to the adductor muscles of the right eye (*R*) and the abductor muscles of the left eye (*L*), thereby inducing a slow conjugate movement of both eyes to the left (slow component). The cortical corrective impulse is immediately sent out from the right cortical centre (*R.C.C.*) to the third and sixth oculomotor centres of the right side (*III*, *VI*), and from thence to the abductor muscles of the right eye (*R*) and the adductor muscles of the left eye (*L*), thereby inducing a quick conjugate rotatory movement of both eyes to the right (quick component). The kathodal (negative) galvanic current therefore induces rotatory nystagmus to the same (right) side, and is symbolized thus: ↷ r.

5. When the anode or positive pole of the galvanic battery is applied to the right ear, nystagmus to the opposite side is induced. The anode appears to inhibit or suppress the nervous impulses in the vestibular apparatus over which it is applied, thus leaving a preponderance of tonus in the opposite labyrinth. The greater impulse or tonus is therefore transmitted through the paths indicated by the red lines (*f*) of the left side, which pass through the left Deiters' nucleus to the right oculomotor nuclei (*III*, *VI*) to the abductor muscles of the right eye and the adductor muscles of the left eye, thereby producing a slow conjugate movement of both eyes to the right (slow component). The cortical correction impulse immediately arises in the left cortical centre (*L.C.C.*) and is transmitted through the left oculomotor centres (*III*, *VI*) to the adductor muscles of the right eye, and abductor muscles of the left eye, thereby producing a quick conjugate movement of both eyes to the left (quick component of the nystagmus). The anode applied to the right ear (the kathode in the hand) induces rotatory nystagmus to the opposite (left) side, and is symbolized thus: ↷ l.

movement of the eyes in an opposite direction to that produced by stimulation of the other half of the crista. In the horizontal canal the stronger nystagmus is produced by stimulation of the canal side of the crista, and here again, the stimulation of each half of the crista produces movements of the eyes in a direction opposite to that produced by the other half of the same crista.

5. Each crista ampullaris forms a ridge across its respective ampulla, and is surmounted by hair cells, the hairs of which project into the under surface of the gelatinous cupola. The cupola with its hair cells is a special end-organ of the vestibular nerve, and receives the impact of the endolymph current. Dr. Shambaugh claïms that if the cupola were actually bent in either direction by the endolymph current, it would be torn from its attachment. The impact or impulse of the endolymph current is "sensed," but the cupola is not bent or inclined in either direction by the current. Whether they are actually bent by an endolymph current, or that they only "sense" the impact of the endolymph, is not actually proved.

The Static Labyrinth.—The term static labyrinth refers only to the semicircular canals and the cristæ ampullares, the end-organs of the vestibular nerve. The function of the end-organs of the utricle and saccule has not been clearly defined. It has been supposed that one has something to do with the orientation of vertical movements, and the other with horizontal movements of the body, but this has not been proved. The function of the cristæ located in the ampullæ, have, however, been quite clearly determined, and it is to these end-organs of the vestibular nerve that we will confine our consideration.

In the ampulla of each semicircular canal is located a crista, which, upon stimulation, gives off nervous impulses that are transmitted through the vestibular nerve to Bechterew's, Deiters', and the angular nuclei in the cerebellum, and from these centres the impulses are distributed to the nuclei of the third and sixth cranial nerves, which supply the extra-ocular muscles of the eyes, and to the muscles of the body and extremities. These impulses, when thus distributed, cause certain "reaction movements" of the eyes, body, and extremities. Under normal conditions these "reaction movements" maintain the equilibrium of the body and eyes; hence, the portion of the labyrinth giving off these nervous impulses is known as the static labyrinth. When, however, the potentiality of these impulses is either lost or exaggerated by disease, or physiological stimulation, the eyes are affected by nystagmus; ataxia, nausea, and vomiting may also occur. In disease of the labyrinth the nervous impulses given off by the diseased labyrinth are always, or nearly always, either diminished or altogether abolished, except in circumscribed labyrinthitis. The cochlea which presides over the function of hearing may be spoken of as the auditory labyrinth; the utriculus saccule and semicircular canals, as the static labyrinth; and the whole, as the static-auditory labyrinth.[1]

[1] It is now thought by some investigators that the cristæ have direct connection with the third and sixth oculomotor centres.

Static Impulses.—The impulses emanating from the cristæ ampullares may be of three types, namely (*a*) normal, (*b*) increased, and (*c*) decreased or abolished.

(*a*) The normal static impulses are those excited in normal individuals by the ordinary movements and positions of the head. There is no consciousness of orientation, as they are normal under ordinary conditions of life.

(*b*) Increased static impulses are those which occur spontaneously without extra otitic stimulation, or which occur in response to slighter stimulations than those required to produce them in normal individuals; or the increased impulses may be due to extraordinary stimulation, as in the turning and caloric tests. Individuals having these increased static impulses are hypersensitive to vestibular stimulations.

(*c*) The decreased static impulses are those which can only be excited by extraordinary movements of the head, or other violent stimulation, as heat, cold, galvanism, and by compression or aspiration of the air in the meatus. Decreased static impulses are found only in individuals with a partially destroyed static labyrinth. (Partial loss of static impulses is sometimes present in circumscribed and in mild forms of serous labyrinthitis.) Static impulses are altogether abolished in diffuse manifest and diffuse latent suppurative labyrinthitis, and the fifth degree of serous labyrinthitis, in hemorrhage of the labyrinth, as in Ménière's disease, and fractures through the petrous portion of the temporal bone.

Spontaneous Nystagmus.—Spontaneous nystagmus of static labyrinth origin is characterized by rhythmical movements of the eyes, the movement in one direction having more speed than the movement in the opposite direction. The slow component is caused by the nervous impulse emanating from the crista or cristæ of the static labyrinth. The quick component is caused by a reflex impulse from a cortical centre. If the function of the cristæ is suddenly inhibited or destroyed by disease, the impulses emanating therefrom are diminished or altogether lost, whereas, if the diseased labyrinth is not destroyed or suppressed, but is stimulated, has an exaggerated tonus, the impulses causing the slow component of the spontaneous nystagmus emanate from the diseased static labyrinth, *i. e.*, a preponderance of impulse tonus or potentiality emanates from the diseased labyrinth, and this sudden change in the balance of tonus causes the nystagmus. As compensation occurs the nystagmus subsides. In one instance "the signs of destruction disharmony" and in the other the "signs of stimulation disharmony" are present. As a matter of fact, we do not find stimulation disharmony present in disease of the labyrinth except in some cases of circumscribed labyrinthitis, and in congestive disturbances of the labyrinth in the course of acute otitis media. When stimulation disharmony is present in acute otitis media it should not be regarded as a sign of labyrinth disease, but as a circulatory disturbance in the labyrinth. Spontaneous nystagmus is usually a combined horizontal and rotatory movement of the eyes. From a purely theoretical view the two labyrinths participate in the induced nystagmus.

Reaction Movements.—While nystagmus is in reality a reaction movement of static labyrinth origin, it is not generally referred to as such. The term "reaction movement" will, therefore, only be used to designate those movements of the body and extremities which are caused by nervous impulses emanating from the static labyrinth in ear disease, and certain centres in the brain, as in cerebellar disease.

Reaction movements of the body and extremities are also induced by the physiological tests, as the turning, caloric, galvanic, and fistula experiments. We may therefore deduce the law that *physiological stimulation of one static labyrinth, or fulminating or progressive disease of one static labyrinth, is always accompanied by nystagmus, and the "reaction movements" of the body and extremities, as ataxia, nausea, and vomiting, until compensation occurs. Compensation occurs after the vestibular tests in from ten to forty-five seconds; in disease from a few minutes or hours to a few weeks.*

It has also been shown by experiments upon animals, that when both labyrinths are simultaneously destroyed neither nystagmus nor reaction movements occur. It has been shown that after the surgical destruction of a labyrinth, compensation occurs much more rapidly than after destruction by some pathological process.

The reaction movements may be of the spontaneous or of the induced type. Spontaneous reaction movements are present in certain types and stages of labyrinth disease, and in cerebellar disease. They are only present a few minutes, hours, days, or weeks, in labyrinth disease, but may be present indefinitely in cerebellar disease. A stronger stimulation is necessary to induce reaction movements than to induce nystagmus. This is true of either the spontaneous or induced reaction movements.

The reaction movements of static labyrinth origin consist of a sense of surrounding objects rotating around the body or of the rotation of the body, and of nausea, vomiting (sometimes), and ataxia, with a tendency to fall toward the slow component of the nystagmus. In cerebellar disease the direction of the slow component exerts no influence on the direction of falling.

Pointing toward the slow component of the spontaneous nystagmus of static origin is the normal reaction in Barany's pointing test. When, therefore, a patient affected by spontaneous nystagmus points to the quick component, or points directly to the finger of the observer, the result is said to be abnormal or suggesting cerebellar disease (see Pointing Test).

The reaction movements are not of as much clinical importance as nystagmus, though they afford valuable information concerning disease of the static labyrinth, and more especially concerning cerebellar disease. According to Barany the reaction movements consist of voluntary movements, the impulses of which are modified in their transmission through the cerebellar cortex by normal impulses from the cristæ ampullares of the semicircular canals. That is, in the cerebellar cortex, motor impulses from the cerebrum are met by centripetal

impulses from the semicircular canals. If these centripetal impulses are abolished on one side by disease, or by abnormal stimulation of the semicircular canals in physiological experiments, the normal voluntary movements are changed in such a way as to result in reaction movements (Braun and Freisner). Inasmuch as the will-power and voluntary movements are factors in the reaction movements, they are not as reliable data upon which to estimate pathological processes as the nystagmus, in which the will-power and voluntary movements are not important factors. In children the will-power and muscle sense are not as highly developed as they are in adults, hence the reaction movements are better developed in children than in adults. Notwithstanding this, the reaction movements are of great value in determining the condition of the static labyrinth and cerebellum, as follows:

(*a*) The failure to induce nystagmus and reaction movements by the caloric and fistula tests, signifies destruction of the static labyrinth. In such a condition infection within the labyrinth might, without symptoms, extend to the meninges and brain until actual meningitis or cerebellar abscess was developed, hence there is an element of danger waiting for developments in such a case.

(*b*) When nystagmus and reaction movements are induced by slighter stimulation than is required in normal individuals (except in neurasthenics) it signifies an increased irritability of the static labyrinth, due to congestion of the labyrinth, as is occasionally found in acute otitis media. This should not be regarded as disease of the labyrinth.

(*c*) When nystagmus and the reaction movements require excessive stimulation, as by the caloric test, to induce them, it signifies diminished irritability of the static labyrinth, as in certain types of circumscribed and serous labyrinthitis.

An increased intensity of reaction signifies a congestion or mild inflammation of the labyrinth and a diminished intensity of reaction means active disease of the static labyrinth with partial suppression or destruction of function. Total abolition of the reaction means the destruction of the labyrinth in diffuse suppurative labyrinthitis.

The *quantitative* estimation of the irritability of the labyrinth has reference to either the presence or absence of nystagmus and ataxic symptoms; that is, the tests are made to determine whether or not nystagmus and ataxia can be induced. If one labyrinth is totally destroyed, the caloric test applied to the affected ear will not induce nystagmus and ataxia, *i. e.*, the static irritability is negative in quantity. The caloric test applied to a normal ear will induce nystagmus and ataxia, *i. e.*, the static irritability is positive in quantity.

The *qualitative* estimation of the irritability of the static labyrinth has reference to the degree of stimulation required to induce nystagmus, its duration and intensity, and is made in three ways, as follows:

(*a*) By the "Reizxchwelle" or strength of irritation, necessary to induce nystagmus. If the turning test is used, the number of rotations

required to induce nystagmus is noted. If the caloric test is used the temperature and amount of water required and the number of minutes necessary to produce nystagmus are noted.

(*b*) The qualitative estimation of labyrinth irritability is also made by determining the duration of the nystagmus with a constant strength and duration of the stimulation with Ruttin's double irrigator.

(*c*) By galvanism with a double electrode (from one pole of the battery) applied to both ears simultaneously, the other pole being held in the hand.

The turning test is also used for the quantitative test.

The Cristæ Ampullares.—The cristæ ampullares are the special end-organs of the vestibular nerve and are situated in the ampullæ of the semicircular canals. Each crista functionates on the plane of its associated canal. That is, stimulation of the crista of the horizontal canal produces nystagmus in the horizontal plane. Stimulation of the crista of the superior canal produces rotatory nystagmus on the frontal plane, etc. If the cristæ of the horizontal and superior canals are simultaneously stimulated, as in the warm caloric test, a combined horizontal (weak) and rotatory (strong) nystagmus follows. As has been previously stated, each crista is a double end-organ, one-half producing nystagmus to one side, and the other half to the opposite side, but in each instance in the same plane of the canal or canals stimulated.

The Cristæ of the Utricle and Saccule.—In the utricle and saccule are end-organs somewhat similar to the cristæ ampullares, the function of which has not been definitely determined, though it is supposed to influence the orientation of movements in the vertical and horizontal planes.

Spontaneous and Induced Nystagmus in Relation to Vestibular and Cerebellar Disease.—Before discussing nystagmus in relation to disease of the cerebellum and vestibular apparatus, a clear distinction between spontaneous and induced nystagmus should be made.

Spontaneous Vestibular Nystagmus.—This form of nystagmus occurs in acute diffuse suppurative manifest labyrinthitis, acute serous labyrinthitis, and at intervals in circumscribed labyrinthitis, and is generally due to either inhibition or destruction of the vestibular apparatus of the affected side, except in circumscribed labyrinthitis, and acute congestion of the labyrinth accompanying acute otitis media. In the latter disease the nystagmus is due to congestion of the labyrinth. Induced nystagmus may be elicited for diagnostic purposes in normal cases, and in the course of labyrinth disease after the spontaneous nystagmus has ceased, and when spontaneous nystagmus is present it may be made either more or less manifest by the usual procedures for inducing nystagmus. When one labyrinth is destroyed, as in diffuse latent suppurative labyrinthitis, or fracture through the base of the skull, and extralabyrinthine compensation has occurred, induced nystagmus may be elicited by stimulating the healthy labyrinth, thereby increasing the potentiality or tonus of its impulses. If one labyrinth is only partially disabled, as in the milder forms of serous labyrinthitis,

the other labyrinth being normal, or when both are normal, induced nystagmus may be caused by a stimulation of the cristæ, thereby establishing a discrepancy between the potentiality, pull, or tonus impulses emanating from the two vestibular apparatuses. The sudden disturbance of the existing balance of tonus causes the nystagmus.

Spontaneous vestibular nystagmus lasts for only a few minutes, hours, days, or weeks after the cristæ are inhibited by compression in diffuse serous labyrinthitis, or by destruction in diffuse suppurative manifest labyrinthitis. In circumscribed labyrinthitis it occurs in periodic attacks, brought on by jarring movements, and independently of jarring movement of the head. The periodic attacks of spontaneous nystagmus, occurring independently of jarring movements, are especially characteristic of circumscribed labyrinthitis.

In spontaneous vestibular nystagmus the discrepancy of potentiality, pull, or tonus impulses, is generally due to a sudden decrease, or total loss of irritability of the crista from disease, thus decreasing or abolishing its potentiality; the cristæ of the healthy labyrinth retaining their normal potentiality or tonus. When spontaneous nystagmus is to the healthy side, the affected vestibular apparatus may generally be assumed to be suppressed, as in serous labyrinthitis, or wholly destroyed, as in diffuse suppurative manifest labyrinthitis, the healthy side remaining normal in tonus, and producing the slow component of the nystagmus. With such an imbalance existing between the two labyrinths it would at first appear that spontaneous nystagmus should continue indefinitely. Its disappearance is due, however, to accommodation, readjustment of function, or compensation extraneous to the labyrinths, rather than between the two labyrinths. The compensation takes place chiefly outside of the labyrinths, though it may take place in the healthy labyrinth. To induce nystagmus it is now necessary to artificially disturb the existing "compensating extralabyrinthine tonus," by either the turning, caloric, or fistula test. According to Neumann it is possible that spontaneous nystagmus may altogether be absent when there is a very exaggerated tonus in the two centrums, because of their close approximation.

Characteristics of Spontaneous Vestibular Nystagmus.—Spontaneous nystagmus may be said to occur in three degrees, namely:

(*a*) The first degree.
(*b*) The second degree.
(*c*) The third degree.

The *first* or *weakest degree* only occurs when looking toward the quick component, and ceases when looking straight ahead, or to the slow component. G. W. MacKenzie has shown that many persons with normal ears have nystagmus when looking to the extreme right or left, hence, if there was no history of spontaneous nystagmus previously the first degree nystagmus should be regarded as physiological nystagmus. When first degree nystagmus is present in disease of the labyrinth it is usually stronger when looking to the diseased side, a point which may differentiate it from physiological nystagmus.

The *second* or *medium degree* of nystagmus occurs when the patient looks toward the quick component, and when looking straight ahead, and it ceases when looking toward the slow component.

The *third* or *strongest degree* of nystagmus occurs when looking in any direction. That is, it cannot be stopped by looking in any direction, even to the slow component, and more often occurs at the beginning of acute diffuse suppurative and serous labyrinthitis, and gradually becomes weaker and weaker, more easily stopped, as it fades into the second and first degrees.

Cerebellar Nystagmus.—Cerebellar nystagmus may be either spontaneous or induced, and is usually to the affected side, whereas in destructive disease of the labyrinth it is to the opposite or healthy side. It is spontaneous when there is disease or irritation in that portion of the cerebellum in which the vestibular centrums (Deiters' and Bechterew's and the angular nuclei) and the vestibulocerebellar tracts are located, and is a sign of stimulation disharmony. Cerebellar nystagmus may be induced by the galvanic test when the labyrinth is destroyed and the vestibulocerebellar tract and the nuclei are still intact. After the destruction of the labyrinth, the vestibulocerebellar tract is gradually destroyed by ascending degeneration, until, finally, the central nuclei on the same side are invaded by the degenerative process, and the destruction is complete. G. W. MacKenzie has shown that rhythmic nystagmus may be induced without vertigo by mild galvanic stimulation. It has been known, however, that nystagmus is more easily invoked than the reaction movements. A stronger stimulation would have induced the vertigo and disturbance of equilibrium.

If the galvanic test is applied, from time to time, the progress of degenerative process may be noted and estimated. Soon after the destruction of the labyrinth, the induced cerebellar nystagmus, by galvanization (see Galvanic Test) is pronounced. As tests are made from time to time, the reaction gradually becomes weaker and weaker, until finally, after many months or years, it ceases altogether, as the vestibulocerebellar tract and centrums are completely destroyed by the degenerative process. (See Plates XIX, XXIII, and XXV.)

The character of cerebellar nystagmus is somewhat similar to vestibular nystagmus, though it differs in a few particulars. It has a slow and quick component, but is more irregular in its oscillations, and appears at intervals. Its direction is usually to the affected side, though it may be to the healthy side (Plate XVII). If the cerebellar nystagmus is produced by the irritation of the centrum from toxic material or congestion, the nystagmus will be to the same side as the disease. If however, the cerebellar disease compresses or paralyzes the centrum, the nystagmus will be to the healthy side. As both centrums are situated near the median line it is quite possible that the cerebellar disease may affect the centrum on the opposite side, rather than the one on the same side. The spontaneous nystagmus may not be manifest until opaque spectacles are applied to the eyes. The foregoing

facts explain the variableness in the direction of nystagmus in cerebellar disease, as basal meningitis, abscess, and tumors. Cerebellar nystagmus tends to continue indefinitely, and to increase in intensity, as the disease causing it continues and progresses indefinitely; whereas spontaneous vestibular nystagmus rapidly decreases in intensity, and after a few minutes, hours, days, or weeks, ceases altogether, especially after destruction of the labyrinth by diffuse manifest suppurative labyrinthitis, and after inhibition by diffuse serous labyrinthitis (Plates XVII and XXV). There is, however, great variableness in the expression of cerebellar nystagmus. The disappearance of the spontaneous nystagmus is not due to the disappearance of the disease, but chiefly to extralabyrinthine compensation. The persistence of nystagmus over many days, weeks, or months, is therefore strongly indicative of cerebellar disease, as abscess, tumor, or tubercle, in the cerebellopontine angle. Cerebellar nystagmus due to the toxemia of erysipelas ceases with the disappearance of the erysipelas, and does not tend to be indefinitely prolonged, as in tumors and abscess of the cerebellum.

Cerebellar Nystagmus due to Erysipelas of the Scalp and Face.—Erysipelas of the face or scalp may be attended by severe headache, congestion, and toxemia of the cerebellum; hence spontaneous nystagmus to the same side may be present in this disease, and is due to congestion, or to the stimulus of the toxic products. The centrums are stimulated as in cerebellar disease. According to Ruttin, who first made these observations upon erysipelas in the mastoid region, spontaneous nystagmus may be the earliest sign of erysipelas, as it sometimes occurs before redness of the skin. In six cases of erysipelas shown me by him, in April, 1911, the nystagmus was to the diseased side, thus exhibiting the signs of stimulation disharmony.

In September, 1911, I observed spontaneous nystagmus to both sides in a case of bilateral erysipelas of the face following a cosmetic operation upon the nose. By having the patient look first to the right, and then to the left, spontaneous nystagmus was made manifest in both directions. The nystagmus subsided with the disappearance of the erysipelas.

Characteristics of Induced Nystagmus by Turning.—In making the turning test it is necessary to differentiate between three types of expression of induced nystagmus, namely, (*a*) induced primary nystagmus, (*b*) induced after-nystagmus, and (*c*) induced after-after-nystagmus.

(*a*) *Induced primary nystagmus*, by the turning test, is the nystagmus which occurs during the turning, and is toward the direction of the turnings. (It is not practicable to observe nystagmus during the turnings, hence the induced after-nystagmus is observed instead.)

(*b*) *Induced after-nystagmus*, by the turning test, is the nystagmus which occurs after the turnings cease, and is in the opposite direction to the primary nystagmus, or opposite to the direction of the turnings. (Plates XIX, XX, and XXV.)

(*c*) *Induced after-after-nystagmus*, by the turning test, is the nystagmus which sometimes occurs when the after-nystagmus ceases, and

is in the same direction as the primary induced nystagmus, that is, toward the direction of the turnings, though weaker than the primary nystagmus. Induced after-after-nystagmus occurs after a prolonged turning test, and is explained as a phenomenon of fatigue from over-stimulation. Induced vestibular nystagmus may be horizontal, rotatory, oblique, vertical, or combined horizontal and rotatory.

Characteristics of Induced Caloric Nystagmus.—Caloric nystagmus is always rotatory and in the plane of the canal stimulated. By the cold caloric test the nystagmus is simple rotatory and is symbolized thus ↷. By the warm caloric test it is combined rotatory and horizontal and is symbolized thus ⇄.

Characteristics of Induced Fistula Nystagmus.—Nystagmus by compression, in fistula of the labyrinth, may be horizontal or rotatory, according to the location of the fistula. If it is in the external limb of the horizontal canal the nystagmus will be horizontal and to the same side, and is symbolized thus →. If the fistula is in the oval window the nystagmus will be horizontal and to the opposite side. If it is in the external arm of the superior canal it will be rotatory and to the same side, and is symbolized thus ↷.

Characteristics of Induced Galvanic Nystagmus.—Induced galvanic nystagmus is always rotatory. Galvanic nystagmus unlike any other induced nystagmus, may be induced even when the labyrinth and its cristæ are totally destroyed through stimulation of the vestibular nerve and Deiters' nucleus. All types of induced vestibular nystagmus have a slow (vestibular element) and a quick (cortical element) component. All types of induced vestibular nystagmus endure from a few seconds to sixty seconds. Spontaneous vestibular nystagmus endures for from a few minutes to three weeks, gradually diminishing in intensity.

Laws of Universal Application.—There are four laws of general application, first announced by Fleurens, Ewald, Hoegyes, and Neumann respectively.

Fleurens' Law.—The movement of the eyes is in the plane of the canals subjected to stimulation. When the cristæ of the horizontal canals are stimulated the eye movements are in the horizontal plane; when those of the superior (frontal) canals are stimulated, the movements of the eyes are in the frontal plane, that is, the eyes rotate upon their pupillary axes; when those of the posterior (oblique) canals are stimulated, the eye movements are in the vertical or saggital plane; and when the horizontal and superior canals are simultaneously stimulated there is a combined horizontal and rotatory nystagmatic movement of the eyes in the corresponding planes, and in the same direction; that is, the horizontal and rotatory nystagmus are in the same direction either to the right or to the left. When one superior canal, and the opposite posterior canal, are simultaneously stimulated the induced nystagmus is oblique in direction and is symbolized thus ↗.

Hoegyes' Law.—Each centrum (right and left) controls the adductor muscle of the same side and the abductor of the opposite side, respec-

tively. For example, the right centrum, Deiters' nucleus, controls the adductor of the same side, and the abductor of the opposite or left centrum controls the adductor of the same side, and the abductor of the opposite or right side, thus producing conjugate movements of the eyes to the right (slow component). Since the publication of Hoegyes' law it has been determined that, while under ordinary conditions the law is applicable, it does not express the whole truth. We now believe that each crista is a double end-organ and that each half emits nervous impulses antagonistic to the other half. The impulses from one half, however, are twice as strong as those given off from the opposite half. The stronger half of the crista determines the dictum of Hoegyes' law, the weaker half of the crista being a negligible quantity in the turning test.

Neumann's Law.—Imagine the right and left horizontal, and the right and left superior canals, respectively, united at their non-ampullated ends, thus forming two half-circles, with their ampullæ at the ends of the half-circles, and the turning-point midway between the ampullæ of each pair of canals, and you have a visualized image of the direction of the flow of endolymph through each pair of canals. The head should be inclined 90 degrees either forward or backward in testing the superior canals (Plate XIX). This law does not apply in testing the posterior vertical (oblique) canals, as they do not lie in the same plane. Their relation to each other is as the two arms of the letter V. When the cristæ of these canals are simultaneously stimulated, the movements of the eyes will be the mean between the angles of the two canals, that is, vertical; hence, when they are simultaneously stimulated the nystagmus is vertical, and is symbolized thus ↑. The right superior and left posterior canals lie in the same plane, *i. e.*, the oblique, and when they are simultaneously stimulated the nystagmus is in the oblique plane. This is also true of the left superior and the right posterior canals. To visualize the relationship of the horizontal canals, clasp the hands behind the head and extend the elbows forward. The elbows correspond to the ampullæ, while the clasped hands correspond to the suppositious, united non-ampullated ends of the two horizontal canals. To visualize the superior canals clasp the hands over the vertex of the head, and allow the elbows to hang by either side of the face. The elbows correspond to the ampullæ of the superior canals, and the clasped hands to the suppositious, united non-ampullated ends of the canals.

Ewald's Law.—The greater physiological impulses are produced in the horizontal canal by the movement or impact of the endolymph from the smooth or non-ampullated end of the canal, toward the ampullæ and utricles; whereas, the stronger physiological impulses from the crista of the superior and posterior canals are produced by a flow of endolymph in the reverse direction, *i. e.*, from the utricles through the ampullæ to the smooth or non-ampullated ends of the canals.

The Cristæ Ampullares.—Each crista ampullaris is a double end-organ of the vestibular nerve, the hair cells constituting the specialized nerve endings. The crista is a ridge-like eminence extending transversely across the ampulla and is surmounted by hair cells on each side of the ridge. Enveloping the hairs is a delicate gelatinous substance called the cupola (see Plates XIX to XXVIII). The hair cells on the utricular half of the crista emanate impulses to certain muscles of the eyes, body, and extremities; while those on the other, or canal half of the cristæ, emanate impulses to the opposing groups of muscles, of the eyes, body, and extremities (see Plates XIX to XXVIII). The two halves of each crista are therefore antagonists. The nerve fibers emanating from one half of the crista are apparently more numerous than those from the other half, or, at least, the impulses emanating from one half or side of the crista, are more than twice as strong as those emanating from the other side. That is, the canal half of the crista of the horizontal canal gives off impulses about twice as strong as those given off by the utricular half of the crista; whereas, in the superior and posterior canals the stronger impulses arise from the utricular halves of the cristæ, and the weaker impulses from the canal halves of the cristæ. The utricular half of the crista of the left horizontal canal, and the canal half of the crista of the right horizontal canal act in unison in pulling the eyes (slow component) to the left.

In each canal the greater reaction follows the stimulations of those hair cells which cause nystagmus to the same side or which cause the slow component to the opposite side. To visualize the relative strength of the impulses given off the cristæ, two lines are used in the plates accompanying the text to represent the nervous tracts from the stronger half of each crista, and one line to represent the nervous paths from the weaker half of each crista.

The nerve fibers arising from the utricular half of the crista of the right labyrinth go to the same muscles as those arising from the canal half of the left labyrinth. Whereas, the nerve fibers arising from the utricular half of the crista of the left labyrinth go to the same muscles as those arising from the canal half of the corresponding crista of the right labyrinth (Plates XIX to XXVIII). The utricular half of a crista of the right labyrinth, and the canal half of the corresponding crista of the left labyrinth, therefore, act in unison.

In the turning test to the right, the flow of endolymph (after the turnings have ceased) is to the right, and the impact of endolymph is against the utricular half of the crista of the horizontal canal of the right labyrinth, and against the canal half of the crista of the horizontal canal of the left labyrinth (Plate XX). A weak impulse is therefore emitted from the right labyrinth, and a stronger one from the left labyrinth, which conjointly pull the eyes (slow component) to the right. The reverse movement of the eyes is induced when the patient is turned to the left.

It appears, therefore, that two impulses may be excited in a given crista in labyrinth disease, and that they may be evoked in making

the various physiological tests in disease and health. Through the interaction of these impulses and the cortical reflex impulses, nystagmus is produced. In acute non-destructive congestion of the labyrinth, we must think of both halves of a given crista as being in a state of stimulation. For example, in congestion of the labyrinth due to acute otitis media (right ear) both halves of the crista of the right horizontal canal are stimulated. The canal half of the crista, giving off the stronger impulses, turns the eyes (slow component) to the left, while the utricular half of the stimulated crista tends to pull the eyes in the opposite direction, or to the right. The two halves of the crista are antagonists, but the canal half, giving off the stronger impulses, determines the direction of the slow movement of the nystagmus.

FUNCTIONAL TESTS OF THE VESTIBULAR APPARATUS

The Turning Test.—This test is made by placing the patient in a revolving chair (Fig. 486) and turning him ten times, either to the right or to the left. If the turnings are made with the head erect, only the cristæ of the horizontal canals are stimulated, and a horizontal nystagmus results. If the head is inclined 90 degrees, either forward or backward, only the cristæ of the superior canals are stimulated, and rotatory nystagmus results. If the head is inclined 45 degrees to either shoulder, one of the posterior vertical and one of the superior vertical canals are brought to the horizontal plane and an oblique nystagmus results. Each revolution of the patient should occupy from one to two seconds.

Fig. 486

Revolving chair used in turning test.

In acute destruction of one labyrinth, with only partial compensation, from two to five turnings will induce nystagmus. This is explained by the fact that there is only a partially established equality of extralabyrinth tonus; hence, the slight added stimulus caused by a few turnings is sufficient to overcome the existing fragmentary balance, and nystagmus results. As heretofore explained, the early compensation is chiefly extralabyrinthine; that is, in the cerebellar centres. Neurasthenic cases, without labyrinth disease are more susceptible to the turning test than non-neurasthenic ones; hence, in such cases less than ten turnings may produce maximum nystagmus when both labyrinths are normal. The absence of other signs of ear disease would readily differentiate these cases from labyrinth disease.

Another fact that should be constantly held in mind in making all tests for pathological processes in the vestibular apparatus is, that the irritability of the diseased organ is diminished or altogether lost, in (*a*) acute diffuse suppurative manifest, (*b*) diffuse latent suppurative labyrinthitis, and in (*c*) acute diffuse serous labyrinthitis (temporarily). In serous labyrinthitis the vestibular and cochlear functions gradually return after the absorption of the serous exudate in the lymph spaces. Formerly it was taught that increased irritability of the diseased vestibular apparatus was a constant factor, whereas, as I have already said, it may be increased in certain diseases, as in congestion of the labyrinth attending acute otitis media and in circumscribed labyrinthitis, but becomes diminished, or altogether lost, in acute, diffuse, suppurative, and serous labyrinthitis. In diffuse manifest and latent suppurative labyrinthitis the irritability of the vestibular apparatus is always permanently lost as the labyrinth is destroyed.

The Rationale of the Turning Test.—As previously stated, each crista ampullaris is a double end-organ of the vestibular nerve, and its physiological activity is increased by stimulation from the impact of the

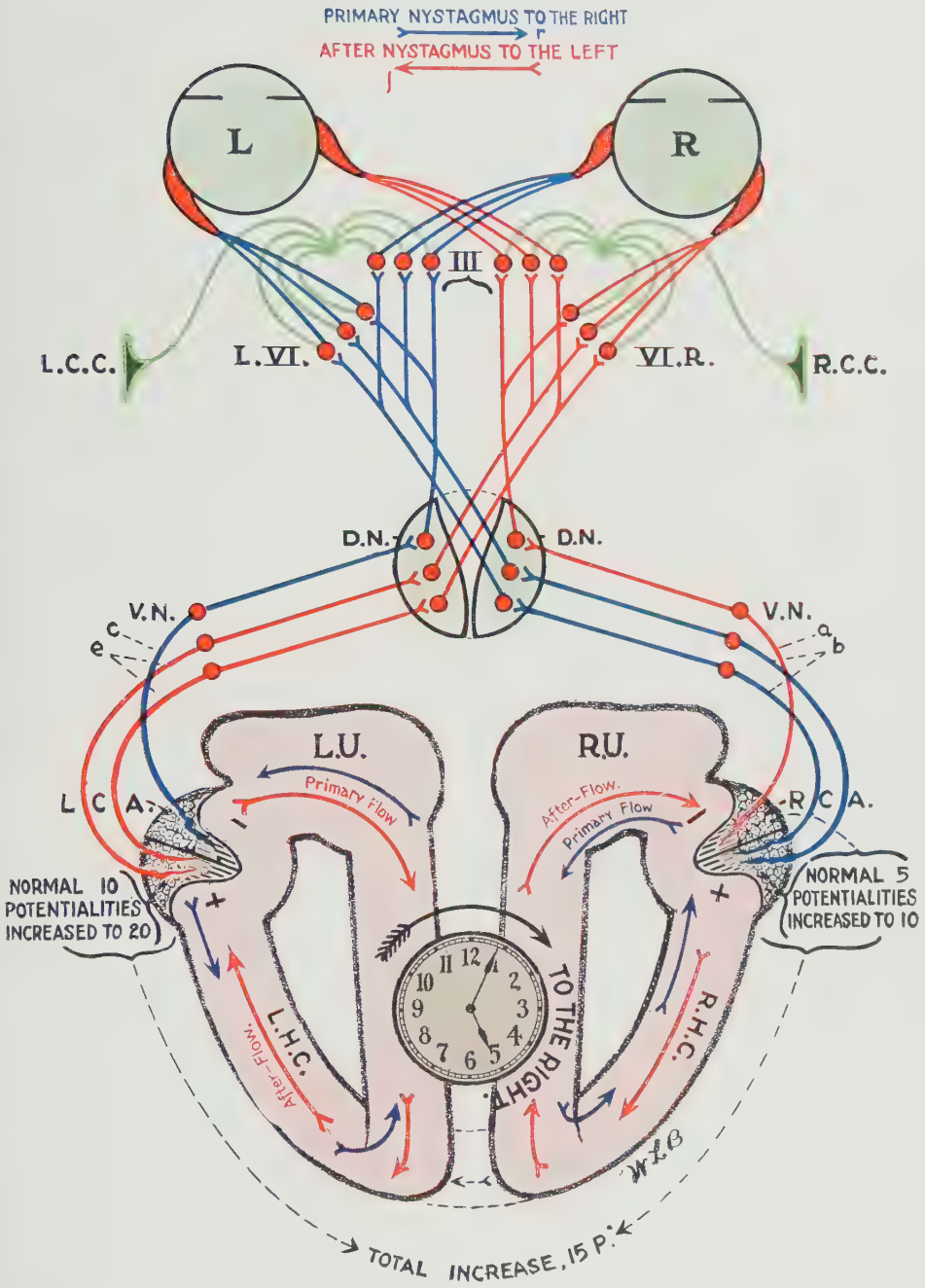
---

PLATE XX

Diagrammatic Illustration of the Nervous Connections of the Horizontal Semicircular Canals with the Eyes; and the Reaction Induced after Turnings to the Right Have Ceased.

L, left eye; *R*, right eye; *L.C.C.*, left cortical centre; *R.C.C.*, right cortical centre; *III*, nucleus of the third cranial nerve; *L.VI*, nucleus of sixth cranial nerve; *R.VI*, nucleus of right sixth cranial nerve; *D.N.*, Deiters' nucleus; *V.N.*, vestibular nucleus; *L.U.*, left utriculus; *R.U.*, right utriculus; *L.C.A.*, left crista ampullaris; *R.C.A.*, right crista ampullaris; — indicates the half of the crista giving off the weaker nervous impulses; + indicates the strong half of the crista; *L.H.C.*, the left horizontal semicircular canal; *R.H.C.*, the right horizontal semicircular canal. Turning to the right is in the direction of the movements of the hands of a watch placed face upward. The blue arrows indicate the direction of the flow of endolymph during the turnings (primary flow). The red arrows in the canals indicate the direction of the flow of endolymph in the canals after the turnings have ceased (after-flow). During the turnings to the right the flow of endolymph (primary flow) is opposite to the direction of the turnings, or to the left, and the impact of this endolymph is against the canal half (+) of the right crista, and against the utricular half (—) of the left crista. The impulses thus excited turn the eyes to the left (slow component) and the cortical correction to the right immediately follows. Turning to the right therefore produces primary nystagmus to same side or to the right. The impact of the after-flow of endolymph in the left canal is against the hair cells of the half of the crista giving off the stronger (+) nervous impulses, whereas in the right canal it is against the weaker half of the crista (—). By tracing the red lines from each crista, through Deiters' nuclei (*D.N.*, *D.N.*) and the right oculomotor nuclei (*III*, *VI.R.*) to the abductor muscles of the right eye and the adductor muscles of the left eye, an idea is formed of the nervous mechanism required to produce the conjugate slow movement of the after-nystagmus. The slow movement is to the right or in the direction of the turnings. A corrective cortical impulse is immediately liberated in the left cortical centre (*L.C.C.*) which is transmitted through the left third and sixth oculomotor nuclei (*III.L.*, *VI.L.*) to the adductor muscles of the right eye and to the abductors of the left eye, thereby producing a quick conjugate movement of both eyes to the left (quick component of the nystagmus). The after-nystagmus is horizontal in the plane of the canals stimulated, and is to the left or opposite to the direction of the turnings. It is symbolized thus, →l.

PLATE XX



Physiological Nystagmus by the Turning Test.

endolymph current against it. In the turning test the cristæ of both horizontal canals are stimulated, one giving off impulses twice as strong as the other; that is, in one canal, the canal aspect of the crista, the side of the greater physiological activity is stimulated, and gives off impulses of, say 20 potentialities, and the utricular aspect of the other crista, side of lesser physiological activity, is stimulated, and gives off an impulse of 10 potentialities. As the two cristæ act in unison, they cause the slow component of the nystagmus (Plate XX).

In the horizontal canals, the head erect, the turnings to the right, the flow of endolymph at the beginning of the turnings is in the opposite direction to the turnings, as the fluid lags behind, according to the law of inertia. At the beginning of the turnings to the right the fluid in the right horizontal canal flows from the canal through the ampulla to the utricle. In the left canal the impact of the current is against the utricular aspect of the cupola, the side giving off the lesser physiological impulses. The halves of the respective crista stimulated act in unison, the right crista emitting the greater impulse (controlling factor), and the left the lesser impulse (complimentary factor), the eyes are turned to the left (slow component). This liberates the reflex from the cortical centre (Plate XX, *L.C.C.*), which gives rise to the quick conjugate movement of the eyes to the right. It is, however, not practical to observe the primary nystagmus which occurs during turning, hence in practice the after-nystagmus is observed instead. The after-nystagmus is in the reverse direction to the primary induced nystagmus and is produced by the change in the direction of the flow or impact of endolymph, when the turnings are suddenly stopped. At the beginning of the turnings the endolymph lags behind the walls of the canals, or flows in the opposite direction to the turnings, but after a few seconds it becomes stationary in the canals, and when the turnings suddenly cease, it flows in the direction of the turnings (Fig. 487, Plate XX). The after-flow of endolymph is therefore to the right, when the turnings have been to the right, and *vice versa*, when to the left.

In the right horizontal canal the fluid (when the turnings have ceased) flows from the utricle toward the canal, thus stimulating the hair cells of the crista giving off the weaker impulses. In the left canal the fluid flows from the canal toward the utricle, stimulating the hair cells of the crista on the side giving off the stronger impulses. The combined impulses from the two cristæ acting in unison cause the slow movement of the nystagmus to the right, and the quick component to the left immediately follows (Plate XX). If, before the turnings, the potentiality of the canal half of the left crista was 10, and the utricular half of the right crista was 5, immediately after the turnings stop, the potentiality pull or impulse in the canal half of the left was increased, say 10 potentialities, while in the utricular half of the right crista it was increased 5 potentialities, the combined increase in the strength of the impulses is 15 potentialities, which

determines the slow component of the after-nystagmus to the right (Plate XX).

The slow component of the after-nystagmus is always toward the same side as the direction of turning, while the quick component is toward the opposite side.

In disease of the labyrinth the same principles apply as in the normal labyrinth, though there is some variation in the expression of the nystagmus; that is, the duration and the amplitude of the excursions may be somewhat modified, and fewer turnings may be required to induce the nystagmus, especially if complete extralabyrinthine compensation has not occurred. The amplitude and duration of the excursions is somewhat proportionate to the degree of discrepancy existing in the tonus (either normal or compensating tonus) of the two vestibular apparatuses. They are also in close relationship to the recentness and suddenness of the disturbed equilibrium.

In testing the superior canals, Neumann's law in reference to imaginary union of the smooth or non-ampullated ends of the superior canals, thus making a half-circle in the frontal plane, with the ampullæ at the ends of the half-circle, holds true, as in the horizontal canals. Hoegyes' law in reference to the centrum of the right and left sides, respectively, controlling the adductor muscles of the eye of the same side, and the abductors of the opposite side, also applies to these canals. Fleurens' law is also exemplified in this experiment, *i. e.*, the nystagmus is in the plane of the canals subjected to stimulation, rotatory on the pupillary axes. To test the superior canals by the turning test, the head must be inclined 90 degrees, either forward or backward, to bring the canals into the horizontal plane. The after-nystagmus induced by turning to the right, with the head forward, is rotatory, and to the right. For an explanation of the difference in the direction of the induced nystagmus consult Plate XIX, in which it is shown that when the head is inclined forward, the crista of the left canal receives the impact of the after-flow on its utricular side, the side giving off the stronger physiological impulses, while the right crista receives the impact on the canal half of the crista, which gives off the weaker nervous impulses. The two cristæ act in unison in producing the rotatory slow eye movement to the right, and the cortical correction to the left follows; hence, the after-nystagmus is rotatory and to the left. Plate XIX shows that when the head is inclined 90 degrees backward, and the turnings are to the right, and suddenly stopped, the crista of the right side is impacted on the side giving off the stronger physiological impulses (+), while the left is impacted on the side giving off the weaker impulses (−); hence, the right crista controls, though both act in unison, and the after-nystagmus is rotatory to the right.

A simpler statement is that (*a*) when the head is inclined forward the after-nystagmus is opposite to the direction of the turnings, and (*b*) when the head is inclined backward the after-nystagmus is in the direction of turnings. It is rotatory in the frontal plane in each

instance. In accordance with Fleurens' law, namely that the nystagmatic movements of the eyes are in the plane of the canals stimulated.

According to Barany the average duration of induced nystagmus from the horizontal canals is forty seconds, and from the superior canals, twenty-six seconds.

The Caloric Tests.—The caloric tests are, generally speaking, more reliable than the others, though when certain conditions are present, as granulations, ceruminous plugs, polypi, atresia of the meatus, or large granulations, the caloric test cannot be successfully performed. Marked elevation of temperature may, for obvious reasons, also interfere with the reaction. When none of these conditions is present the caloric test is, perhaps, the most delicate, and conveys the most significant information of the various tests at our command. The reaction depends upon projecting a stream of water, of either a considerably higher or a lower temperature than that of the body, against the drum-head or the promontorium, when perforation of the membrana tympanum is present. Colder, rather than warmer, than the body temperature is preferable, as the patient often does not tolerate water of high enough temperature to ensure a reaction, even when functional activity is present. The reaction is dependent upon either raising or lowering the temperature of the endolymph in the outer and more exposed portion of the utriculus, and the membranous horizontal and superior canals. The ampullæ of these canals are near together, just above the oval window. The nystagmus is always rotatory, though it may be combined rotatory and horizontal, the rotatory element always being the stronger. The posterior canal is too deeply situated to be influenced by either cold or heat; hence, it cannot be tested for the caloric reaction. The caloric, like the turning test, depends upon establishing a circulation of the endolymph through the utriculus and membranous semicircular canals, or in the case of the cold caloric test, upon inhibition of the nervous impulses in the tested ear. The cold test induces a flow of endolymph against the weaker half of the crista of the superior canal (Plate XIX) and the nystagmus should be weaker than that produced by the warm test. As a matter of fact, it produces a stronger reaction, which may be explained upon the theory of inhibition. The circulating fluid being impacted against the cupola of the crista ampullaris, stimulates the hair cells upon the impacted side, and this increases the impulse, physiological pull, or potentiality, as explained under Turning Test.

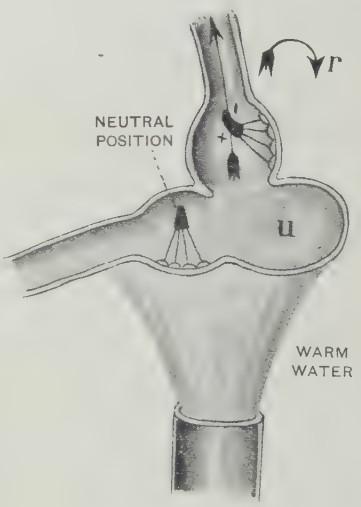
The caloric test has been regarded as both a qualitative and a quantitative test. G. W. MacKenzie, however, says that it is unreliable as a quantitative test, as it is impossible in most cases to have the same conditions present in both ears. That is, in one ear the drum-head is intact, and in the other perforated or absent, and if present in both ears, one may be thicker than the other. Inspissated pus or mucus may be present in one ear and absent in the other. Polypi or granulations may be present in one ear only; cerumen may be present in one ear and absent

or of smaller quantity in the other; one meatus may be smaller or partially closed by atresia; the affected or inflamed ear has a higher temperature than the uninflamed ear, etc. All these conditions would interfere with the equality of the tests of the two ears, and as one or

Fig. 487

Showing (*a*) the caloric test (warm water), right ear, producing nystagmus, the quick component of which is to the affected or tested side; (*b*) the negative galvanic current (—) applied in front of the right ear, producing nystagmus to the same side; (*c*) turning the patient to the right with the quick component of the primary nystagmus (during turning) to the right.

Fig. 488



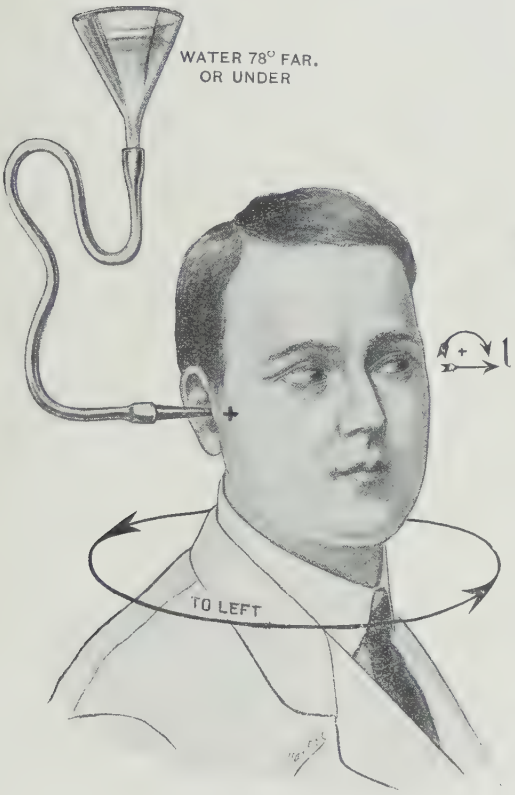
Schematic drawing, showing the influence of hot water applied to the right middle ear: *u*, the utriculus. As the endolymph in the utriculus is warmed, it rises through the anterior vertical semicircular canal, and thus stimulates the crista ampullaris of this canal upon the (+) side of greatest physiological activity. As the horizontal canal is on a lower level than the utriculus, the endolymph remains stationary. The result of warm irrigations is therefore limited to rotary nystagmus to the right.

more of them is present in nearly every individual, especially if one ear is diseased, MacKenzie claims the caloric test is of little or no value in estimating the degree of destruction present in the affected ear.

On the other hand Barany, Neumann, Alexander, and most other writers and observers, regard the caloric test as one of the best quantitative tests at our command, though they call attention to the above conditions as factors which may render it of no value as a quantitative procedure.

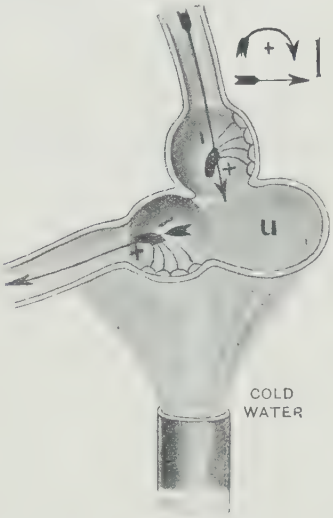
The Caloric Test with Cold Water.—This test is performed by stimulating the vestibular apparatus with a small stream of water at about

FIG. 489



Showing (*a*) caloric test (cold water), right ear, with nystagmus to the left; (*b*) the positive galvanic electrode (+) in front of the right ear, causing nystagmus to the left; (*c*) turning the patient to the left, causing primary nystagmus to the left. The total result is a combined horizontal and rotary nystagmus to the left.

FIG. 490



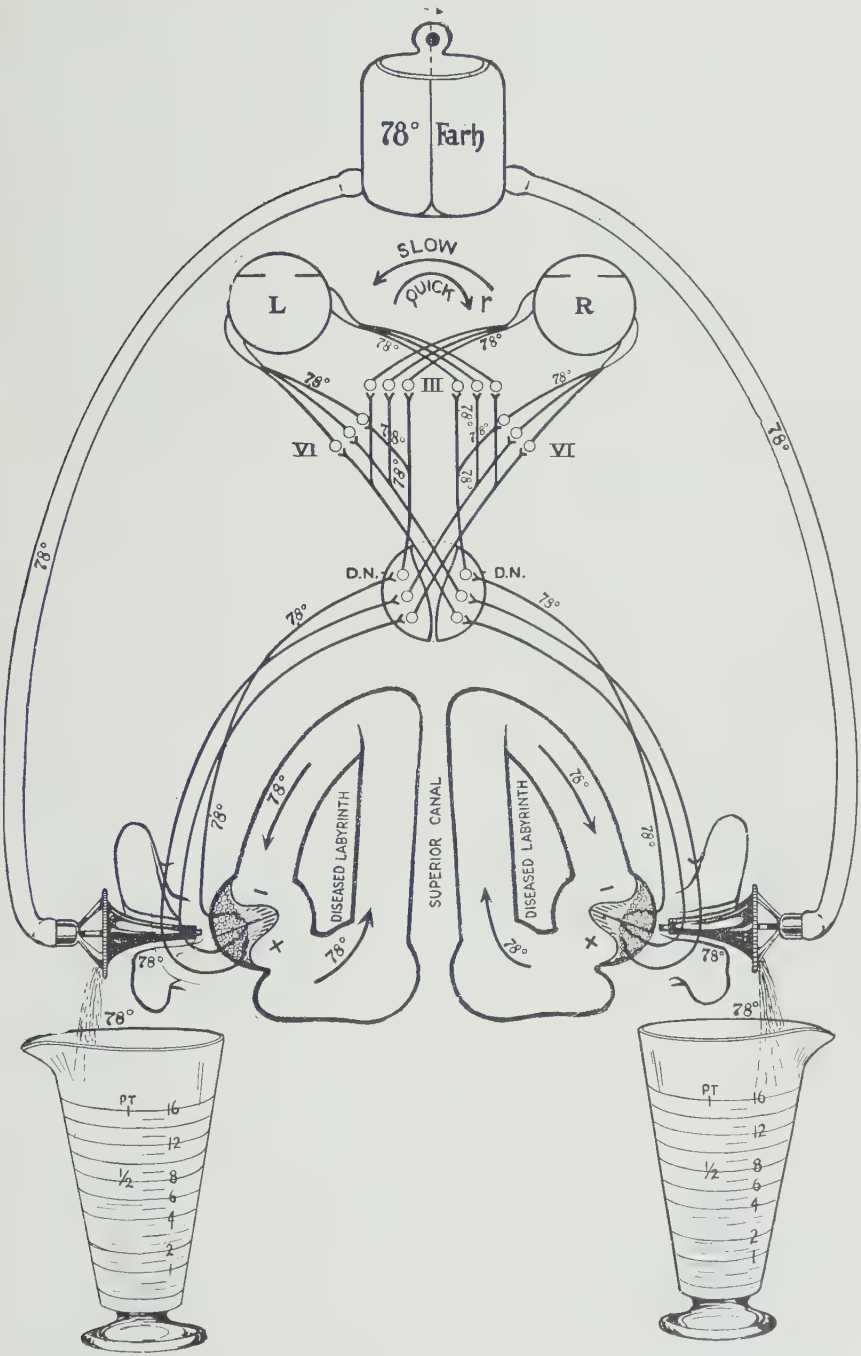
Schematic drawing, showing the influence of cold water applied to the right middle ear: *u*, the utriculus. As the endolymph in the anterior vertical and horizontal canals and the utriculus is cooled, it seeks the lowest level, hence the movement of the endolymph in the anterior vertical canal is from the ampulla to the utriculus. The crista ampullaris is thus stimulated upon the side of least physiological activity *and causes rotary nystagmus to the left.* The endolymph also flows downward from the utriculus through the ampulla of the horizontal canal, *and stimulates the crista ampullaris upon its side of least physiological irritability and produces horizontal nystagmus to the left.* The total result of cold-water irrigation is, therefore, a combined horizontal and rotary nystagmus to the left or opposite side.

78° F., though in some cases water of a lower temperature will be required to induce nystagmus. A fountain syringe, elevated slightly higher than the head of the patient, may be used in making this test (Figs. 489 and 490), or a Politzer bag with a suitable tip may be used. Force is not necessary, as the only object of the procedure is to cool the outer wall of the utriculus and membranous canals, thus inducing

a circulation of the endolymph through them. According to a well-known physical law, when that portion of water or other fluid contained in a vessel is cooled, it sinks or flows downward, thus creating a circulation of the fluid within the vessel. As the external limb of the superior canal and external wall of the utriculus are cooled, there is a downward flow of the endolymph, which causes an impact against the hair cells on the canal or weaker half of the crista ampullaris of the superior canal (Fig. 490). (See preceding paragraph for other explanation.) The potentiality of this half of the crista is thereby increased, while the crista of the superior canal of the opposite labyrinth remains unaffected. We will assume that before the test was applied each canal half of the crista had a potentiality or pull of 10, and that during and for several seconds after the test, the crista of the right or irrigated labyrinth was decreased to 5 potentialities. The potentiality of the left crista being normal, greater than that of the right or tested ear, which is diminished to 5. The left crista having the greater potentiality, in accordance with Hoegyes' law, turns the eyes to the right (slow component), and the corrective quick component to the left immediately follows. The nystagmus being named after the direction of the quick component, it is said to be to the left, or to the opposite side, or away from the side being tested (Plates XIX and XXVII). And, in accordance with Fleurens' law, the nystagmus corresponds with the plane of the canal stimulated, *i. e.*, it is rotatory, and in the frontal plane. The eyes rotate upon their pupillary axes. It should be said in addition that the cold caloric test may produce a combined horizontal and rotatory nystagmus, because the endolymph may also flow backward through the horizontal canal, which is sometimes inclined slightly downward, a fact emphasized by Barany, Neumann, and J. R. Fletcher. This causes inhibition of the crista of the right horizontal canal. The response to the inhibition applied to the two canals is a weak horizontal and a stronger rotatory nystagmus, which is due to a stronger tonus in the left labyrinth. The total result is called a combined weak horizontal and a strong rotatory nystagmus (Figs. 489 and 490).

According to Neumann a prolonged induced nystagmus may be established in cerebellar disease by irrigating the healthy side with cold water. This is known as Neumann's enduring nystagmus. The nystagmus induced by cold water in a normal ear, when there is no labyrinth or cerebellar disease, is of much shorter duration. The enduring nystagmus induced by the cold caloric test, applied to the normal side in cerebellar disease, is due to the fact that the spontaneous nystagmus of cerebellar origin is augmented by the inhibition following the cold caloric test. The cerebellar nystagmus is to the diseased side, and the induced cold caloric nystagmus caused by inhibiting the healthy side leaves the diseased side with a great preponderance of tonus, which results in the increased and prolonged or enduring nystagmus.

FIG. 491



Ruttin's double irrigator for making a comparative test of both ears at the same time.

The Caloric Test with Warm Water.—Water of about 120° F. should be used in making this test. It is applied in the same manner as cold water, and the reaction depends upon establishing a circulation of the endolymph. In this instance the temperature of the superficially exposed endolymph is elevated above that more deeply situated, and in consequence it rises through the ampulla and external arm of the superior membranous canal, thus stimulating the hair cells of the crista on its utricular half, the side giving off the stronger impulses (Figs. 487 and 488). We will assume that before the test the potentiality or tonus of the utricular half of the crista of each superior canal was 10, and that during the test it was increased to 20 in the tested (right) ear. This portion of the vestibular apparatus having a preponderance of potentiality or pull, turns the eyes away from the tested side, *i. e.*, to the left (slow component), and the cortical correction immediately reverses the movement of the eyes (quick component) to the right, or side being tested. Briefly stated, the warm caloric test causes rotatory nystagmus to the same side. The crista of the horizontal canal is not affected. The result of the test is a rotatory nystagmus to the same side (Plate XIX).

Fig. 492

Ruttin's double irrigator.

In suspected double circumscribed or serous labyrinthitis it may become necessary to test both ears simultaneously to determine which side retains the more functional activity. Ruttin's double irrigator is used for this purpose (Figs. 491 and 492). It consists of two ear speculæ mounted with ball-and-socket joints upon a head-band. The irrigation tips being permanently fixed within the speculæ. The head-band is adjusted to the patient's head, the speculæ inserted in the meatuses, and the water turned on simultaneously into both meatuses. The same amount of water should flow into each ear, as if one receives more than the other in a given time, it will be more cooled and render the test of no value. Both labyrinths must be equally and simultaneously cooled. The water flowing from each should, therefore, be collected and measured as a check upon the accuracy of the experiment. If the amount of water flowing into each ear is equal in quantity and temperature, and flows against the ears within the same period of time, the more sensitive ear will be inhibited (cold caloric) first, that is, the nystagmus will be away from the more sensitive and more nearly normal ear toward the more affected ear.

To avoid nausea and vomiting during the caloric test, have the patient look in the direction of the expected quick component, that is, to the opposite side when cold water is used, and to the same side

when warm water is used. This will induce nystagmus with the minimum of irrigation, as nystagmus is favored by looking in the direction of the quick component, and is suppressed by looking in the direction of the slow component. Before applying the Ruttin's double caloric test, carefully inspect both ears to see if the physical conditions are about the same on both sides. If atresia of one meatus or polypi on one side is present, or a larger amount of heavy secretion is present in one ear than in the other, the accuracy of the test will be affected.

If the caloric, or any other test, causes nausea and vomiting in a case upon which an operation is contemplated, the operation should be postponed until the next day to allow the nystagmus and nausea to subside.

When the tests are made in the office, great care should be taken to avoid inducing nausea and vomiting, as it may be several hours before the patient thus affected can be removed to his home. In using water of about 78° F., have the patient look toward the opposite side, the direction of the expected quick component, and watch for the first appearance of the nystagmus, and stop syringing when it appears. In making the qualitative test, it is only important to know if nystagmus can or cannot be induced; that is, is the vestibular apparatus functionating or dead? In making the quantitative test it is necessary to know how difficult it is to arouse the vestibular apparatus to functional activity. The intensity and duration of the nystagmus are also factors in the quantitative test.

FIG. 493

The fistula test, causing irregular nystagmatic movements of the eyes.

Clinical Significance of the Caloric Test.—In labyrinth disease the caloric test is used for two purposes, namely, (*a*) as a qualitative test to determine whether the labyrinth is responsive to stimulus or is irresponsive to it. If responsive to stimulation there is usually no immediate danger of the infection extending to the meninges or brain.

If it is not responsive to the cold stimulus the infection of the meninges and brain could not be anticipated until such infection had actually occurred, as the labyrinth is dead, and cannot give rise to vestibular symptoms, as vertigo, nausea, vomiting, and ataxia. In such a case, with a recent vertiginous attack, followed by complete deafness and negative caloric and fistula tests, an immediate labyrinth operation may be advised to prevent the extension of the infectious process to the meninges and brain.

(*b*) The second object of the caloric test in labyrinth disease is to make a quantitative test of the vestibular apparatus. The amount of cold water used, the time consumed in its application, and the duration of the induced nystagmus should be noted. If nystagmus is induced by a small amount of cold water, in a short time, and the nystagmus endures for nearly the normal number of seconds, the vestibular apparatus is but slightly affected and operation on the labyrinth is strongly contra-indicated. If, however, it requires long irrigation to produce but slight nystagmus of short duration, the vestibular apparatus is nearly destroyed, and close watch and repeated tests should be made to determine whether the destruction is progressing. If so the attending otologist should be prepared to do a labyrinth operation should the indications arise. If the caloric test elicits no vestibular response the labyrinth is either inhibited, as in severe serous labyrinthitis, or is destroyed, as in acute diffuse manifest suppurative and latent suppurative labyrinthitis. In serous labyrinthitis operation is not indicated. If acute diffuse manifest or latent suppurative labyrinthitis is shown, an operation may be advised. The indications are suggested in this connection to show the clinical value of the qualitative and quantitative tests.

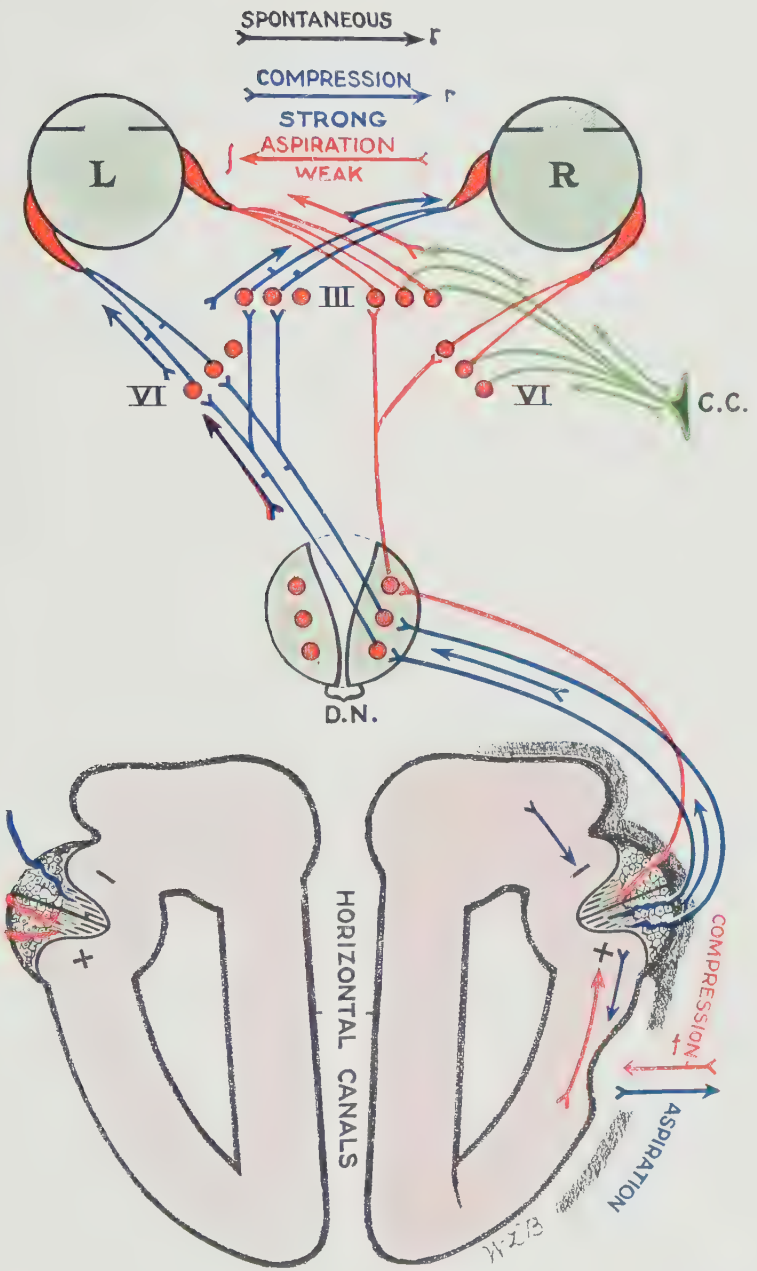
The Fistula Test.—The fistula test is usually made by compressing air within the external meatus with a Politzer bag fitted with a six-inch rubber tubing and an olive-shaped ear tip which is inserted into the meatus (Fig. 493). Either compression or suction may be used, though compression is usually practised. The nystagmus is

PLATE XXI

Diagrammatic Illustration of the Mechanism of Induced Fistula Nystagmus, the Fistula Being in the External Limb of the Horizontal Canal and Posterior to the Facial Ridge.

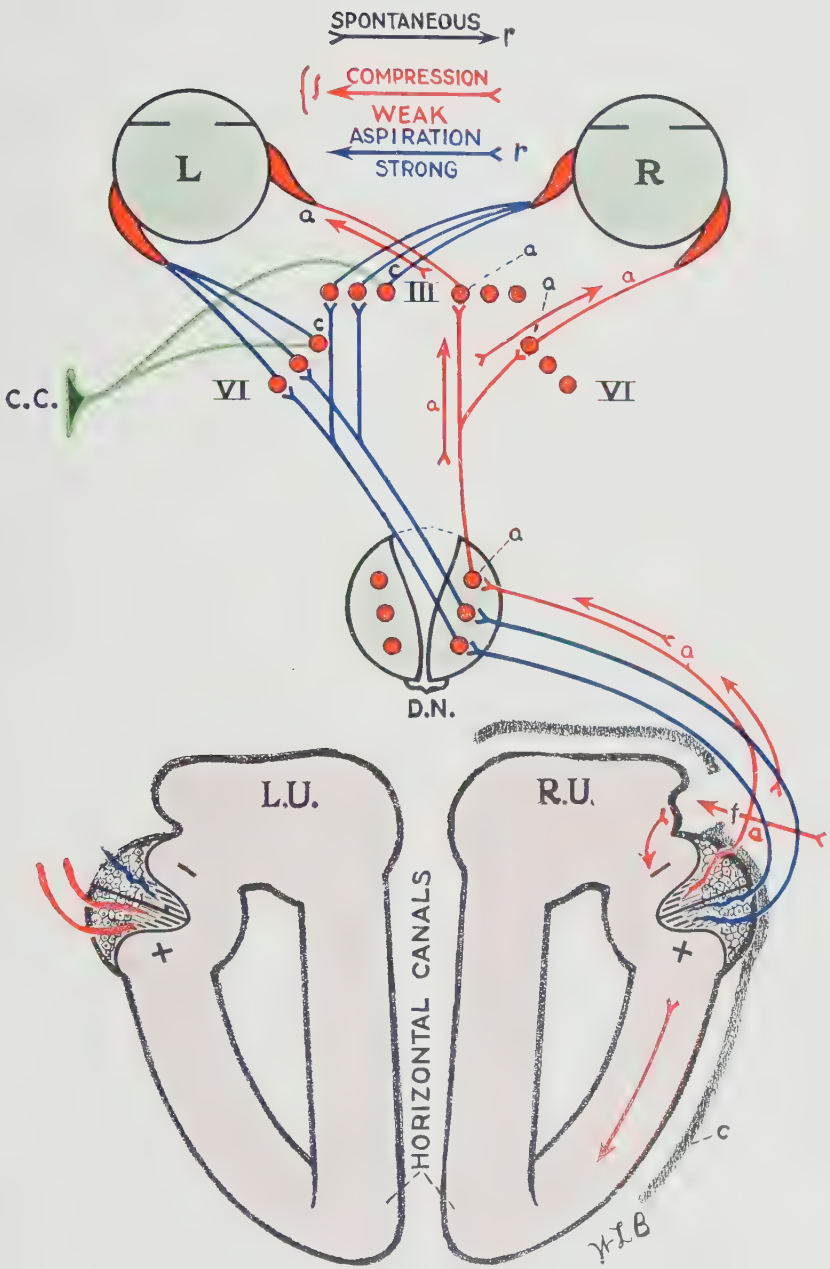
The compression is applied at the fistula (*f*) which causes an endolymph current from the canal to the utricular –against the canal—half of the crista. This half when stimulated gives off nervous impulses twice as strong as those given off from the utricular half. The course of the impulses is indicated by the two blue lines to the abductor muscles of the left eye (*L*), and to the adductor muscles of the right eye (*R*). A slow conjugate movement of both eyes to the left results (slow component). The right cortical centre (*C.C.*) immediately gives off an impulse which traverses the right third and sixth oculomotor nuclei, from whence it is conveyed to the adductor of the left eye and the abductor muscle of the right eye, thereby inducing a quick conjugate movement of both eyes to the right. The nystagmus thus induced is horizontal, strong, and to the right, and is symbolized thus $\xrightarrow[\text{strong}]{}$r. It is strong and more enduring because it emanated from the stronger half of the crista ampullaris.

PLATE XXI



Fistula of Horizontal Canal, with Spontaneous and Induced Nystagmus.

PLATE XXII



Fistula of Oval Window, with Spontaneous and Induced Nystagmus.

usually to the same side (Plate XXI). As a fistula of the oval window or promontory is a more serious condition than fistula of the horizontal canal, it is of some interest to determine its location. Hence, when the induced nystagmus is to the opposite side the fistula is probably in the oval window, or somewhere anterior to the descending portion of the facial nerve, the region of greater danger. If the induced nystagmus is to the same side, the fistula is probably situated in the horizontal canal posterior to the facial nerve, the region of lesser danger. If the fistula is over the promontorium and opens into the vestibule, the nystagmus is usually to the opposite side, as the flow of endolymph caused by compression is from the utricle through the ampulla to the smooth end of the horizontal canal, and the crista is stimulated on its utricular side, which pulls the eyes (slow component) to the same side, and the cortical reflex immediately following, pulls the eyes to the opposite side (quick component), thus constituting nystagmus to the opposite side (Plate XXII). Aspiration will reverse the direction of the flow of endolymph, and the nystagmus will be reversed in direction. The location of the fistula does not absolutely determine the direction of the flow of endolymph, hence the nystagmus may be to the same or to the opposite side, regardless of the location of the fistula.

If the fistula is in the external limb of the horizontal canal and the compression test is made, the flow of endolymph is from the canal through the ampulla to the utricle. The impact of the endolymph is against the canal half of the crista, which emits nervous impulses that traverse the path indicated by the blue lines shown in Plate XXI. In this illustration the right horizontal canal is fistulous, and the impulses arising in the canal half of the crista are transmitted to the right Deiters' nucleus, and thence to the left third and sixth oculomotor nuclei, and from there to the abductor muscles of the left eye, and the adductor muscles of the right eye. This induces a conjugate movement of both eyes to the left (slow component), a corrective cortical impulse is immediately excited in the right cortical centre which is transmitted to the right third and

---

PLATE XXII

Diagrammatic Illustration of the Mechanism of Induced Fistula Nystagmus in Fistula of the Oval Window.

Compression is applied at *f*, a fistula at the oval window which communicates with the vestibule. This causes a flow of endolymph backward against the utricular or weaker half of the crista. The impulse aroused by the impact traverses the course indicated by the single red line (*a*) and finally reaches the adductors of the left eye and the abductors of the right eye, thereby inducing a slow conjugate movement of both eyes to the right (slow component). A corrective cortical impulse is immediately liberated in the left cortical centre (*C.C.*) and is transmitted to the third and sixth oculomotor nuclei (*III^c*, *VI^c*) and thence to the adductors of the right eye and abductors of the left eye, thereby inducing a quick conjugate movement of both eyes to the left (quick component). The compression test applied to the right ear, with fistula anterior to the crista, is followed by induced weak nystagmus to the left which is symbolized thus: $\underset{\text{weak}}{\curvearrowleft}$ l.

sixth oculomotor nuclei, and thence to the adductor muscles of the left eye and the abductor muscles of the right eye, thereby producing nystagmus to the right. In fistulous cases, in which the labyrinth is not destroyed, we are dealing with circumscribed labyrinthitis. These cases usually respond normally to the turning and the caloric tests. The clinical significance of the positive fistula reaction is that the vestibular apparatus is still functionating, and an operation is not indicated. The significance of a negative fistula test is *nil*, as a fistula may or may not be present.

Hindrances to the Fistula Test.—The presence of cholesteatoma, cerumen, large polypi, atresia, or granulations may interfere with the application of this test, as either condition may block the fistula and prevent the action of the compressed air upon the portion of the membranous labyrinth exposed by the bony fistula.

The caloric test is less reliable than the fistula test when the endolymph is coagulated. If, therefore, the caloric reaction is negative, and the fistula test positive, it may be taken as a sign that the endolymph is coagulated. It may be necessary to compress the bulb several times to induce nystagmus.

Nystagmus induced by the fistula test is an indication of fistula, though the absence of nystagmus is not a sure sign that fistula is not present. When the reaction is present "fistula symptom" is said to be positive. The fistula test is negative when the labyrinth is totally destroyed, even though a large fistula is present. The bony fistula may be so small it cannot be seen at the time of the mastoid operation, yet still be large enough to give rise to fistula symptoms upon compression. When present we say, fistula symptom is plus or positive; when absent we say, fistula symptom is minus or negative.

The Clinical Significance of the Fistula Test.—The compression and aspiration tests are of diagnostic value under the following conditions:

1. When there is a bony defect or fistula without involvement of the membranous labyrinth the fistula test is positive, *i. e.*, nystagmus is induced. The caloric and turning tests also give normal positive reactions. The prognosis in these cases is good and the usual therapeutic and surgical measures demanded for the cure of the middle-ear and mastoid disease are indicated. The labyrinth needs no other treatment.

2. The spontaneous nystagmus of acute diffuse manifest suppurative labyrinthitis with fistula is not increased by the compression, as the labyrinth is totally destroyed. The spontaneous nystagmus present is due to the sudden loss of tonus in the destroyed labyrinth or, rather, to the sudden loss of equality of tonus between the two labyrinths. The prognosis is grave, and the labyrinth may, and indeed often should, be operated as soon as all hearing is lost.

3. In diffuse latent suppurative labyrinthitis (chronic stage of acute diffuse manifest suppurative labyrinthitis) the fistula test is sometimes positive in reaction. That is, the cochlea may be totally destroyed,

while the vestibular apparatus is nearly, though not quite, destroyed. This is explained by the fact that the cochlear portion of the eighth cranial nerve is more easily destroyed by suppurative inflammation than the vestibular portion. The prognosis is grave in these cases. That is, there is always a probability of the infectious process extending at some subsequent time to the meninges or cerebellum. Cerebellar abscess often follows chronic diffuse latent suppurative labyrinthitis.

4. In circumscribed labyrinthitis without bony fistula, the fistula test is negative in reaction. Such cases should always be tested, because the absence of the fistula symptom is of great diagnostic and prognostic value. These cases are more favorable than those with fistula.

The Galvanic Test.—According to J. R. Fletcher, the work done by Alexander, Neumann, Frey, Hammerschlag, and Barany, on nystagmus by galvanization, is not conclusive. According to Neumann, galvanism may produce nystagmus when the labyrinth is totally destroyed, which, he says, is due to the direct galvanization of the vestibular nerve and centrum. The kathode (minus or negative pole) affects the nervous tissue more than the anode (the plus or positive pole) and induces nystagmus to the same side (Plate XXIII).

According to Neumann, if there is no reaction from either the turning, caloric, or galvanic tests the centrum is destroyed. If only the labyrinth is destroyed, and the vestibular nerve and centrum are normal, the turning and caloric tests will not produce nystagmus; whereas the galvanic test may produce it, as the electric current acts directly upon the vestibular nerve and centrum.

Neumann regards Brunnung's theory, namely, that the kathode produces a flow of endolymph in the canals by galvanism, as unproved and improbable, as he has shown that nystagmus is produced by galvanism when the labyrinth is completely destroyed. This he regards as being due to the galvanism of the vestibular nerve, or of Deiters' nucleus. Galvanic induced nystagmus is always rotatory and toward the kathode or away from the anode.

Galvanism after a recent acute suppurative destruction of the labyrinth (diffuse manifest suppurative labyrinthitis) may cause marked induced nystagmus by direct action on the vestibular nerve. The response to galvanism gradually diminishes in intensity as the vestibular nerve degenerates, and finally, when the centrum is also completely degenerated, nystagmus can no longer be excited by it.

Bipolar irritation with one electrode over each mastoid process requires from 2 to 5 m.a. of current, while the monopolar application with one electrode to the mastoid, the other in the hand, requires 20 to 25 m.a. of current to produce nystagmus in normal cases.

The quick component of induced galvanic nystagmus is always toward the kathode (negative pole), whether the applications are bipolar or monopolar. If the kathode is applied to the right mastoid process, the nystagmus will be to the right. If both the kathodal

and anodal electrodes are held in the hands, both ears being normal, there will be no nystagmus. If, however, either labyrinth is diseased but not totally destroyed, and both the kathodal and anodal electrodes are held in the hands, the nystagmus will be toward the healthy side, as this is the more sensitive of the two, has the greater tonus, and pulls the eyes to the opposite or diseased side (slow component), after which the quick component of the induced nystagmus occurs to the same or healthy side. The galvanic test alone should not be used to determine whether the labyrinth is still functionating, as it will induce reaction when the labyrinth is totally destroyed, provided the vestibular nerve and centrum are intact.

The kathode applied in front of the right ear produces a stimulation of the crista ampullares and Deiters' nucleus on that side, and causes a slow conjugate movement of the eyes to the left, and the cortical correction to the right (same side) immediately follows (Plates XXIII and XXIV). That is, before the kathode current is applied the tonus impulses emanating from each labyrinth is equal. The kathode stimulation increases the tonus on the right side, whereas it remains the same on the left side. The impulses, therefore, were stronger on the right, and according to Hoegyes' law, the eyes were turned to the left or opposite side (slow component of the nystagmus), and the cortical correction immediately occurs, and the eyes are turned to the right side (quick component of the nystagmus).

The anode applied to the right ear produces an inhibition of the cristæ and Deiters' nucleus on that side, and causes a slow conjugate movement of the eyes to the right, and the cortical correction to the left (opposite) side immediately follows (Plate XXIV).

That is, before the anode is applied to the right ear the tonus impulses are the same in both labyrinths and centrums. After the anodal current is applied to the right ear, the tonus impulses are inhibited or diminished in the right labyrinth and centrum, leaving a preponderance of tonus in the left labyrinth and centrum, which, according to Hoegyes' law, turns the eyes to the right (slow component) and the

PLATE XXIII

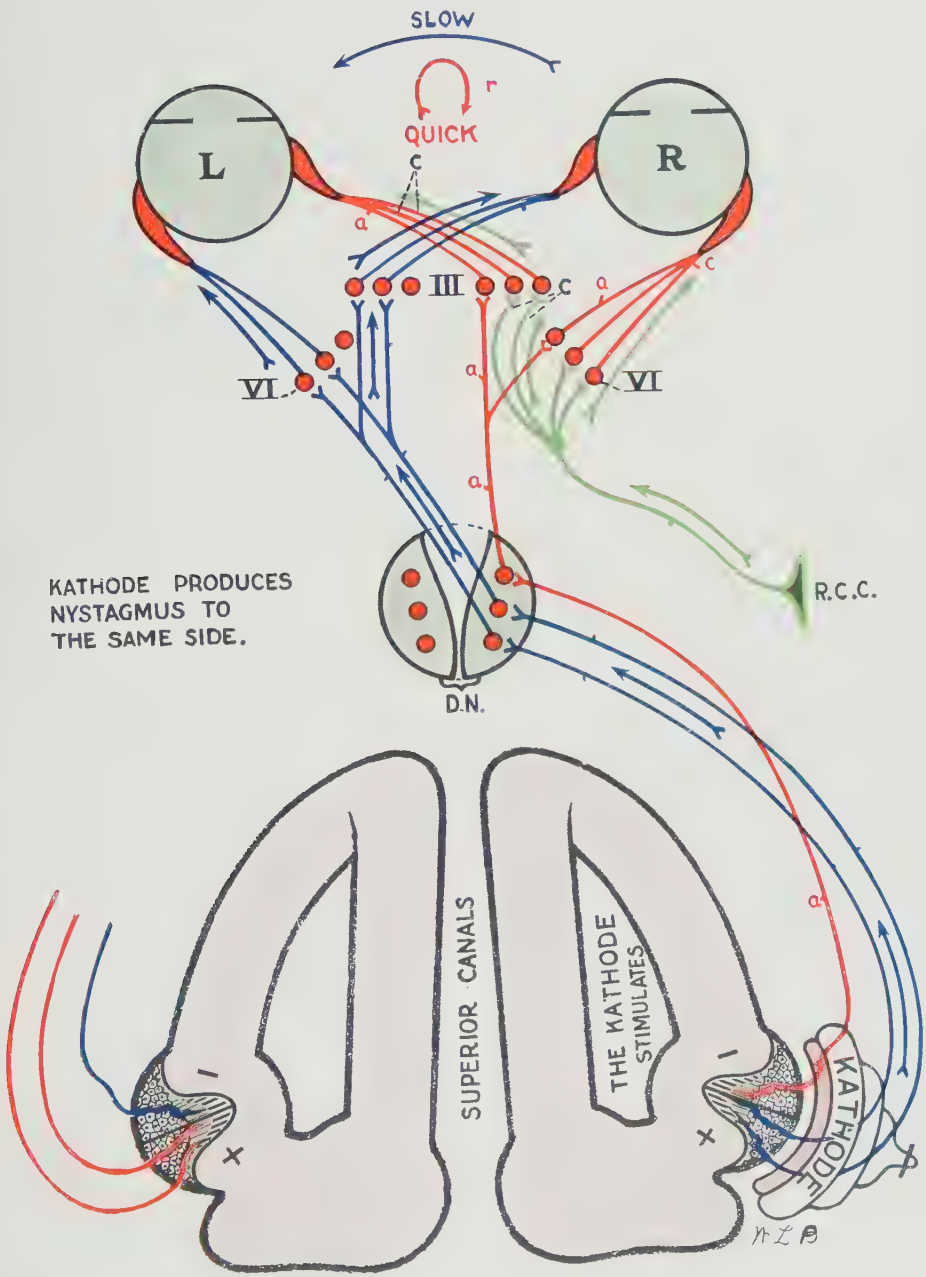
Diagrammatic Illustration of the Mechanism of Induced Kathodal Galvanic Nystagmus.

The kathode or negative pole of the battery is applied to the right ear, the vestibular apparatus of which is stimulated to greater physiological activity. The increased impulses are transmitted through the paths (*bb*, blue lines) to Deiters' nucleus, and thence to the third and sixth oculomotor centres of the left side (*III*, *VI*), and finally to the adductor muscle of the right eye, and to the abductor muscle of the left eye, thereby causing a slow conjugate movement of both eyes to the left (slow component). A corrective cortical impulse immediately arises in the right cortical centre (*R.C.C.*), and is transmitted to the right third and sixth nuclei, and thence to the abductor muscles of the right eye, and to the adductor muscles of the left eye, thereby producing a quick conjugate movement of both eyes to the right (quick component of the nystagmus). The nystagmus is rotatory and to the right or same side, and is symbolized thus: ↷ r.

The kathode induces nystagmus to the same side.

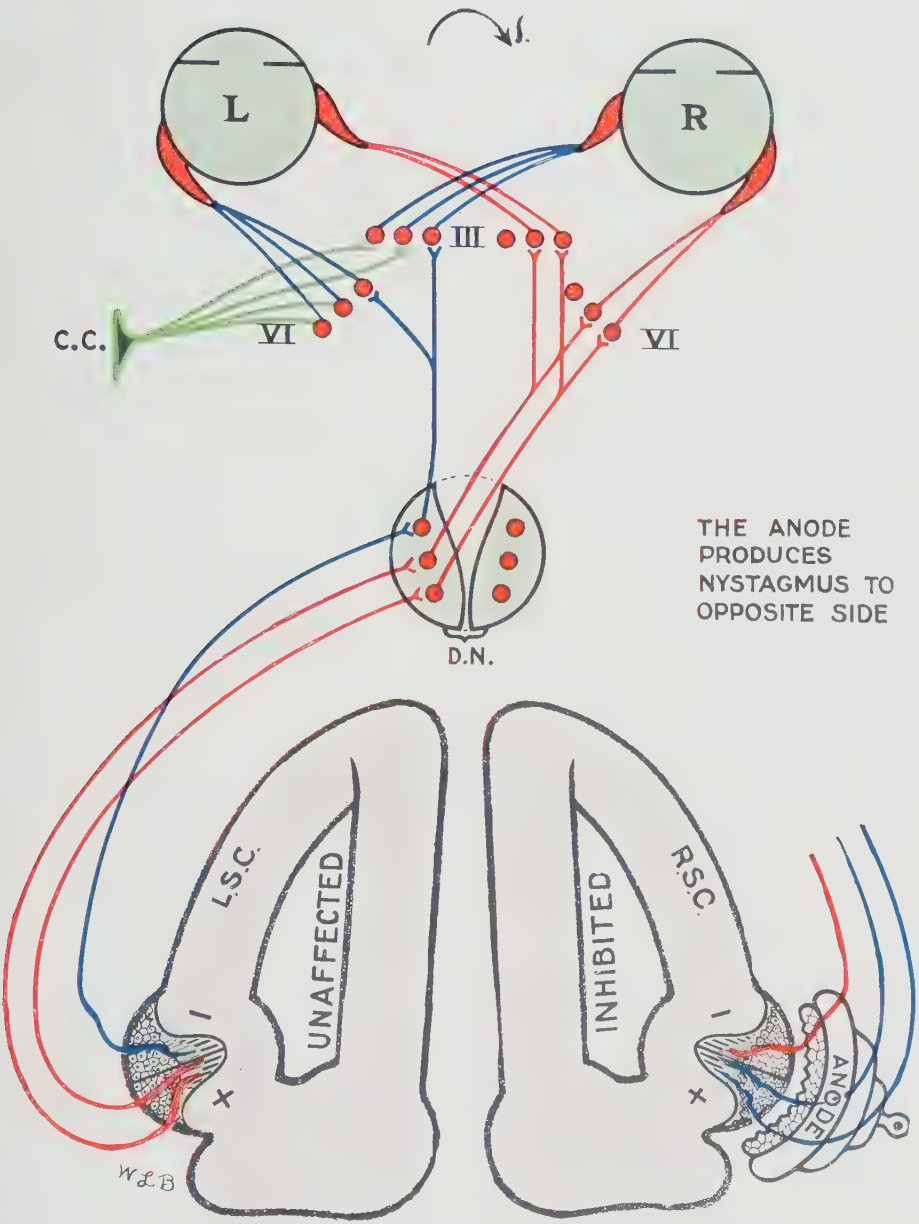
The anode induces nystagmus to the opposite side.

PLATE XXIII



Kathodal Induced Nystagmus.

PLATE XXIV



Anodal Nystagmus to the Opposite Side.

cortical impulse immediately follows and turns the eyes in the opposite direction to the left (quick component).

Clinical Significance of the Galvanic Test.—The galvanic test is generally regarded as of less clinical importance than the other tests, as nystagmus produced by it may not be due to stimulation of the labyrinth, but may be due to stimulation of the vestibular nerve and Deiters' nucleus. Inasmuch as the nystagmus may be due to stimulation of the vestibular portion of the labyrinth or the vestibular nerve and Deiters' nucleus, the test is of doubtful diagnostic value in determining the functional activity of the cristæ. When, however, the labyrinth is known to be totally destroyed the galvanic test is of great value in estimating the degree of extralabyrinthine degeneration. By this test the prognosis of the degeneration of the vestibular nerve and Deiters' nucleus may be estimated from time to time until all response is abolished, an occurrence signalizing the total degeneration of the vestibular nerve and the central nuclei associated in function with it. These nuclei are Deiters', Bechterew's, and the angular nucleus.

Some Clinical Problems.—I will attempt to suggest the clinical significance of the various vestibular tests by reciting the clinical phenomena of a hypothetical case. The ultimate purpose of all clinical and laboratory observations is the amelioration and cure of disease. This is true of labyrinth disease. In the following paragraphs will be found the essential elements for illustrating the clinical application of the various vestibular tests.

(*a*) When the labyrinth of the affected ear is totally destroyed, that is, when there is a total and permanent deafness in the affected ear, and all vestibular (static) function is also lost, there is therefore no hope of restoring either the hearing or the static function, insofar as this labyrinth is concerned. The static function is, however, rapidly adjusted in the extralabyrinthine nervous mechanism of the cerebellum, and need give the clinician no concern. The deafness being forever lost, and the static function being automatically adjusted, neither requires treatment.

(*b*) If the patient still suffers from chronic otorrhea and mastoiditis with recurrent attacks of the same, he needs a radical mastoid operation.

PLATE XXIV

Diagrammatic Illustration of the Mechanism of Anodal Galvanic Nystagmus. When the anode or positive pole is applied to the right ear (*ANODE*) the impulses are suppressed in that ear, while they are unaffected in the opposite or left ear. The impulses from the left ear having a stronger tonus or pull, turn the eyes to the right—trace red lines to abductors of the right eye (*R*) and to the adductors of the left eye (*L*)—thus producing the slow movement or component of the nystagmus. A corrective cortical impulse is immediately liberated in the left cortical centre (*C.C.*), which is transmitted through the third and sixth oculomotor centres (*III*, *VI*) to adductors of the right eye (*R*) and the abductors of the left eye (*L*), which causes a quick conjugate movement of both eyes to the left (quick component of the nystagmus). The nystagmus is rotatory (as all the cristæ are stimulated) and to the left, and is symbolized thus: ↷ l. The anode produces nystagmus to the opposite side. The kathode produces nystagmus to the same side.

(*c*) There also remains another condition of grave clinical significance, namely, chronic latent suppurative labyrinthitis. There are, however, no manifest symptoms of the disease, as both the cochlea and vestibular apparatuses are totally destroyed, and rendered functionless, and extralabyrinthine compensation has occurred and nystagmus is no longer present. The infection in the labyrinth canals may still be present just as truly as it was when the symptoms were manifest, and compensation had not occurred. The infection may therefore "silently" extend to the meninges and brain and cause meningitis or cerebellar abscess. The mortality of meningitis is over 95 per cent., while that of cerebellar abscess is more than 75 per cent. under operation. The great problem presented in these cases is the *prevention* of meningitis and cerebellar abscess, for, if either of these pathological conditions develops, the patient's life is in the gravest danger. The relief of the chronic otorrhea and mastoiditis is of secondary importance, though from the patient's point of view, it may be of the first importance. It becomes the province of the attending otologist to ascertain all the facts in relation to each case, and to advise the patient or his friends as to their true significance, and the course of treatment required to insure the most favorable result.

The clinical problems therefore become resolved into the following alternatives: (1) The radical mastoid operation to cure the mastoiditis and chronic otorrhea. If this is done without reference to the labyrinth, what may be the outcome? Clinical experience has shown that if, after the complete destruction of the labyrinth, the mastoid operation is performed without the radical exenteration of the labyrinth at the same time, the infection within the labyrinth often subsequently extends to the meninges (diffuse suppurative meningitis, leptomeningitis) and results in death. Clinical observation has also shown that if in such cases the labyrinth is operated, life expectancy is increased. I have seen a few cases not operated, or which had only a mastoid operation, subsequently develop meningitis or cerebellar abscess.

Clinical observation also shows that if the radical mastoid operation and labyrinth operation are done at the same time in such cases, before intracranial involvement occurs, the results were almost uniformly favorable. That is, the combined radical mastoid and labyrinth operation is a relatively safe procedure, provided, of course, the operator has mastered the technique, and meningeal involvement has not occurred. If such involvement has occurred the outlook is grave, either with or without an operation.

2. The second alternative is to do the radical mastoid operation, exercising the greatest care to avoid trauma in the region of the promontorium, oval window, and external limb of the horizontal semicircular canal. But even with the greatest care reactionary inflammation is often excited in the labyrinth, with subsequent extension to the meninges. This alternative is therefore not free from danger.

3. The third alternative is to "wait and watch" developments. This is, doubtless, a fascinating thing to do. I remember once watching

a cat play with a favorite pet mouse. All went well for a while, there were no signs of danger—to the mouse. But suddenly the feline nature asserted itself, and poor mousie went to its reward. As previously stated, there are usually no symptoms of progress in latent labyrinthitis, as the labyrinth is destroyed and rendered functionless, and the canals are so small that any acute infection therein could not materially influence the temperature. Any marked elevation of temperature present must be from involvement outside the labyrinth. There is no guide as to the progress of the infection toward the meninges within the labyrinth in such cases; hence it is only when the meninges are actually affected that danger is suspected, and then it is usually too late to avert danger, though prompt operation even then may prevent a fatal issue.

In the case to be cited there was a history of total destruction of the labyrinth attended by a severe vertiginous attack one year previously. There was a total loss of hearing in the affected ear, and chronic otorrhea with acute exacerbations of mastoiditis. These exacerbations had been present since the vertiginous attack one year ago. There has been no giddiness since the original seizure. The patient seeks relief from the chronic otorrhea and mastoid attacks. He incidentally hopes his hearing may be improved.

Of what use may the functional test of the vestibular apparatus be in such a case? Again, we must be guided by past experience, which shows *that so long as there is a vestige of hearing in the affected ear, there is no need for an immediate labyrinth operation.* If there is a remnant of vestibular function left it is obvious that if an acute process lights up in the labyrinth and causes further destruction, there will be symptoms to warn the attending otologist of the impending invasion of the cranial content. There will be giddiness, nausea, disturbed equilibrium, and spontaneous nystagmus to the healthy side. It is obvious that the vestibular apparatus should be tested for functional reactions, as only in cases with such reactions is it entirely safe to "wait" for developments. In cases in which vestibular reaction is wholly lost the first "signs of development" would be the symptoms of either diffuse suppurative meningitis or cerebellar abscess. To "wait for developments" in a case of latent labyrinthitis in which both the vestibular and cochlear functions are wholly destroyed is therefore not devoid of danger.

The same principles of thought apply with equal relevancy to acute diffuse serous, circumscribed, and other forms of labyrinthitis. The presence of vestibular reaction, as shown by the tests, indicates two important safeguards to the patient, namely, (*a*) a remnant of the vestibular nervous apparatus is still present and acting as a barrier to the extension of the infection to the meninges and brain. (*b*) Should this barrier be broken down by the advance of the disease, the event is signalized by the usual vestibular symptom complex, namely, spontaneous nystagmus to the sound side, vertigo, nausea and vomiting, and ataxia. Several hours, or possibly a few days, weeks, or months

might intervene between the onset of these symptoms and the possible intracranial involvement. In any event the surgeon has ample time to perform the labyrinth operation before the intracranial involvement occurs. Jansen claims, however, that meningitis may develop before the complete destruction of the labyrinth. We are therefore not always able to anticipate the onset of the meningitis or cerebellar abscess. The inception of meningitis is usually signalized by a reversal in the direction of the nystagmus.

In view of the foregoing facts it becomes highly important to determine the presence or absence of vestibular reaction, and to also estimate the degree and amount of stimulation required to elicit the reaction. If, in the case just cited, it could be shown by the various tests that vestibular reaction was present (even though the patient was totally deaf) the labyrinth operation should be postponed until a recurrence of spontaneous vestibular reaction, at which time the combined mastoid labyrinth operations should be seriously considered. If vestibular reaction could not be induced by the caloric and fistula tests, it would be extremely hazardous to advise waiting for developments. On the contrary, the patient or his friends should be plainly told the danger of "waiting," and of the comparatively slight danger attending the combined operations. He should also be told of the danger of having the mastoid operation done alone, and that, if the mastoid operation is done, the labyrinth operation should also be done at the same time. Probably the most conservative and rational method of procedure would be to proceed with the mastoid operation, and reserve decision as to the advisability of the labyrinth operation until the presence or absence of fistula is determined. If fistula is present the labyrinth operation, preferably Hinsberg's, should be performed. If fistula is absent a labyrinth operation need not be done. The absence of the fistula makes an extension of the infection to the meninges and cerebellum more improbable; whereas the presence of fistula makes the extension of the infection to the meninges and cerebellum more probable. In other words, the absence of fistula in a case with total destruction of the labyrinth, in which a mastoid operation is indicated, may be taken as a contra-indication to the labyrinth operation; whereas the presence of a fistula may be considered as a factor indicating the labyrinth operation. There can be no hard-and-fast rules in reference to these cases. Each must be analyzed, and the attending surgeon must act in accordance with his best judgment.

Qualitative and Quantitative Estimation.—With the turning test both the qualitative and quantitative estimation of the vestibular function of the horizontal and superior semicircular canals may be determined, though the chief value of this test is in the qualitative estimation. The horizontal canals, when the head is erect, lie in the horizontal plane, or nearly so. The superior canals, when the head is erect, lie approximately in the frontal plane. To bring the latter into the horizontal plane, during the turning test it is necessary to incline the head 90 degrees either backward or forward. The posterior

canals cannot be simultaneously tested by the turning test. This test, therefore, is only applicable to the horizontal and superior canals.

To test the horizontal canals, the patient is seated in a revolving chair, with his head erect, and opaque glasses over his eyes, as suggested by Hans Abels. This is done to prevent fixation of vision, as this might interfere with the expression of the vestibular stimulation. The subject is turned ten complete revolutions in the chair, and suddenly stopped and the eyes observed over the rims of the opaque spectacles. From ten to twenty seconds should be consumed in the ten turnings. The quick component of the resultant induced after-nystagmus will constitute the direction of the nystagmus. If the quick component of the nystagmus is to the right, the nystagmus is said to be to the right. If it is to the left, it is said to be to the left. If the nystagmus is horizontal in direction it is called horizontal nystagmus. If it is rotatory (around the pupillary axis) it is rotatory nystagmus. If it is both horizontal and rotatory it is a combined horizontal and rotatory nystagmus. In combined nystagmus the movements of the eyes are always in the same direction, *i. e.*, if the horizontal nystagmus is to the right, the rotatory nystagmus is to the same side. Only one type of nystagmus, namely, the horizontal, can be induced by stimulating the horizontal canal. As previously stated, it is necessary to incline the head either forward or backward 90 degrees if we wish to test the superior canals by the turning test (Plate XXI).

After allowing the patient to rest a few minutes, he is turned ten times to the left, the turnings suddenly stopped, and the amplitude and duration of the nystagmus noted. If both ears are normal the nystagmus will continue for about forty seconds after turning in each direction. If, however, one labyrinth is destroyed (say the right) and the turnings are to the right, the after-nystagmus will be to the left, and will endure about twelve seconds (Plate XXV). If he is then turned to the left an equal number of times the nystagmus will be to the right, and will endure about half as long as when the turnings were to the right. This is due to two facts, namely, (*a*) the right labyrinth being totally destroyed, is not stimulated by the turnings in either direction; the nystagmus in each instance being due to a stimulation of the respective halves of the crista of the left horizontal canal. After the turnings to the right cease, the flow of the endolymph in the left horizontal canal is in the direction of the turning. *i. e.*, to the right, hence it impacts the crista of the left canal upon its canal aspect, the side of greater physiological activity, and gives rise to nystagmatic movements of the eyes to the left, with a maximum arc of excursions, and with a duration of about twelve seconds. After the turnings to the left, the after-flow of endolymph is to the left, hence it impacts the crista of the left horizontal canal upon its utricular aspect, the side of lesser physiological activity, and gives rise to nystagmic movements of the eyes to the right, with a minimum arc of excursions, and with a duration of about six seconds, or half the duration after turnings in the opposite direction.

Clinical Significance of the Turning Test.—What is the significance of the lessened intensity and duration of the nystagmus after turning to the left? This: in a normal labyrinth the impulses emanating from the canal half or aspect of a horizontal canal are more vigorous, twice as enduring, as those emanating from the utricular aspect or half of the same crista. By turning toward the affected ear, and eliciting an after-nystagmus of twice the duration of that induced by turning toward the sound ear, the vestibular apparatus of the affected side is shown to be wholly unresponsive to such stimulation. The affected ear is destroyed as shown by this test. If the result had been twelve seconds after turning to the right, and nine seconds after turning to the left, it would have shown the right labyrinth to be still functionating, though somewhat crippled. The duration of the nystagmus is greatly reduced after destruction of one labyrinth.

The *quantitative test* of the horizontal canals takes into account the number of turnings required to induce nystagmus, the amplitude of the excursions of the eyeballs, and the duration of the nystagmus. This test is used to determine the degree of imbalance existing between the two static labyrinths. In disease of the labyrinth the quantitative

---

PLATE XXV

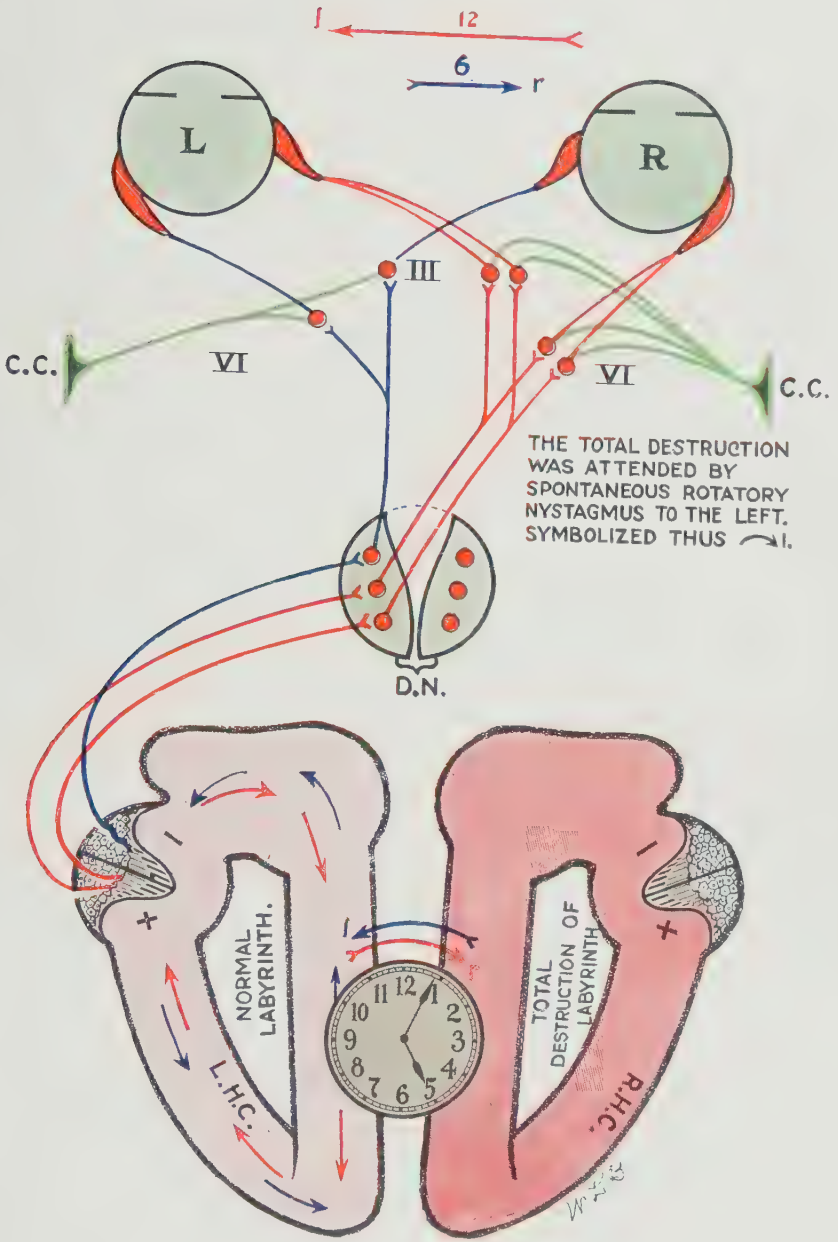
Diagrammatic Illustration of Mechanism of Induced Nystagmus by Turning, the Right Labyrinth Having Been Totally Destroyed Six Months Previously; also Illustrating the Immediate Effect of Total Destruction.

1. The right labyrinth (*R.H.C.*) is represented as having been totally destroyed six months previously. When the turnings to the right have ceased the after-flow of endolymph is to the right (red arrows) in the left normal horizontal canal (*L.H.C.*), and impacts against the canal half (+) of the crista ampullaris which gives off impulses more than twice as strong as those given off by the utricular half of the crista. The path pursued by these impulses is represented by the double red lines to the abductors of the right eye (*R*) and to the adductors of the left eye (*L*), thus producing a slow conjugate movement of both eyes to the right (slow component). A corrective cortical impulse immediately emanates from the left cortical centre (*C.C.*, left side) which causes a quick conjugate movement of both eyes to the left (quick component). The result is a strong horizontal nystagmus to the left, which continues for twelve seconds, and is indicated thus $l \xleftarrow{12''}$.

2. The patient, after resting a few minutes, is turned ten times to the left and suddenly stopped, and the after-flow of endolymph is to the left, as shown by the blue arrows. The impact of the endolymph is against the utricular half of the crista ampullaris left horizontal canal which gives off impulses about one-half as strong as those from the canal half of the crista. These impulses are transmitted along the path indicated by the single blue line to the adductor of the right eye (*R*), and to the abductor of the left eye (*L*), which causes a slow conjugate movement of both eyes to the left (slow component). The corrective cortical impulse is immediately given off from the right cortical centre (*C.C.*, right side), and is transmitted to the abductors of the right eye (*R*) and to the adductors of the left eye (*L*), thereby producing a quick conjugate movement of both eyes to the right (quick component). The resultant nystagmus is weak horizontal to the right, and it endures for about six seconds. It is symbolized thus, $\xrightarrow{6''}$r. The compensation is still extra-labyrinthine. Vestibular compensation has not occurred.

3. When one labyrinth is totally destroyed the discrepancy in duration, etc., between the nystagmus after turning to the right, and after turning to the left should be in the ratio of about 2 to 1. If it is twelve to nine seconds, it shows the apparently destroyed labyrinth as still having some function, *i. e.*, it is not totally destroyed.

PLATE XXV



Acute Total Destruction of Right Labyrinth as shown by the Ratio of 2 to 1.

test is essentially a test of the degree of compensation that has occurred. The test is made in the same manner as the qualitative test, except that instead of turning the patient ten times in the chair, he is turned twice, suddenly stopped, and the eyes observed behind the opaque glasses. If this fails to induce nystagmus he is turned three or four times, and the eyes again observed. If this fails he is turned five or six times, and so on, until nystagmus is induced. Note is made of the intensity and duration and the number of turnings required to induce the nystagmus. After turning in one direction the patient is allowed to rest a few minutes to regain static equilibrium, and he is then turned in the opposite direction a sufficient number of times to induce nystagmus. The number of turnings, intensity, direction, and duration are again noted and compared with the data of the opposite labyrinth. If the duration of the nystagmus after turning toward the sound ear is more than half as long as that induced after turning toward the affected ear, the vestibular apparatus of the affected ear is either normal or only partially destroyed. A labyrinth operation is contraindicated, even if the hearing in that ear is totally destroyed. The meninges and brain are still protected by an anatomical, as well as an exudate barrier, from invasion. Should these barriers become destroyed, the destruction will be signalized by a recurrence of spontaneous nystagmus, nausea, vomiting, and ataxia; warnings which usually give the attending otologist ample time to perform the labyrinth operation, if he so chooses, before the cranial content becomes involved.

The tests are made, not so much to show whether or not the vestibular apparatus is diseased, as to show whether or not it is (*a*) partially, or (*b*) wholly destroyed. If it is only partially destroyed an operation is not indicated. If it is wholly destroyed a labyrinth operation may be and often should be done.

PATHOLOGY OF LABYRINTHITIS

The pathology of labyrinthitis, as given by various European writers, and as presented by Braun and Freisner in their work on "The Labyrinth," may be summarized as follows:

The infective microörganism is usually identical with that presented in the acute otitis media and mastoiditis, or in the acute exacerbation of the chronic otitis media and mastoiditis giving rise to the labyrinth inflammation. In rare cases the labyrinthitis is secondary to mumps and to cerebrospinal meningitis, and the infective microörganism in these cases is that of the primary disease.

The Avenues of Infection.— Several avenues of invasion have been recognized and chief among them are the following:

(*a*) Fistula of the horizontal semicircular canal (vestibular).
(*b*) The oval window (vestibular and cochlear).
(*c*) The round window (cochlear).
(*d*) Fistula of the promontory (cochlear).

(*e*) Fistula of the cochlea at the tympanic orifice of the Eustachian tube (cochlear).

(*f*) Fistula of the facial canal (vestibular). (Grunert.)

(*g*) Through the circulation, as in mumps, though in this disease the infection may extend through the sheath of the facial nerve.

The *most frequent site* of fistula is the external limb of the horizontal canal.

The next most frequent site is the oval window. The *most dangerous site* of fistula is the oval window. As the destructive process is more extensive in cases infected *via* the oval window, the histological findings are more abundant in this type of case; that is, more of them go to postmortem than those infected *via* the horizontal canal, though infection by this route occurs much more frequently.

Changes in the Oval Window.—The following changes may be observed through the meatus before, as well as during, the mastoid operation:

(*a*) Destruction of a part or the whole of the annular ligament of the foot-plate of the stapes.

(*b*) Destruction of a part or whole of the foot-plate of the stapes.

(*c*) Displacement of the foot-plate of the stapes.

(*d*) Destruction of the cartilaginous and bony margin of the oval window.

(*e*) Either pus or granulations may be present in the oval window.

Changes in the Round Window.—As the round window is hidden in the recess at the posterior margin of the promontory, pathological changes cannot be observed through the external auditory meatus before operation.

Changes in the Static Labyrinth.—The pathological changes in the static labyrinth may range anywhere from hyperemia, serous exudate, purulent infiltration, round-cell infiltration, and subsequent organization, to destruction of the bony capsule of the labyrinth.

The pathological process may be diffuse or circumscribed. If circumscribed, it may be limited to the immediate vicinity of the fistula, especially if the fistula is in the horizontal canal. Indeed, many cases of fistula in this region are observed in which the static labyrinth is but little disturbed in function, and the cochlea not at all disturbed. In the latter class of cases there may be congestion of the endosteum lining the bony canal, and of the membranous canal, *via* the connective-tissue bands uniting it to the endosteum. In other cases there is round-cell infiltration in the membranous canal, and in the connective-tissue supports in the immediate vicinity of the fistula. In others the perilymph spaces in the immediate neighborhood of the fistula may be filled with round-cell infiltration, which may subsequently become organized into dense connective tissue, thus permanently blocking further extension of the infective process. In some cases the cristæ of the horizontal and superior semicircular canals are either congested or infiltrated with round cells.

Infection through the Oval Window.—According to Alexander, when a low-grade infection causes a slow perforating ulcer of the liga-

mentum ovale, the purulent secretion accumulates on the inner surface of the foot-plate of the stapes from which it gradually dissolves and drops into the cisterna perilymphatica vestibuli, the lymph space in immediate anatomical communication with the oval window. The cisterna perilymphatica is separated from the utricle by a heavy connective-tissue septa which protects the utricle from infection except in severe and active cases of otitis media with perforation through the oval window. In severe active infection this barrier does not prevent the extension to the utricle; indeed, the infection in such cases is usually diffuse and involves the entire membranous labyrinth. In the mild cases, the infection is often quite effectually shut off from the perilymph spaces of the ampulla, and is limited to the cisterna perilymphatica vestibuli, though it may extend to the ampulla and invade the perilymph spaces and the semicircular canals. The infection may be limited to the perilymph, or it may also involve the endolymph spaces, as previously stated.

Circumscribed cochlear inflammation is usually limited to the cisterna perilymphatica vestibuli and the first half of the basal whorl of the cochlea. According to Ruttin this limitation is due to the mild inflammation causing the perforation of the oval window as stated in the preceding paragraphs. The pus accumulates on the inner surface of the foot-plate of the stapes and gradually dissolves and falls through the perilymph into the cisterna perilymphatica vestibuli, and into the beginning of the scala vestibuli of the basal whorl of the cochlea, where it rests on Reisner's membrane.

Ruttin has shown that when the infection extends (by rupture) from the cisterna perilymphatica vestibuli to the endolymph spaces, it usually invades the deeper ones, as the saccule, canalis reuniens, cæcum vestibulare, or the vestibular portion of the ductus cochlearis. If the perforation is in the round window the infection gains entrance to the scala tympani of the basal whorl of the cochlea, where, if the infection is mild, it may remain circumscribed, or if it is severe, it becomes diffuse and destroys the entire labyrinth.

With the same grade of inflammation, a perforation through the oval window is more serious than a perforation through the round window. In the first instance both the vestibular and cochlear apparatuses may become involved; whereas in the second only the cochlea may be involved.

Circumscribed labyrinthitis is, however, generally found in the perilymph spaces of the horizontal semicircular canal. The connective-tissue barriers at the mouth of the ampulla prevent the extension of the infection to the vestibule. Cholesteatoma may cause a gradual erosion of the bony capsule of the canal and set up a perilabyrinthitis, which extends to the endosteum, and from thence to the fibrous-tissue bands uniting the endosteum and the membranous canal. The endosteum and connective-tissue bands become swollen and infiltrated, and completely wall off the perilymph spaces beyond the localized area of infection. The contiguous portion of the membranous canal may also become swollen and infiltrated, thus

localizing the area of infection. Small abscesses may form in the membranous wall, which, thus weakened, may rupture and admit infection to the endolymph and membranous canal. This portion of the canal being swollen and infiltrated, may confine the infection to this area, or it may become diffuse.

Any portion of the bony capsule may be the site of fistula, though, as previously stated, the external limb of the horizontal canal is the most frequent location. Fistula in the posterior canal is difficult to locate even when present, as the canal is very deeply situated.

Mechanical Pathological Changes in Diffuse Purulent Labyrinthitis.—According to Alexander, the following mechanical pathological changes occur in diffuse purulent labyrinthitis:

(*a*) Tearing Reissner's membrane.

(*b*) Destruction of neuro-epithelial cells.

(*c*) Displacement of the membrana tectoria.

Inflammatory Pathological Changes in Diffuse Purulent Labyrinthitis.—(*a*) Exudate of pus in the perilymph and endolymph spaces.

(*b*) Dilatation of the bloodvessels.

(*c*) Soft tissues infiltrated with round cells and serum.

(*d*) Swelling and necrosis of epithelial cells.

(*e*) Suppuration may extend into the aquaductus cochleæ and vestibuli.

(*f*) The spiral ganglion, nerve fibers in the modiolus, and the internal auditory canal, as well as the lymph spaces may be involved.

(*g*) The fibers of the eighth nerve may be degenerated.

Bone destruction may result from the formation of granulations on the endosteum of the bony canals, and from the formation of an abscess in the subarachnoid space at the fundus of the internal auditory meatus over the area cribrosæ (Politzer and Lange), and from an embolus of the internal auditory artery. The soft tissues may also be necrotic in these conditions.

By the term "caries," we refer to the superficial death of tissue, as in ulceration; whereas by the term "necrosis," we refer to death of tissue *en masse*. Both caries and necrosis may be present in diffuse suppurative labyrinthitis.

Caries results from an inflammation usually beginning in the membranous labyrinth, and if the inflammation is not intense, only caries or superficial destruction occurs. If, however, the inflammation is intense, destruction *en masse* or necrosis of the tissue occurs. This process starts as a perilabyrinthitis (inflammation of the bony capsule), and of the periosteum lining the bony canals. The granulations extend into the Haversian canals of the bone and destroy the bone, the destroyed bone being replaced by granulation tissue. The Haversian canals become more and more spongy or porous. The spaces thus created become filled with granulation tissue, gelatinous intercellular substance, and adventitious bloodvessels. The bone becomes softened and decalcified.

Necrosis of the bony capsule is due to embolus or destruction of the internal auditory artery, which has but slight anastomosis with

the middle-ear vessels. Inasmuch as it is practically an end-artery its obstruction by embolus results in death of bone *en masse*. An abscess in the internal auditory canal may compress the artery and cause death or necrosis of a large portion of the bony capsule of the labyrinth. In scarlatinous otitis media the inflammatory reaction is often very intense, and results in necrosis of the soft tissues of the labyrinth, which in turn destroys arterial branches, which in turn destroys branches of the internal auditory artery, and is followed by necrosis of portions of the bony capsule.

The necrotic bone is circumscribed and bounded by carious or ulcerating bone tissue which is gradually eroded and absorbed, until eventually the necrotic bone lies free as a sequestrum in a bed of granulation tissue (Braun and Freisner). The sequestrum usually includes the cochlea, as it is almost exclusively supplied by the internal auditory artery; whereas, only a portion of the capsule of the static labyrinth is supplied by it.

If the sequestrum includes the inner wall of the tympanum (promontorium) it may be thrown off through the middle ear and external meatus. If it is enveloped in healthy bone it must remain *in situ* until the enveloping healthy bone is destroyed or surgically removed. The sequestrum cannot be absorbed, as it contains no bloodvessels. Even though only a portion of the bony capsule is necrotic the whole nervous labyrinth is usually destroyed.

Necrosis and exfoliation of the bony capsule of the labyrinth does not occur in all cases, nor, indeed, in most cases of diffuse suppurative labyrinthitis. In many the round-cell infiltration becomes organized into fibrous tissue which fills the entire labyrinth, or the labyrinth channels may become filled with hyperplastic or bony tissue, which has its origin in an irritation of the endosteum.

GENERAL DIAGNOSIS[1]

Barany's Fixation Apparatus.—When spontaneous nystagmus exists, the degree of involvement of the labyrinth may be accurately estimated by the responsiveness of the vestibular apparatus to added external irritation. Before irrigating, a fixation point must be found where the nystagmus ceases, or is minimal. For this purpose, Barany has devised an instrument which is made fast to the head of the patient by a headband. A metal plate with a dial, from which a metal rod extends at right angles, bearing a shorter pendant rod, which can be moved back and forth and from side to side, form the essential parts of this instrument. The patient fixes his eyes upon the pendant rod, which is moved to a point where the nystagmus is least or altogether disappears. When this point is determined, the affected ear is gently

[1] The section on General Diagnosis is prepared by Dr. J. R. Fletcher. The preceding portions and the remainder of this chapter on the labyrinth and its diseases, as it appears in this edition, is written by the author.

irrigated with cold water. If this induces an additional reaction, the nystagmus will reappear or increase while the patient looks at the fixation point.

In severe cases of spontaneous nystagmus, this method of examination must be very exact, as the correct diagnosis depends largely on the caloric test in conjunction with Barany's fixation apparatus. If, for instance, the history of a case is not clear, and the spontaneous nystagmus is to the diseased side (extralabyrinthine), and the nystagmus is not reinduced or increased by this test, meningitis or cerebellar abscess must be present. If a positive reaction is induced, neither of these diseases can exist, as not a single case of either has been found up to this time in which the labyrinthitis has remained circumscribed. (I have elsewhere stated that Jansen claims meningitis has occurred before complete destruction of the labyrinth.

Nystagmus in Circumscribed Labyrinthitis.—In circumscribed labyrinthitis the following classification must be observed:

I. Erosion with fistula.[1]
   (*a*) Erosion with normal irritability.
   (*b*) Erosion with diminished irritability.
II. Traumatic with traumatic neurosis.

Erosion with Fistula.—Circumscribed disease of the labyrinth is characterized by *attacks of vertigo* and nystagmus, and always by some impairment of hearing. Erosion with fistula is always secondary to disease of the tympanic cavity, which not only involves the drum and ossicles, but often also the bony median tympanic wall. The form of circumscribed labyrinthitis of greatest interest in the study of nystagmus is erosion with fistula. This form may remain circumscribed for a long time, or it may become diffuse, or it may heal with the formation of connective tissue over the fistulous opening, gradually ossifying and closing it.

Barany described vertigo as being of two kinds:

1. That which occurs without external cause.
2. That which occurs with external cause.

1. This type comes on at any time and under all circumstances, while the patient sits quietly at a desk, during a meal, while walking, and even during sleep. Such attacks are, as a rule, quite severe and of long duration. They may last from one-half to several hours. The nystagmus is of the spontaneous rotatory type, the quick component of which is directed to the diseased side. There may also be a weaker nystagmus, the quick component of which is directed toward the sound side. Nystagmus occurring under these conditions is characteristic of circumscribed labyrinthitis. The accompanying phenomena, nausea, vomiting, and the sensation of movements of objects, are quite severe.

2. The external causes of the second form of vertigo are rapid movements of the head, stooping forward, rising, inclining the head back-

[1] Irrigation is dangerous when a fistula through the lateral labyrinth wall exists. Unless the water is very gently passed into the ear, pus may be carried to new points, easily converting a circumscribed into a diffuse labyrinthitis.

ward, and especially toward the shoulder of the diseased side, and going from a hot to a cold room, or *vice versa*. Usually these attacks are not severe and their duration is short, lasting only from a few seconds to a few minutes. The nystagmus is rotatory and directed to the diseased side; vomiting, as a rule, is absent. In the interval between the attacks of nystagmus the patients frequently feel perfectly well, and often show no signs of nystagmus or disturbances of equilibrium.

Symptoms of cochlear disease (tinnitus, marked deafness, loss of bone conduction, and of hearing for the tones of the upper register) are very often associated with either form of these attacks. Both forms occur in cases of erosion of the labyrinth in the course of acute or chronic suppurative otitis media.

Between attacks it is usually possible to induce weak rotatory and horizontal nystagmus in response to both caloric tests. Occasionally the rotatory nystagmus, following turning while the head is inclined 90 degrees forward, will last longer than the normal horizontal nystagmus. The latter always denotes a pathological condition.

In case of diminished irritability there is a moderate degree of rotatory and horizontal nystagmus (a combined spontaneous nystagmus), to both right and left, which is usually strongest to the diseased side, but sometimes to the sound side. Upon inclining the head backward, that is, placing the superior canal in the horizontal plane, vertigo and rotatory nystagmus take place in about 50 per cent. of the cases. The quick component of the nystagmus is directed to the diseased side; the duration is about fifteen seconds. After waiting ten minutes, the same procedure will give a like result. Compression and aspiration in the auditory canal and middle ear produce no nystagmus, though eye movements occur, but are only minimal. The response to cold water is quite typical, as to direction and plane of the nystagmus, but it is very weak. Turning ten times in the direction of the diseased side, head erect, produces horizontal after-nystagmus to the opposite side, of about thirty seconds' duration, a reduction of one-fourth the normal average.

Fistula is a consequence of erosion of the labyrinthine wall. When the bony wall has been broken through, the membranous canal containing the endolymph is exposed. Pressure upon it causes a displacement or flow of endolymph. Suction causes a return flow. Movements of the eyes of a nystagmatic character, produced by compression and aspiration of air in the external auditory canal and in the tympanic cavity, are the sign of fistula, and aid in differentiating labyrinthitis from cerebellar abscess.

When the vestibular apparatus responds normally to the caloric test, compression and aspiration of the membranous canal through the fistula in the bone cause long, slow movements of the eyes, or an active nystagmus of some seconds' duration. When the response to the caloric test is partly or completely lost, very slight movements of the eyes may be observed during the test for fistula. It is also true that

exceedingly small movements of the eyes by compression and aspiration have been observed by Barany, Hennebert, and many others, in the absence of fistula. In such cases the response of the vestibular apparatus to heat and cold is normal. The latter fact excludes fistula, as (see above) the movements of the eyes must then be very long and slow. The directions of the movements differ in different cases. The movements which result from the compression are, however, always in the opposite direction to those which result from aspiration.

II. **Traumatic Circumscribed Labyrinthitis with Traumatic Neurosis.**—Such cases suffer attacks of vertigo with or without the external causes mentioned above. In these attacks the quick component of the nystagmus is directed to the diseased side. The consciousness of an injury to the head, followed by impairment of hearing, of vertigo, Romberg's phenomenon, hemiparesthesia, sensitive spots, trembling of the eyelids, unsteady gait, especially with closed eyes, causes great apprehension on the part of the patient and finally develops neurasthenia.

These are medicolegal cases. Because of this, and for purposes of diagnosis, the history must be carefully studied. It is important to know whether the patient had vertigo, nausea and vomiting, or whether he was unconscious directly after the accident. If the equilibrium was disturbed, was it sufficient to compel him to lie down; was the hemorrhage from the ear and nose? Did vertigo come on while in bed? Did movements of the head or turning in bed cause vertigo or nystagmus? Did the vertigo come on first upon arising from bed, or after going to work? Did the vertigo tend to increase or diminish? Answers to these questions determine not only the extent of injury and incapacity, and the kind of vertigo, but also the correctness of the statement.

Vertigo, and in consequence incapacity for work, is the common complaint of those who receive injuries to the head, whether malingerers or not. In true cases, inclining the head backward causes vertigo, slight nausea, and weak rotatory nystagmus to the injured side. The nystagmus cannot be immediately reproduced, though the patient experiences a strong vertigo and slight nausea.

Syringing the injured ear between attacks with water of 78° F. or lower, produces typical strong nystagmus to the opposite side. The same procedure on the sound side gives nystagmus to the diseased side. Severe vertigo, nausea and vomiting, pallor, free perspiration, and trembling of the whole body form the usual clinical picture of traumatic circumscribed labyrinthitis with traumatic neurosis. The nystagmus, which is accompanied by vertigo, is like the spontaneous during attacks, but stronger. Between attacks, turning to the opposite side to the injury, the head erect, causes after-nystagmus quite like the normal. Objects seem to turn around the patient. There is no nausea, and, therein it is unlike spontaneous nystagmus. About three turnings, with the head inclined 90 degrees forward, produces rotatory nystagmus, with vertigo and nausea, which the patient identifies as being similar to the spontaneous attacks. If the patient, with or without suggestion from the examiner, identifies the horizontal primary, or after-nystagmus

with the spontaneous attacks, he is malingering and his story is untrue. Those who have the real trouble make no mistakes.

Traumatic Destruction of the Labyrinth.—An injury may cause fracture through, or sufficient hemorrhage into, the labyrinth to destroy it completely. The symptoms will then be the same as in acute diffused manifest suppurative labyrinthitis, or after the labyrinth operation. In short, the symptoms of labyrinth destruction are the same from all causes, if the patient is conscious. If the patient survives, the after-symptoms are those of complete labyrinth destruction of one side, with total deafness on the injured side.

If the patient claims to be unable to work because of vertigo, the history, together with complete one-sided deafness, negative response to the caloric test of the injured side, and modification of the normal physiological nystagmus in response to turning will disprove the claim, as there is a readjustment of the function of equilibrium in these cases. Incomplete destruction is followed by the attacks of vertigo mentioned above. Complete destruction causes severe vertigo, nystagmus, nausea, and vomiting at the time of the destruction, all of which tend to improve from the time of onset, finally ceasing altogether.

Nystagmus from Toxemia.—Smokers, drinkers, and those who suffer from auto-intoxication have spontaneous attacks of vertigo and nystagmus, which may or may not be accompanied by vomiting. In much the greater number of such patients the membrana tympani is intact, the vestibular apparatus responds to all tests, and perception of sound is normal. The nystagmus is vestibular in character, arising from toxic influences acting upon the centres in the fourth ventricle or disabling the vestibular nerve. Slight attacks of vertigo are also found in those who consider themselves, and who, upon examination, seem to be perfectly healthy. They have such attacks upon arising in the morning and when stooping quickly. Temporary congestion of the head probably causes them.

Vertigo and Nystagmus in Neurasthenics.—Spontaneous attacks of vertigo of cerebral origin occur especially in neurasthenics. The vertigo comes on when the vision is fixed on an object for some time, and causes disturbances of equilibrium. The movements of the eyes are not of the vestibular type, though they are constant. These patients may fall, but in no definite direction. Apparent movement of surrounding objects is noticed by them. They also have attacks of vertigo of the true vestibular character when bending forward, arising in the morning, or upon sudden movement of the head. The vertigo, produced by turning ten times is stronger than the spontaneous attacks. They become pale, tremble, perspire, and lose consciousness completely or partly. Any or all of these symptoms may be present. One or two turnings, with the head inclined 90 degrees forward, produces vertigo and rotatory nystagmus, which the patients identify with their spontaneous attacks. They occur without disease of the ear, and stamp the neurasthenic, as do also the following symptoms, in disease of the ear.

In neurasthenics with circumscribed labyrinthitis rapid movements of the head produce a stronger vertigo than in neurasthenia alone. In about 50 per cent. of these cases such attacks can be produced upon the first examination by quickly inclining the head backward while the patient is in a sitting posture. Rotatory nystagmus to the diseased side and vertigo occur and cannot be reproduced by the same manipulation for ten to fifteen minutes. It is probable that the rapid movement of the head causes an expenditure of energy the regeneration of which requires this time (Barany). Vestibular disease tends to shorten the duration of horizontal after-nystagmus; neurasthenia tends to prolong it. In neurasthenics who have vestibular disease, the duration of the horizontal after-nystagmus[1] is normal, because the two tendencies counteract each other (Barany).

Nystagmus in Acute Destruction of One Labyrinth.—The immediate symptoms of destruction of one labyrinth, from whatever cause, are strong rotatory and horizontal nystagmus, the quick component of which is directed to the sound side, occasionally to both sides. Severe vertigo, nausea, and vomiting, apparent movement of surrounding objects, sensation of turning of the body, and inability to walk, are often complained of. The patient must lie down, and quickly finds lying on the sound side to be more comfortable, because when in this position, in looking at surrounding objects the eyes are directed to the slow component which minimizes or abolishes the spontaneous nystagmus. It will be remembered that one of the characteristics of vestibular nystagmus is that it is diminished by looking toward the slow component and increased by looking toward the quick component. From the position assumed the eyes are of necessity directed toward the slow component, and all annoying symptoms are quickly relieved. The position voluntarily assumed while in bed is quite suggestive.

The caloric and fistula tests are negative. After two or three days the symptoms begin to disappear, the nausea and vomiting being the first to subside in persons of a stable nervous system. On the third day there is no vertigo, while the patient keeps quiet, though the nystagmus persists. With quick movements of the head the nystagmus increases and the vertigo again comes on. When the complete operation on the labyrinth is done, the nystagmus and accompanying symptoms subside much more quickly than after diffuse suppurative labyrinthitis. This suggests that the stimulation of Deiters' nucleus through the trunk of the vestibular nerve in the latter case is so great that the coördination is delayed. As the conditions are the same whether the destruction is traumatic or toxic, the impression is conveyed through the nerve trunk. The cristæ ampullares, being destroyed, the preponderance of tonus is in the opposite labyrinth, and the nystagmus is therefore directed to the sound side (Plate XXV). The removal of restraint allows the sound side to functionate violently, causing the compound nystagmus and accompanying symptoms

[1] Primary, after-, and after-after-nystagmus always refer to nystagmus produced by the turning test.

to be severe. It must be remembered that a horizontal nystagmus frequently appears toward the diseased side when the nystagmus to the sound side is diminished. Barany does not attempt to explain this phenomenon, as to do so would be pure speculation.

In two or three weeks after the destruction of the labyrinth all symptoms disappear except a little nystagmus to the sound side, and occasionally slight horizontal nystagmus to the diseased side. These are symptoms of latent labyrinthitis which of course follows acute destruction. In the period of latency the sound side loses some of its responsiveness to both the caloric and the turning tests, probably on account of the changes which take place in the centres in the readjustment of the equilibrium.

Nystagmus in Diffuse Latent Suppurative Labyrinthitis.—Weak rotatory nystagmus exists to both sides when the eyes are in the extreme lateral position, though it is somewhat stronger to the sound side. There is no nystagmus when the patient looks straight ahead, unless opaque spectacles are used, in which case very slight nystagmus occurs to the sound side. The caloric test of the diseased side is negative. Cold water in the sound ear usually produces a strong rotatory nystagmus to the opposite side. In some cases this reaction is weaker than normal. Evidently the readjustment both in the centres and the vestibular end-organ differs in individuals. It is probable that the normal end-organ takes up the function previously performed by both, and in one case transmits a strong impression and in another a weak impression to Deiters' nucleus.

The galvanic tests for both the anode (positive pole) and the kathode (the negative pole) are negative, or nearly so. Aspiration and compression tests are negative. Ten turnings to the diseased side, with the head erect, produce horizontal after-nystagmus to the sound side of about thirty seconds' duration when opaque spectacles are used. Ten turnings to the sound side, the head erect, produces horizontal after-nystagmus of fifteen seconds' duration toward the diseased side when opaque spectacles are worn. The same turning to the diseased side with the head inclined 90 degrees forward produces rotatory after-nystagmus to the sound side of twenty seconds' duration, if the spectacles are worn (Plate XIX). Ten turnings to the sound side, with the head inclined forward 90 degrees, produces rotatory after-nystagmus of ten seconds' duration, if the spectacles are worn. (In Plate XXV twelve and six seconds are used.) These turning reactions are typical of latent uncomplicated labyrinth disease of one side, and may be used clinically and relied upon when the caloric test is made uncertain by atresia or stricture of the external auditory canal, the presence of a cholesteatomatous mass, or of acute suppurative otitis media. If the duration of the after-nystagmus to the sound side is below the averages given above, that to the destroyed side will not be more than half as long, except in those cases in which complete intralabyrinthine compensation has occurred, as reported by Ruttin. If the duration to the sound side is greater than the average, the same relation will persist.

Nystagmus by turning should be frequently produced by the surgeon in both normal and pathological cases if he means to become thoroughly acquainted with this valuable aid to diagnosis.

Nystagmus in Meningitis.—Differential Diagnosis.—In the early stage the differential diagnosis between meningitis and cerebellar abscess is very difficult. The condition of temperature marks the greatest difference. The nystagmus in both cases is the same. In meningitis the temperature is, as a rule, relatively high, though abscess may also begin with this symptom. All the pressure symptoms in the posterior fossa may accompany circumscribed meningitis in this situation. Hemiataxia has, however, never been observed in Politzer's clinic. Nystagmus of the same vestibular character as in cerebellar abscess is produced by involvement of the vestibular nerve in the internal auditory canal. Sudden diminution of sound perception in the ear is more indicative of meningitis. Severe stiff-neck and hyperesthesia of the skin are symptoms more frequently encountered in meningitis than in cerebellar abscess. If the meningitis extends to the convexity, general convulsions, sunken abdomen, small, quick pulse, Cheyne-Stokes respiration, and total unconsciousness occur, and these make the diagnosis simple, and, it may be added, operative interference less effective. In meningitis serosa there are also symptoms. The changes of temperature are slight. Sinus thrombosis, especially of the cavernous and lateral (sigmoid) sinuses, and middle-ear suppuration, especially when complicated by mastoiditis, may cause meningitis without involvement of the labyrinth. In these cases the symptoms are nystagmus, vertigo, vomiting, headache, and occasionally facial paralysis. The nystagmus is of retrolabyrinthine (namely, intracranial) origin.

Optic neuritis, choked disk, unconsciousness, and convulsions form a symptom-complex which never characterizes an uncomplicated otitis media, though unconsciousness and convulsions may be present in very young children. In the latter cases, simple paracentesis, or operation for acute mastoiditis, will often cause the symptoms to disappear. In hysteria we often find otitis media with hemianesthesia, hemiparesis, vertigo, nausea, and disturbances of vision, but the hemiparesis and anesthesia are on the diseased, instead of the opposite side.

Nystagmus of Intracranial Origin.—Intracranial nystagmus is of the vestibular type, with the difference that, instead of growing continually weaker, and ceasing altogether in from twenty (rotatory nystagmus) to forty (horizontal nystagmus) seconds, on the average (physiological vestibular nystagmus), or in from a few minutes to several days (pathological vestibular nystagmus), it grows constantly stronger without the tendency to cease. The early differential diagnosis between vestibular and intracranial nystagmus depends largely upon the responsiveness of the vestibular apparatus to the caloric and turning tests. In cases in which the vestibular responsiveness is lost, a positive diagnosis can be made from the character of the spontaneous nystagmus (Barany, Neumann). When a labyrinth is non-responsive and a strong rotatory nystagmus to the same side is present, the nystag-

mus must arise from some intracranial disease. When the vestibular end-organ is completely destroyed, it cannot produce nystagmus. The nystagmus which occurs to the diseased side cannot emanate from the sound side, because by the loss of coördination it would overbalance and produce a nystagmus to the side opposite to the destroyed labyrinth. The presence, however, of a stronger irritation through the course of the vestibular nerve, or from Deiters' nucleus of the diseased side, will produce nystagmus to the diseased side (Plate XVII). The accompanying vertigo is very marked. If a labyrinth is destroyed, and there is a strong rotatory nystagmus with the quick component directed to the opposite side, it is natural to suppose that it is caused by the sound vestibular apparatus. This is, however, not necessarily true.

If the nystagmus increases, instead of diminishing in intensity, as in labyrinth destruction, then it is of intracranial origin, probably due to a cerebellar abscess irritating the opposite half of Deiters' nucleus.

With deafness and nystagmus of the intracranial type, and intact tympanic membrane, tumor along the course of the vestibular nerve is most probable.

In labyrinth suppuration, in which the vestibular apparatus of the affected side does not respond to the physiological tests, and in which the nystagmus is toward the diseased side, circumscribed meningitis of the posterior fossa may be present. This nystagmus is of the same character as that emanating from the vestibular apparatus, or that caused by cerebellar abscess. The differential diagnosis is made chiefly from the peculiarities of the pulse, temperature, etc.

Neumann says that in cerebellar abscess the nystagmus is always of the rhythmic character, so thoroughly described by Barany. The differentiation between the vestibular nystagmus of cerebellar origin and that from the semicircular canals is made, on the one hand, through the exact examination of function of the vestibular apparatus, and on the other, through the course of the disease. It is not always possible to make a diagnosis between labyrinthitis and intracranial lesion. There are, of course, irregular cases. The labyrinth must first be removed from consideration by operation when the tests fail.

The nystagmus induced by circumscribed labyrinthitis is directed to the diseased side. Should the disease progress to the destruction of the irritability of the vestibular apparatus, the direction of the nystagmus changes. It moves toward the sound side and remains there until the entire labyrinth is destroyed. It then gradually diminishes in intensity, and in a short time ceases. If the complete labyrinthine operation of Neumann is performed while the nystagmus is directed to the sound side, it remains unchanged for the first day, and then decreases notably for two or three days, and in a short time ceases altogether. During the time the nystagmus is directed toward the diseased side, the response to irritation is the same as in a normal ear. By irrigating with cold or warm water, the typical nystagmus as described by Barany appears as indicated above. When the direction of the nystagmus changes to the

sound side, the irritability of the labyrinth is usually lost, but if the labyrinth still responds to irrigation, the nystagmus is very weak or of short duration. As the disease progresses, the irritability of the labyrinth fails completely, and the nystagmus remains directed to the sound side. The nystagmus of cerebellar origin is, however, directed to both the diseased and the sound sides, though that directed to the diseased side overbalances the other. In the cases of otitic cerebellar abscess examined by Neumann, in which the exact examination of nystagmus was made, the cerebellar abscess was always a complication of labyrinthine suppuration. In these cases the differentiation of cerebellar from labyrinthine nystagmus was as follows:

1. When the nystagmus is directed toward the diseased side, either a circumscribed labyrinthitis or a cerebellar abscess may be present. In circumscribed labyrinthine disease, irritability from irrigation is normal; at the same time the symptoms of a labyrinthine fistula may exist; that is, compression and aspiration of air or pressure on the wall of the labyrinth cause nystagmatic eye movements. When irritability to irrigation is lost, direct pressure with a probe, or galvanization, will produce nystagmus.

Under the latter circumstances, the diagnosis of cerebellar abscess cannot be made before the labyrinth is destroyed by operation. These indications, worked out by Neumann in Politzer's clinic, should justify adding the labyrinthine operation to the radical mastoid operation when in doubt. After the operation on the labyrinth, the nystagmus, if of vestibular origin, must change its direction to the sound side. Neumann has not observed a single case of cerebellar abscess associated with circumscribed labyrinthine suppuration. If, after the labyrinthine operation, rotatory nystagmus remains directed to the diseased side, the diagnosis of cerebellar abscess or some other disease in the posterior fossa of the same side is immediately made, because a destroyed labyrinth never causes nystagmus to the same side. Barany and Neumann are of the opinion that the nystagmus toward the sound side emanates from the sound side. If in spite of the operative destruction of the labyrinth the nystagmus remains directed to the diseased side, it must be retrolabyrinthine in origin, through irritation of Deiters' nucleus or the vestibular nerve at the base of the brain.

2. If the labyrinth does not respond to irritation (the various tests), and the spontaneous rotatory nystagmus is toward the affected side, and the pulse and temperature are characteristic, a diagnosis of cerebellar abscess may be made.

3. If spontaneous nystagmus toward the sound side is present (the opposite middle ear being diseased) and the labyrinth on the diseased side is not responsive, it may be of either labyrinthine or cerebellar origin. In such a case it is impossible to differentiate before the labyrinth operation. If the nystagmus disappears in two or three days after the operation, it is of vestibular origin. If, however, it does not cease after the operation, but increases in intensity, or changes its direction to the diseased side, it is of intracranial origin.

PRACTICAL DEDUCTIONS

1. Vestibular nystagmus is the result of a *sudden* loss of balance between the right and left vestibular apparatuses. The sudden loss of tonus may be due to (*a*) artificial stimulation, as the turning, caloric, fistula, and galvanic tests (induced nystagmus); or (*b*) it may be due to intralabyrinthine stimulation, or paralysis, as in the various forms of labyrinthine inflammation, serous and suppurative, or hemorrhage (spontaneous nystagmus).

2. When both vestibular apparatuses are normal, and no unusual artificial stimulation is being applied, the tonus impulses which constantly influence all the voluntary muscles of the eyes, body, and extremities, are equal on the two sides and there is, therefore, perfect coördination of all the voluntary muscles. When, however, an unusual stimulus is applied, as in continuous turning of the body, or irrigation of one ear with cold water, etc., the tonus impulses in the ears or ear thus stimulated are increased, and incoördinate movements of the eyes and extremities result; that is, nystagmus, and the reaction movements of the body and extremities occur. These reactions are known as induced nystagmus, and induced reaction movements. When the body is rotated again, but in the opposite direction, or the opposite ear is irrigated with cold water, the nystagmus and the reaction movements of the body are reversed in direction, but are of about the same intensity and duration. Both vestibular apparatuses are thus shown to be normal (see paragraph 9).

3. When one vestibular apparatus has been totally destroyed by fracture through the petrous portion of the temporal bone, and extralabyrinthine compensation has taken place, that is, spontaneous nystagmus has ceased, and the right labyrinth, the one recently destroyed, is irrigated with cold water, neither nystagmus nor the reaction movements of the body will occur, as the labyrinth is dead. If the normal left ear is irrigated, nystagmus and the reaction movements will be aroused. Such a patient having passed through the acute diffuse manifest stage of labyrinthitis without involvement of the meninges and brain, there is no indication for an immediate labyrinth operation. Such cases do not always, or even usually, pass through this stage so fortunately. Indeed, many of them subsequently develop meningitis or cerebellar abscess, and die. If seen at the time of total destruction of the labyrinth, as shown by total loss of hearing and vestibular response to the caloric test, etc., it would have been perfectly proper to have seriously considered the advisability of performing a labyrinth operation at once. Authorities differ on this point, though an immediate operation seems to afford the better chance for the life of the patient. Some of them will die even though an operation is done. Probably more will die if it is not done, especially if it is postponed long after latency is established, as cerebellar abscess often develops in the course of diffuse latent suppurative labyrinthitis. The

case having, however, safely passed through the acute destruction, the question of operation must be viewed differently. If the patient still suffers from mastoiditis and requires a mastoid operation for its cure, the labyrinth should also be operated at the same time, especially if during the mastoid operation a fistula of the labyrinth is found, and more especially if it is located in the oval window. Experience has shown that if only the mastoid operation is performed in such cases, a reactionary inflammation may be lighted up and extend to the meninges or cerebellum and result fatally. Furthermore, the infection may extend to the meninges or cerebellum at any subsequent time, even without a mastoid operation. As the hearing is already destroyed in the affected ear, and cannot be rendered worse by a labyrinth operation, and the operation is not, of itself, attended by a high mortality rate (provided the meninges or cerebellum are not involved), the period of latency affords a peculiarly favorable time to perform the labyrinth operation.

If, in the example referred to in the preceding paragraph, the right labyrinth has been only partially destroyed (one month previously) and extralabyrinthine compensation had occurred, and the right ear had been irrigated with cold water, a weak nystagmus toward the normal side would have been induced, but of shorter duration than normal; whereas cold irrigation in the sound ear would have induced nystagmus of normal intensity and duration to the diseased side. These results indicate a partial destruction of the right or affected labyrinth. When partial function is shown, there is no probability of the infection immediately invading the meninges and brain, at least, not without fulminating vestibular symptoms; hence no indications are present for an operation on the affected labyrinth.

4. Serous labyrinthitis is not usually a dangerous disease, though it may become so if it is converted into the diffuse suppurative type. When thus converted it should be treated in all respects as an acute diffuse suppurative manifest labyrinthitis.

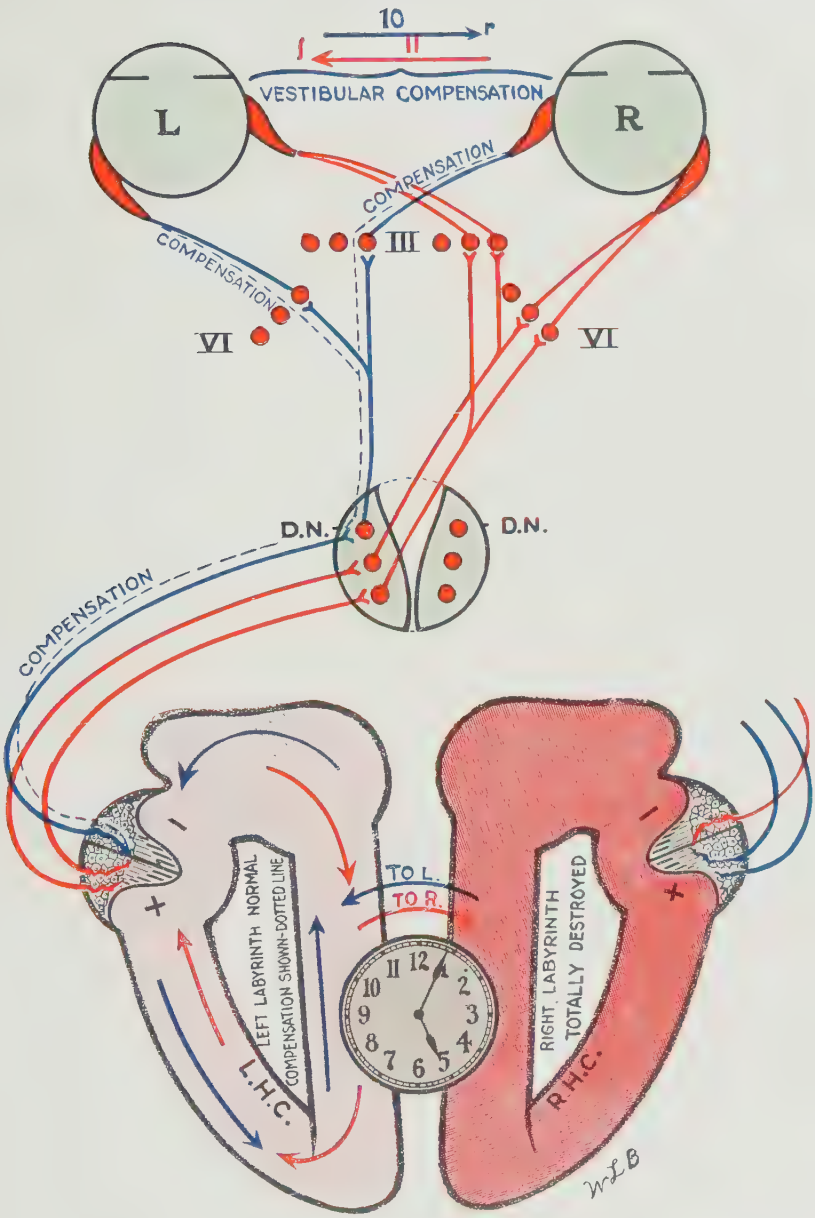
5. Circumscribed labyrinthitis is not, of itself, a dangerous disease, but when it becomes converted into an acute diffuse manifest suppurative labyrinthitis it is a serious condition which must be treated as such.

6. The presence of even a vestige of hearing in the affected ear is a contra-indication to the labyrinth operation, not because there is no danger of the infectious process invading the meninges and cerebellum, because there is this danger; but because, if it should extend to the cranium, warning, in the form of complete deafness in the affected

PLATE XXVI

Right labyrinth totally destroyed several years ago, showing complete vestibular compensation after the lapse of several years. The two halves of the crista of the healthy labyrinth become equal in tonus, whereas normally their tonus is 2 to 1, the canal half of the crista having a tonus of 2 and the utricular half a tonus of 1. Doubtless the stronger half atrophies and the weaker half hypertrophies until the two become equal.

PLATE XXVI



Complete Vestibular Compensation Several Years after the Labyrinth was Destroyed.

ear, and of nystagmus and the reaction movements of the body and extremities, will occur at the moment of the destruction. When these danger signals become manifest the labyrinth operation should be performed without delay, as the delay of a few days might allow meningitis or other intracranial involvement to develop, and thus render the prognosis quite grave; whereas prompt operative measures would have rendered the prognosis very good. Jansen and others have reported cases in which meningitis occurred before complete destruction of the labyrinth, though this is exceptional.

7. The presence of a vestige of vestibular reaction to the caloric or fistula test is also a contra-indication to the labyrinth operation, at least to an immediate operation. The presence of vestibular reaction to the caloric and compression stimulation shows a mechanical anatomical barrier between the labyrinth and the cranial content. Should these barriers be suddenly destroyed, the spontaneous nystagmus and the reaction movements will again become manifest, and thus warn the attending otologist of the progress of the infection toward the meninges and cerebellum. When, subsequently, the tests show the total destruction of hearing and vestibular reaction, an immediate labyrinth operation may be advised.

8. In pathological processes of the labyrinth the vestibular tests are performed to estimate (*a*) the presence or absence of vestibular reaction, (*b*) the degree of stimulation required to produce vestibular reaction, and (*c*) the intensity and duration of the vestibular reaction. By ascertaining these facts and considering them in association with all the other clinical phenomena, the type of labyrinth disease may be determined, the prognosis formulated, and the indications for treatment outlined.

9. The reactions induced by the vestibular tests in disease of the labyrinth are sometimes greatly modified by the amount and type of compensation which has occurred. For example, in three or four weeks after an acute sudden destructive process in the labyrinth, compensation apparently occurs; that is, the spontaneous nystagmus and reaction movements so far subside that the ordinary methods of observation do not detect them. Slight stimulation would, however, make them manifest again. Two years later it might require a stronger stimulation to make them manifest. Eight or ten years later compensation may be complete; so complete, indeed, that the two halves of each crista of the normal labyrinth may have equal tonus, as evidenced by inducing nystagmus of nearly equal intensity and duration by the turning test to the right, and then to the left (Plate XXVI). Such reactions to the turning tests would ordinarily be interpreted as showing both labyrinths as normally functionating, whereas, as a matter of fact, in the instance cited, one labyrinth is totally destroyed and out of commission, and complete intralabyrinthine compensation has occurred. Hence, as I have already repeatedly said, the tests of the vestibular portion of the two labyrinths are of clinical value insofar as they show the equality or the inequality of tonus impulses emana-

ting from them, and more especially in circumscribed, serous, and acute diffuse manifest suppurative labyrinthitis, and in the earlier months or years of diffuse latent suppurative labyrinthitis. The duration of the reactions are greatly shortened but are of about equal duration.

10. The functional tests of the vestibular apparatus are also of value in differentiating labyrinthitis from cerebellar disease, meningitis, etc. The nystagmus in cerebellar disease may be greatly intensified by the cold caloric test; or, if it is but slightly manifest before the caloric test, so slight, indeed, as to escape ordinary observation, it will produce a nystagmus of great intensity and duration. This is known as Neumann's enduring nystagmus reaction.

11. When meningitis occurs in the course of an acute diffuse manifest suppurative labyrinthitis, the nystagmus may change its direction to the normal side, whereas, before the meningitis occurred, it was to the diseased side.

12. Spontaneous vestibular nystagmus rapidly declines in intensity, and within a few hours, days, or weeks disappears.

13. Cerebellar nystagmus tends to continue indefinitely and to increase in intensity.

14. In spontaneous vestibular reaction movements, the patient tends to fall toward the slow component of the nystagmus, and the direction of falling is influenced by the position of the head, whereas, in cerebellar nystagmus, the slow component of the nystagmus and position of the head have no influence on the direction of the falling.

Other facts might be tabulated under the caption of this section, but the above summary will suffice to place in concrete form the more important practical deductions in reference to nystagmus, and the vestibular tests in relation to labyrinth disease.

CHAPTER L

LABYRINTHITIS: ILLUSTRATIVE CASES

So much has been written about the theoretical aspect of the labyrinth, and disease thereof, that there is danger of the real and immediate demands upon the clinical otologist being submerged under a mass of literary opinions, which may well leave him in a nebulous haze of confused ideas. It will be my endeavor in this chapter to formulate some of the more simple and practical problems, in relation to disease of the labyrinth, with which the clinical otologist is more or less frequently confronted. The more theoretical and abstract propositions, which are of so much interest, and the solution of which will ultimately benefit the profession and suffering humanity, have been discussed in the preceding pages, hence our attention at this time will be confined to the consideration of the well-recognized clinical manifestations of labyrinth disease, and more especially as they are observed in practice. We will assume that not all otologists are fully prepared to give these cases the attention they need. This is due to confusion as to the relative importance of the clinical phenomena, and the theoretical problems which are usually considered in the discussion of these cases. While the theoretical problems are of importance in the analysis of these cases, the clinical aspect must ever be given the greater attention if we wish to be of the greatest service to our patients.

The consideration of a few typical cases selected from my practice may help to "get a line" on this disease, to the extent that we may at least give those thus afflicted intelligent advice, rather than evasive or unintelligible information, such as was given by an otologist who said he operated upon a labyrinth to improve the hearing.

The type of labyrinth disease more often and easily recognized is characterized by the total destruction of hearing in one ear, which at the time of destruction was either a diffuse manifest or a diffuse serous labyrinthitis, and in its later manifestation is known either as diffuse latent suppurative labyrinthitis or as a dead auditory labyrinth from profound serous disease, though total destruction of the labyrinth is probably rare in serous labyrinthitis. In simple words, this is a case in which weeks, months, or years previously there was a diffuse manifest suppurative labyrinthitis or a diffuse serous labyrinthitis of the fifth degree (see Serous Labyrinthitis) which caused total destruction of the organ of hearing (the organ of Corti of the cochlea), and in addition to this, the static labyrinth (semicircular canals, ampullæ, and

crista ampullares) was wholly or nearly wholly destroyed. I have seen several cases in which there was total destruction of the cochlea, but in which there remained some vestibular function, as shown by the fistula and caloric tests. This is explained upon the theory that the vestibular portion of the eighth cranial nerve is more resistant than the cochlear portion. It is probable, therefore, that in so-called diffuse manifest suppurative labyrinthitis total destruction does not always occur in the vestibular end-organs, *i. e.*, the cristæ ampullares. Some authors classify these cases as severe serous labyrinthitis, upon the theory that acute manifest suppurative labyrinthitis is always attended by complete destruction of both the cochlear and vestibular apparatuses. Further observation and postmortem findings will be required to establish the truth respecting the matter. It is perfectly conceivable, however, that in acute diffuse manifest suppurative labyrinthitis, the cristæ ampullares may be only partially destroyed, and that in diffuse serous labyrinthitis the cochlea may be totally destroyed and the cristæ only partially destroyed. We are advised by eminent authorities, however, that those cases in which there was total and permanent deafness and abolition of vestibular reaction, were of the acute diffuse manifest suppurative type of labyrinthitis, and that those cases in which total deafness and *partial* loss of vestibular reaction were of the diffuse serous type of labyrinthitis. The solution of this phase of the subject assumes considerable importance, as serous labyrinthitis, even when of the severest degree, is not as liable to be attended or followed by intracranial disease as is acute diffuse manifest suppurative labyrinthitis, especially if fistula is present. If total destruction of the cochlear and static labyrinth should occur in serous labyrinthitis, there would be no actual justification for the labyrinth operation, as this process is not liable to involve either the brain or meninges. If an operation is performed it is based upon the assumption that the total destruction of the static and auditory labyrinths points almost incontrovertibly to a suppurative labyrinthitis. If, on the contrary, it can be demonstrated that the total destruction of the cochlear and vestibular apparatus is due to acute diffuse manifest suppurative labyrinthitis the indications for the labyrinth operation are much stronger and may be imperative. For example, if upon inspecting the ear through a perforation in the membrana tympani, pus is observed oozing through a perforation in the promontorium, the case is undoubtedly of the suppurative type.

It appears, therefore, that until more discriminating methods of diagnosis are adopted, that we must either maintain an expectant watchful attitude toward the doubtful cases, or regard all such cases as suppurative and a grave menace to life. The latter attitude carries with it the assumption that these doubtful cases should be given the benefit of the doubt, and treated or operated as diffuse manifest suppurative labyrinthitis. This assumption carries with it another one, namely, that more lives are saved by the operation than would be

by "waiting for developments," or to express it differently, more cases will die if such cases are "watched" for intracranial involvement than if operated at the time of the total destruction. Most authorities prefer to keep these cases under the closest observation, and if no progression is indicated, *i. e.*, if the temperature does not remain elevated, and if headache, etc., do not become more pronounced, to avoid a labyrinth operation. These cases seek advice as to their defective hearing, chronic otorrhea, etc. The problems are, What can be done to either relieve or cure the remaining disease? Is his health or life endangered? If so, what can be done to improve his health, or prevent death? These are the problems confronting the otologist. He is not so much concerned with the refinements of the pathology or the theories explaining the various reactions to the tests of the labyrinth as he is with the immediate needs of his patient.

I have selected typical cases from my practice to illustrate some of the clinical aspects of labyrinthitis, and hope to present them as individual problems to each reader. As each case is presented, I will endeavor to formulate the mental problems presented in its analysis. By this method of presentation I hope to put the consideration of labyrinth disease upon a simple clinical basis suited to the embryo labyrinthologist, rather than upon a complex and often confusing laboratory basis, which is only available to the more advanced student.

Case I.—Mr. F., aged forty-five years. Consulted me March 6, 1912, complaining of deafness in the right ear, from which there had been a chronic discharge for ten years. Ten years ago he had an attack of acute tonsillitis which was followed by a severe inflammation of both ears, followed by a discharge of purulent matter. During the acute attack he become nauseated, and vomited when he attempted to move about. He was confined to his bed for several days. He was giddy when upon his feet, and had to be supported to the toilet room. The room seemed to whirl about him, though in what direction he does not remember. Was totally deaf in the right ear at that time, and has remained so since then. Has had numerous giddy attacks since the right ear was destroyed, though he has never fallen to the ground during one of them.

At the time he consulted me he was complaining of giddiness, deafness in the right ear, and a thin seropurulent discharge from the same ear. Had recently passed through an attack of acute coryza which he thought brought on the giddiness and increased the discharge.

The first problem presented in this case was, Is the patient totally deaf in the right ear, or is he only partially so? As the solution of this problem would reflect light upon the general character of the disease in the right ear, it was important to determine at once whether or not the patient was totally deaf in the affected ear. Total deafness rarely or never occurs in adhesive processes in the middle ear or in otosclerosis. It only occurs in diffuse suppurative labyrinthitis, diffuse serous labyrinthitis, and in fractures through the petrous portion of the temporal bone, etc. Hence, if this case was totally deaf in the

affected ear, it would, in view of the previous history, point to a complete destruction of the auditory labyrinth during the primary disease, ten years previously. At that time he obviously suffered from either diffuse serous labyrinthitis in the fifth or extreme degree, or from acute diffuse manifest suppurative labyrinthitis, and is at present affected with a dead, auditory labyrinth, with bony fistula, and still has some function of the static labyrinth. Had he been affected by diffuse manifest suppurative labyrinthitis ten years ago, both the auditory and static labyrinths would, in all probability, have been totally destroyed. As the static labyrinth still has some function left, as shown by the attacks of giddiness and the slight response to the fistula compression test, the original disease may have been serous in character, though it is possible that he may have had manifest suppurative labyrinthitis without total destruction of the static labyrinth. Partial destruction is, however, rare in this type of disease.

To determine the question of partial or total deafness was the first problem to be solved. How was this done? To the otologist, trained in labyrinth work, this question may appear puerile. I know some very reputable otologists, however, who in their present state of knowledge could not answer it. Their methods of observation pursued are often misleading. Only recently a physician of deservedly high reputation, but who had not fully grasped the labyrinth problem, brought a case to my office for consultation. When I offered to test the hearing he exclaimed, "There is no use testing the hearing, as I did it yesterday, and found he heard the watch on contact with the affected ear." I replied that I only wished to determine the "degree of hearing," and proceeded to use Barany's noise apparatus to make the test for total deafness, and found the patient to be totally deaf in the affected ear.

The test for total deafness (destruction of the auditory labyrinth) is made with Barany's noise apparatus which consists of a small alarm clock (minus the time element) provided with an ear-piece fitting the external auditory meatus. This was tightly inserted into the external auditory meatus of the sound ear of the patient, and pressure upon a button set off the alarm or noise apparatus. This completely shut off the hearing in the sound ear, but did not affect it in the diseased ear. I then addressed the diseased ear with increasing loudness up to the shouting voice, and the patient did not hear a sound; that is, he was totally deaf on the affected side. This showed total destruction of the auditory labyrinth of the affected ear. The static labyrinth still retained some function, as was shown by the fistula test and by the history of the attacks of giddiness. The cold caloric test elicited nystagmus to the opposite side, though it was very weak and of short duration. The turning test to the right, or toward the affected ear, induced a nystagmus to the left of eight seconds' duration. Turning to the left induced a nystagmus to the right of eight seconds' duration. The ratio was 1 to 1, showing complete vestibular compensation. The turning test therefore corroborated the

caloric test. Having settled the first problem, namely, that the cochlear labyrinth was totally destroyed, the next problem was, Is the static labyrinth totally or only partially destroyed? The history of recurrent attacks of giddiness and the results of the fistula test showed that the static labyrinth (cristæ ampullares) still retained some function. The original disease, therefore, was probably serous labyrinthitis rather than diffuse manifest suppurative labyrinthitis, as in diffuse manifest suppurative labyrinthitis both the cochlea and static labyrinths (the vestibular nerve endings) are almost always totally destroyed. It should be repeated, however, that we have no diagnostic means at our command whereby we can determine with absolute certainty whether the disease is serous or suppurative in character.

A mastoid operation, done alone, might excite a reactionary inflammatory process in the labyrinth, and cause its extension to the meninges or brain. If the mastoid operation is imperative, safety would be more assured by removing the labyrinth at the same time. If this is not done the closest observation of the case should be maintained and the earliest fulminating symptoms, as severe persistent headache, giddiness, nausea and vomiting, should be regarded as imperative indications for the labyrinth operation. I cannot refrain from again repeating what I have so often said before, that waiting for these fulminating symptoms is somewhat like waiting to see the mouse go into the cat's stomach before attempting to rescue it. In the presence of such manifestations an immediate labyrinth and mastoid operation is indicated, especially if a fistula is present. The hearing being already wholly destroyed, does not constitute a contra-indication to the operation. A contra-indication to the labyrinth operation is the likelihood of exciting meningitis by it. This is not likely, however, as with the Jansen-Neumann operation the meninges at the site of probable infection would be freely exposed (see Surgery of Labyrinth). The danger usually mentioned as attending this operation is the possibility of injuring the dura. This is not probable if the dura is elevated before removing the petrous bone containing the semicircular canals. In this case the whole of the diseased labyrinth should be removed as it is suppurating, a wide, open wound established, and the meninges widely uncovered and drained. Thus operated the patient should be permanently cured of the offensive discharge and giddiness, and be forever relieved of the menace of cerebellar abscess and meningitis. Case I, as referred to, was operated by me in May, 1912, by the Jansen-Neumann method, immediately following the radical mastoid operation. His recovery was uneventful and the discharge has ceased, and giddiness is no longer a factor in the case.

Case II.—Mrs. B., aged fifty years. Twelve years ago she had a discharge from the same or opposite ear, she could not remember which. Six weeks ago she had acute tonsillitis, followed by an earache on the left side. Paracentesis of the drum-head was performed by a competent otologist, and this was followed by a purulent discharge.

Four days before I was called she had an attack of vertigo, giddiness, nausea and vomiting, and nystagmus to the right or healthy side. She was prostrated, and movements in bed aggravated the symptoms. Her condition at the time of the consultation was as follows: (*a*) Spontaneous rotatory nystagmus to the right or healthy side; (*b*) total deafness in the left ear, as shown by Barany's noise apparatus in the sound ear; (*c*) fistula symptom absent; (*d*) the knee reflex on the left side was slightly exaggerated; (*e*) Babinsky was negative; (*f*) the pupils were unequally contracted; (*g*) the temperature ranged from 101° to 102° F.; and (*h*) she complained of continuous headache.

The patient was removed to a local hospital, where I performed a combined radical mastoid, and a Jansen-Neumann labyrinth operation. The facial nerve was not injured. The patient was in excellent condition at the end of the operation, but died one week later of meningitis.

The first problem in this case was to make the diagnosis. This was not an easy one, though it was fairly obvious that it was diffuse manifest suppurative labyrinthitis, complicated by, or immediately threatened with meningitis. The caloric and fistula tests, applied to the left ear, were negative. That is, no nystagmatic reactions were induced by them. This was very significant. Inasmuch as the labyrinth was destroyed, as shown by (*a*) the complete deafness upon the affected side, and (*b*) the caloric test, did not produce vestibular response (because the static labyrinth was destroyed), I made a diagnosis of diffuse manifest suppurative labyrinthitis, complicated by meningitis. I differentiated it from diffuse serous labyrinthitis, because in the latter disease neither the caloric nor fistula test induced vestibular reaction. Furthermore, meningitis was suggested by the unequally contracted pupils, slightly exaggerated knee-jerk, and severe headache, etc. The atrium of the intracranial invasion was probably through the sheath of the eighth nerve, *via* the internal auditory canal or through some other point on the posterior wall of the pyramid.

The second problem was the indications for treatment. Obviously local and systemic remedies were out of the question. A general law relating to acute infection is that free drainage of an infected cavity will usually result in a cure of the infectious process. In this case the infection was in the tympanic cavity, the mastoid cells, labyrinth, and the cranial cavity around the opening of the internal auditory canal on the posterior wall of the petrous portion of the temporal bone. To establish drainage of these areas it was necessary to do a radical mastoid operation, and a labyrinth operation, which would expose the meninges as deep as the internal opening of the internal auditory canal. Meningitis is as easily cured as peritonitis, provided equally good drainage can be established. This is possible at the very inception of meningitis in this region, if the Jansen-Neumann operation is performed, though if the operation is delayed the deeper arachnoid spaces become filled with coagulated serum and pus, which will not drain away, no matter how extensive the opening may be made. Hence, if

the operation is performed early enough a cure may sometimes be expected, as the reported cases show. If, however, the meningeal inflammation has spread much beyond the area immediately surrounding the atrium of infection a cure should not be expected, as drainage cannot be fully established. In this case I was in some doubt as to the extension of the meningitis, though the pupils were unequal, the knee-jerk slightly exaggerated, and headache severe. There was, however, a possibility of opening and draining in time to avert a fatal issue. I therefore decided to do a combined radical mastoid and Jansen-Neumann labyrinth operation at once. The subsequent development of the case, and the death of the patient, justified my earlier fears as to the extension of the meningitis. This case illustrates the extreme danger of delaying the labyrinth operation beyond the time of the inception of meningitis. Indeed, it would have been safer in this case to have operated as soon as total destruction of the labyrinth occurred, as shown by the total loss of hearing, rather than to have waited for the inception of the meningitis. The mortality rate in such cases could be greatly lowered if this were always done. In diffuse manifest suppurative labyrinthitis, with total loss of hearing and the absence of caloric and fistula reactions, a combined mastoid and labyrinth operation would probably lower the mortality rate. Spinal puncture would have given positive information as to the presence of suppurative meningitis, but this was not done because a suitable hollow needle was not available, as the patient was in a village far removed from the base of supply.

Total destruction usually occurs at the time of the "attack," consisting of nystagmus to the unaffected side, giddiness, nausea and vomiting, and disturbance of equilibrium. Little or no warning of the impending disaster is given. The case, just previous to the attack, was either one of chronic otorrhea or mastoiditis, or acute mastoiditis, with or without circumscribed labyrinthitis. Immediately after the "attack" occurred the hearing should have been tested with the noise apparatus, and if total deafness was shown, and especially if there was no reaction to either the caloric or fistula test, the labyrinth operation should have been considered at once. Delay only invited danger. The operation could have done no harm, as the hearing was already destroyed, and it might have saved the life of the patient. The patient would almost certainly have died without the operation, and might have been saved by an *early* labyrinth operation.

Case III.—Mr. Y., aged thirty-four years, complained of recurring attacks of dizziness upon stooping, quick movements of the head, and other unaccustomed movements. He occasionally had attacks of even greater severity independent of the stooping or jarring movement of the head and body. He was suffering from one of the attacks when I first saw him. The attacks were characterized by vertigo, nausea, and vomiting, staggering gait, and spontaneous nystagmus. The nystagmus was directed to either side, especially when the eyes were turned in either direction, though it was stronger toward the affected

side. When looking to the right the nystagmus was to the right, and when looking to the left the nystagmus was to the left, though it was stronger when looking to the right. The nystagmus was rotatory in type. There had been discharge from the right ear since childhood. The vertiginous attacks had occurred at intervals for four or five years.

The rotation test showed both labyrinths to be functionating normally. An examination of the ears showed the left to be normal. In the right ear there was a total loss of the membrana tympani, malleus, and incus. A thin, purulent secretion filled the fundus meati. There was no mastoid tenderness, though the skiagraph, subsequently made, showed extreme cloudiness of the mastoid cells, and of the antrum. The hearing was impaired in the right ear, the whispered voice being heard five feet. Weber lateralized to the right; Rinne in the right ear was negative; in the left, positive. Tinnitus was present but not marked. Fistula symptom was present. The caloric reaction was present about equally in both ears.

The first problem in this case was the diagnosis. This was not difficult in view of the history of recurring attacks of giddiness and nausea upon stooping, and sudden or jarring movements of the head or body. Of much greater significance, however, were the attacks of giddiness, spontaneous nystagmus, etc., when resting quietly in a chair, and in bed. In addition to these phenomena, the positive fistula reaction left no room for doubt as to the nature of the disease. A diagnosis of circumscribed labyrinthitis with fistula was made.

The second problem was the treatment. Should it be expectant, local, or surgical? Inasmuch as the disease was localized and non-progressive, and the hearing intact, operation on the labyrinth was not considered, as the presence of hearing, even in the slightest degree, is a positive contra-indication to surgery of the labyrinth. The mastoid operation remained to be considered. It was obviously indicated insofar as the mastoid was concerned. The influence of the mastoid operation upon the future course of the labyrinth disease was taken into account. Experience has shown that with the proper precautions the mastoid operation may be performed without arousing the circumscribed labyrinth disease into greater activity, or causing it to spread and become diffuse. Without these precautions the mastoid operation might be very dangerous. Having demonstrated the nature of the disease, and that a bony fistula was present, I was fully aware of the dangers of the situation, and proceeded with the mastoid operation, with full confidence as to the favorable outcome of the same.

The precautions observed were (*a*) the avoidance of meddlesome probing of the labyrinth fistula, and (*b*) the avoidance of currettage of the inner wall of the tympanic cavity, especially as granulations were present. Traumatism of the inner wall of the bony wound was scrupulously avoided. As to the labyrinth, nothing was done. The radical mastoid operation was performed and a bony fistula, the size of a large grain of wheat, was found leading to the horizontal canal. Granu-

lations filled the bony fistula. Two years have elapsed since the operation and the vertiginous attacks have been less frequent and much less severe in degree. The probabilities are that the labyrinth disease will remain circumscribed and the toxic and congestive irritations will cease, and an ultimate recovery from the "vertiginous attacks" take place.

Case IV.—Female, aged twenty-three years. She was complaining of sudden and severe attacks of vertigo, nausea, and vomiting, prostration, and of falling to the right. Moving about and attempting to sit up in bed to take food increased the vertiginous attacks. She was very deaf in the right ear. At times the surrounding objects seemed to whirl about her.

Upon testing the right ear, with Barany's noise apparatus in the normal left ear, a remnant of hearing for the shouted voice was shown. Fistula symptom was present, but required two or three violent compressions of the air-bag to produce it. The caloric reaction in the affected ear was present but weakened. Spontaneous nystagmus to the sound ear (sign of destruction disharmony) was present.

There was a history of several vertiginous attacks during the past six years, though they were much less severe than the present one. Her hearing previous to the recent severe attack was good, though somewhat impaired. She has had otorrhea with occasional mastoid pain and tenderness for eleven years. Eleven years ago she had an attack of tonsillitis, which was followed by acute otitis media, eventuating in chronic otorrhea, which has been accompanied by the aforesaid vertiginous attacks at varying periods of time.

The first problem in this case was the diagnosis. In view of the fact that deafness, while profound, was not total, and that there was a history of recurrent vertiginous attacks of moderate degree of severity during the preceding eleven years, that fistula symptom was present, that no previous operation had been performed upon the left ear, and that the present attack was severe and attended by almost total deafness, I made a tentative diagnosis of diffuse serous labyrinthitis. Had diffuse suppurative labyrinthitis been present the loss of hearing would have been both sudden and complete. The presence of some hearing and of static function in the affected ear ruled diffuse manifest suppurative labyrinthitis out of consideration. The only other type of labyrinthitis attended by almost total deafness of sudden development, is the serous variety. Hence a tentative diagnosis of diffuse serous labyrinthitis was made. If the disease subsequently proved to be of the serous type, the hearing would gradually be restored, an occurrence which could never happen in diffuse manifest suppurative labyrinthitis.

Within five days the vertiginous symptoms had almost disappeared and within two weeks the hearing was restored to such an extent that the whispered voice would be heard at three feet. There remained no longer any doubt as to the serous nature of the labyrinthitis. Operation in this case was not considered.

CHAPTER LI

SURGICAL DISEASE OF THE LABYRINTH

GENERAL REMARKS ON LABYRINTHITIS

Suppurative inflammation of the labyrinth is one of the most serious menaces to life, hence an accurate description of its characteristic phenomena should be delineated for the enlightenment of those who practise medicine and surgery. Unfortunately observations have thus far been too few to enable one to give an absolutely correct and dependable clinical picture of the infectious processes as they occur in the labyrinth. Indeed, their manifestations are so various in different individuals, and in the same individual at different times, that the task becomes all the more difficult and complicated. Then, too, the prognostic significance and the therapeutic indications attending each of the manifestations of labyrinthitis render it a most difficult and intricate problem. The advancement of knowledge along these lines, has, however, been sufficiently definite to warrant a fairly accurate clinical definition of the various labyrinthine inflammatory diseases. There is, however, still so much unknown as to the pathology, and even the physiology of the labyrinth, that we do not feel warranted in laying down hard-and-fast rules in reference to the description of the various clinical manifestations of labyrinthitis, or as to the treatment. The student of this subject should first learn all he can about the anatomy, physiology, and functional tests of the labyrinth, and then study the clinical observations recorded in the literature, and, having mentally digested them, he is prepared to treat them with a relative, if not an absolute, degree of inteliigence. If, in the following descriptions of the various types of labyrinth disease, the author should appear to speak too dogmatically, it may be said in extenuation that medical history is largely made up of dogmatism, to be repented of today and redogmatized tomorrow.

Of all the forms of labyrinthitis, acute diffuse suppurative labyrinthitis is attended by the most immediate and serious consequences. It always means permanent total deafness in the affected ear, and often the death of the patient. Hinsberg has estimated that 1 in every 100 cases of suppurative middle-ear disease develops into suppurative labyrinthitis. Von Stein, in 420 cases, found suppurative labyrinthitis ten times, or in 2.2 per cent. of the cases. As many of the intracranial diseases are due to labyrinthitis, this disease becomes a subject of grave clinical importance. Labyrinthitis in any form is

not of itself dangerous. The danger lies in the intracranial complications which are liable to occur. Of these diffuse meningitis (leptomeningitis) is the most serious, as it has a death rate of nearly 100 per cent. Cerebellar abscess has a death rate of more than 75 per cent.; hence the most important motive influencing the nature of the therapeutic measures to be adopted is to prevent the extension of the infectious process to the cranial cavity. This motive is apparently given a secondary place by some writers who advise waiting until actual intracranial involvement occurs before instituting radical remedial measures. Kerrison advocates waiting, in acute diffuse manifest labyrinthitis, for headache and continued high temperature before operating. Others advocate a radical exenteration of the mastoid process and labyrinth as soon as complete deafness and a negative caloric reaction occur, claiming by this method of procedure, a reduction of the death rate. Others contend that the labyrinth operation is of itself an added element of danger, as the brain, they say, is thereby exposed to infection. This claim is, it appears to me, not based upon either logic or observation. I am assuming that the operator is thoroughly qualified, and that he performs the Hinsberg or some other equally good operation, by which the labyrinth is adequately opened for drainage, and that meningitis is not already present.

While the other types of labyrinthitis are not of immediate danger to life, they are, nevertheless, possessed of the same potential dangers that are present in the acute suppurative manifest labyrinthitis; that is, they are all liable to become acute diffuse suppurative in type, in which even they present all the dangers of a primary diffuse suppurative labyrinthitis. In diffuse serous labyrinthitis, circumscribed labyrinthitis, and perilabyrinthitis, a labyrinth operation is not usually indicated so long as each remains true to its type, but should they become converted into diffuse manifest suppurative labyrinthitis, the indications for operative treatment are identical with those of primary diffuse manifest suppurative labyrinthitis.

The question seems therefore to resolve itself into the following: *Is the death rate in acute diffuse suppurative labyrinthitis influenced by any method of operation; and if it is favorably influenced, or if it is unfavorably influenced by any method or operation, by which method is it either favorably or unfavorably influenced?*

We can, perhaps, arrive at a more correct conclusion if we reason by analogy than if we reason from statistical data, as these are necessarily limited. We have learned through the reports of many cases of uncomplicated chronic mastoiditis, that complete recovery is very favorably influenced by the proper operative technique. We have, furthermore, learned by experience that improved technique in mastoid surgery has advanced with experience, and that the number of cases requiring a second operation has correspondingly diminished. Fifteen years ago it was frequently reported through the literature that 25 per cent. of the radical mastoid operations failed to produce the desired results.

Experience and improved technique have reduced the failures to about 5 per cent. Indeed, we can now effect cures in the same class of chronic disease by the various types of the modified radical operation. If the Jansen-Neumann and Hinsberg operations give good results now, further experience will improve the technique, and still further enhance the good results.

Let me again emphasize certain elementary facts: (*a*) So long as a vestige of hearing is present the case is not one of acute diffuse manifest suppurative labyrinthitis, but is either circumscribed, serous, or some other type of labyrinthitis, and a labyrinth operation is contraindicated.

(*b*) When the deafness is complete, as shown by Barany's noise apparatus, the case is probably one of acute diffuse manifest suppurative labyrinthitis, though it may be diffuse serous labyrinthitis, as may be shown by the presence of caloric reaction, or the fistula symptom.

(*c*) Complete loss of hearing and the absence of caloric and compression reactions in the affected ear means, therefore, that the patients' life is in jeopardy from impending meningitis or cerebellar abscess.

(*d*) Meningitis and cerebellar abscess may be prevented by an early suitable operation, and the life of the patient spared.

(*e*) If, on the contrary, we wait for signs of intracranial involvement before operating, the death rate in operated cases will be nearly 100 per cent.

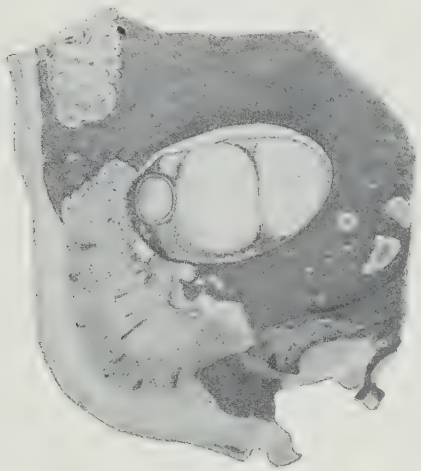
The whole problem as it appears to me is this: An immediate diagnosis of acute diffuse suppurative labyrinthitis followed by an immediate combined mastoid and labyrinth operation will prevent a fatal issue in many of the cases; whereas a delay of from three to seven days, for the purpose of detecting the advance of the infectious process to the cranial content, will result in the subsequent death of many of the patients who might otherwise have been saved.

CIRCUMSCRIBED LABYRINTHITIS

Circumscribed labyrinthitis is an infectious inflammatory disease of a circumscribed area of either the vestibular or the cochlear portion of the labyrinth, or of both together. The vestibular apparatus (utricle, saccule, and semicircular canals) is more often affected than the cochlea. Occasionally both the vestibular and cochlear apparatuses are involved. Circumscribed vestibular-apparatus disease is a less dangerous process than circumscribed cochlear disease, or circumscribed vestibulocochlear disease. When the cochlea is the site of a circumscribed inflammation it is much more liable to become diffuse and terminate in the complete destruction of the whole labyrinth. This is especially true if the initial infection in the tympanum is virulent, and invades the cochlea. If the infection is mild, and slowly perforates into the cochlea it may become localized indefinitely.

The external limb of the horizontal canal is the most frequent site of circumscribed labyrinthitis, either with or without fistula; fistula is, however, usually present. Circumscribed labyrinthitis, localized in the cochlea has been studied histologically (microscopically) more frequently than the same process limited to the horizontal canal. This is so because circumscribed cochlear labyrinthitis more often goes to postmortem (Fig. 494).

Fig. 494



Circumscribed labyrinthitis. (Ruttin, Annals of Otology, Rhinology, and Laryngology.)

Etiology.—The usual cause of this disease is a chronic suppurative otitis media, mastoiditis, or cholesteatoma. Fistula of the bony wall of the labyrinth usually occurs over the membranous horizontal canal as it lies in the zone of stress (the aditus ad antrum) where the purulent secretion passes over it flowing from the antrum into the middle-ear cavity, and where the cholesteatoma causes pressure necrosis of the external labyrinth wall. The lymph spaces around the exposed membranous labyrinth subsequently become infected, but become walled off by protective granulation tissue. The infection may also extend into the membranous canal, in which event the process is liable to become diffused through the entire labyrinth, though it, too, may become walled off by round-cell infiltration. Fistula may also occur at the oval and round windows, the promontorium, and at the tympanic mouth of the Eustachian tube. Bony fistula in the last four positions may involve the membranous and nervous cochlea, and the condition become much more serious, as the entire labyrinth may become involved.

The Rationale of the Symptoms.—The character of the symptoms of circumscribed labyrinthitis are largely determined by the following factors:

(*a*) The presence of fistula.

(*b*) The absence of fistula.

(*c*) The location of the fistula.
(*d*) The stimulation of the cristæ.
(*e*) The inhibition of the cristæ.
(*f*) The circumscribed area of the labyrinth involved.

The Presence of Fistula.—The presence of fistula is usually attended by the fistula symptom when either compression or aspiration of air in the external auditory meatus is performed (see Fistula Test). The condensed or rarefied air either compresses or expands the membranous labyrinth at the point of bony fistula, and thereby causes a molecular movement of the endolymph in the semicircular canals, in a direction either from the utricle through the ampulla to the canal, or from the canal through the ampulla to the utricle. The direction of the impact of endolymph determines the direction of the nystagmus. If the impact is from the utricle through the ampulla to the canal, there is a weak nystagmus to the opposite side (Plate XXII); whereas if it is from the canal through the ampulla to the utricle, there is a stronger nystagmus to the same side (Plate XXI). If it is to the right by compression, it will be to the left by aspiration, and *vice versa*. Fistula symptoms (nystagmus upon compression or aspiration) may be negative (absent) even when fistula is present; that is, the endolymph may be coagulated and difficult to displace by either compression or aspiration, though in some cases of this type several forcible compressions of the bulb will elicit nystagmus. Fistula symptoms may also be absent or negative when the fistula is present, if the localized inflammatory process has destroyed the functional capacity of the crista nearest the fistula. Localized destruction of the cristæ is, however, comparatively rare.

The Absence of Fistula.—The absence of fistula in circumscribed labyrinthitis is more rare than the presence of fistula. It is readily conceivable, however, that localized infection and inflammation may become established in either the perilymph or endolymph spaces without fistula. Infection may occur through either the blood or lymph vessels. The absence of bony fistula is, of course, not attended by fistula symptoms (nystagmus) upon either compression or aspiration; that is, it is negative. If, therefore, there is a history of recurrent attacks of spontaneous nystagmus, vertigo, nausea, vomiting, and ataxia, and during the stage of quiescence fistula symptom cannot be elicited, fistula is probably not present.

The clinical significance of "fistula symptom present," and of "fistula symptom absent" in circumscribed labyrinthitis is as follows: In those cases in which fistula symptom is present the inflammation is somewhat more liable to become diffuse than it is in those in which it is absent.

The Location of the Fistula.—The influence of the location of the fistula upon the expression of the induced nystagmus during the periods of quiescence is quite characteristic, and the diagnostic and prognostic deductions to be drawn therefrom are often of the greatest value. If for example, the bony fistula is in the external arm of the right hori-

zontal canal, the compression test will cause a flow of endolymph from the canal through the ampulla to the utricle. The impact of the endolymph is therefore against the canal half of the crista ampullaris of the horizontal canal. The impact excites a nervous impulse in the hair cells of the crista which is transmitted through the right Deiters' nucleus to the adductor muscles of the right eye, and the abductor muscles of the left eye (Hoegyes' law), thereby causing a slow conjugate movement of both eyes to the left. This is immediately followed by a corrective impulse in the right cortical centre, which is transmitted to the antagonists of the muscles stimulated by the vestibular nervous impulse, and the eyes are quickly turned to the right; that is, the compression induces nystagmus to the same side stimulated, or to the right in the example cited (Plate XXI). Aspiration would, of course, induce nystagmus to the opposite or left side. The significance of these reactions is that the fistula is in the horizontal canal, a relatively safe location. If, on the contrary, compression is accompanied by induced nystagmus to the opposite side (left in this instance), it signifies a bony fistula anterior to the ampulla of the horizontal canal, in either the oval window, round window, or promontorium (Plate XXII). As previously stated, fistula in either of these regions is more often followed by diffuse suppurative labyrinthitis and total destruction of the static-auditory labyrinth. And, furthermore, diffuse suppurative labyrinthitis is often attended or followed by either meningitis or cerebellar abscess, and death. The direction of the fistula nystagmus upon compression is, therefore, of important diagnostic and prognostic value.

The Cristæ Either Stimulated or Inhibited.—We will now turn our attention to the spontaneous nystagmus, present during one of the so-called vestibular attacks which occur at more or less frequent intervals during the course of circumscribed labyrinthitis. In most cases the direction of the spontaneous nystagmus is to the affected side, which signifies that the cristæ are stimulated, *i. e.*, the "signs of stimulation disharmony" are present, as in induced nystagmus. In other cases the spontaneous nystagmus is to the unaffected side, signifying an inhibition or destruction of the impulses from the crista of the semicircular canal of the affected labyrinth, *i .e.*, signs of destruction disharmony are present. Inhibition of function signifies a greater involvement of the crista (the end-organ of the vestibular nerve) than does stimulation of function. Stimulation disharmony is, however, more often present (Plates XXI and XXII).

The Area of Involvement is Circumscribed.—As the name of this type of labyrinthitis signifies only a circumscribed area of the labyrinth is involved. The symptoms are therefore characteristic of the circumscription. If, for example, the whole vestibulocochlear apparatus is functionally destroyed, as in acute diffuse manifest suppurative labyrinthitis, or the fifth degree of diffuse serous labyrinthitis is present, the spontaneous nystagmus will be to the opposite side (Plate XXV), and the hearing totally destroyed in the affected ear. In circumscribed

labyrinthitis, the spontaneous nystagmus (during the vestibular attacks) is usually to the same side (Plates XXI and XXII), and the hearing is but slightly or moderately diminished. Even in those cases in which the spontaneous nystagmus is to the opposite side, or to either side when looking to one side and then to the other, the hearing is but slightly or moderately diminished. The presence of any degree of hearing and spontaneous nystagmus to either or both sides is characteristic of circumscribed labyrinthitis. In serous labyrinthitis of a lesser degree than the fifth (see Serous Labyrinthitis), there may be some hearing present, but the spontaneous nystagmus is always to the unaffected side (Plate XXV), that is, the signs of destruction disharmony are always present.

Symptoms.—Having discussed the rationale of the symptoms of circumscribed labyrinthitis, only a brief discussion of the symptoms in relation to the course of the disease will be necessary.

Circumscribed labyrinthitis is usually attended by recurring attacks of giddiness upon stooping, quickly turning the head, jarring movements, as in cycling or motoring over rough roads, jumping on and off cars, or other sudden and unaccustomed movements. What is of greater diagnostic significance in these patients is that in addition to the dizziness excited by sudden movements, *the vestibular attacks occurring independently of sudden or jarring movements of the head. They occur while the patient is sitting in a chair, or lying quietly in bed, or even while asleep.* During these attacks, vertigo, giddiness, nausea, and spontaneous nystagmus are present, and are more violent and of longer duration than the attacks occurring upon sudden jarring movements. The spontaneous nystagmus may be to either or both sides. It is rotatory in character, and is made more manifest by turning the eyes to the quick component. The turning and caloric tests usually show both labyrinths to be functionating normally, though when the endolymph is coagulated the reaction may be absent or difficult to induce in the affected ear. After the "attack" the symptoms disappear and only recur with the succeeding "attacks." During the vestibular attacks the spontaneous nystagmus may be to the diseased side or to the normal side, or to both sides, when looking to the outer angle of either eye, though it is usually stronger to the diseased side. The hearing may be impaired but not destroyed; indeed, it may be very good. The hearing distance for the whispered voice is generally reduced to from $\frac{1}{2}$ to 1 meter, though in a case reported by Ruttin, it was 8 meters. The turning and caloric reactions are usually well pronounced. Fistula symptom is usually present, though it may be absent, as when the endolymph is coagulated, and when cholesteatomatous plugs or granulations block the fistula. When the fistula symptom attending the compression test is absent it is difficult to make a diagnosis. In many instances in which the attacks are of short duration the condition is one of circulatory disturbance rather than of circumscribed labyrinthitis. This is especially true of those cases in which the attacks only occur upon sudden or jarring move-

ments of the head. In true circumscribed labyrinthitis the patients have the attacks independent of such movements of the head, as when lying quietly in bed.

Subjective noises are present in about one-third of the cases. In 50 cases reported by Ruttin they were present in 17 and only severe in 2.

Diagnosis.—(*a*) Vestibular symptoms occur in attacks at more or less frequent intervals and are excited by sudden or jarring movements of the head and body. During the vestibular attacks the spontaneous nystagmus may be either to the affected or to the unaffected side, or to both sides.

(*b*) The intervals between the attacks are free from vestibular symptoms.

(*c*) The hearing is impaired but not destroyed.

(*d*) Caloric reaction is present (positive) in the affected ear.

(*e*) Turning reaction is present (positive) in the affected ear.

(*f*) Fistula symptom (nystagmus upon compression) is usually present or positive in the affected ear, though it may be absent.

(*g*) Vertiginous attacks occur when the patient is physically quiet (very significant).

Indications.—A mastoid operation may be indicated. The labyrinth should not be operated, as hearing is present, and in the event of the disease becoming acute diffuse manifest suppurative labyrinthitis, ample warning of impending meningitis is usually given by the occurrence of complete deafness and the loss of all vestibular reaction to the various tests. When this occurs an immediate operation upon the labyrinth should be considered, though serous labyrinthitis is still a possibility. During the mastoid operation in cases complicated by known circumscribed labyrinthitis, great care should be exercised to avoid probing, or other meddlesome interference with the fistula and inner wall of the tympanum and antrum. There is always a strong temptation to probe the fistula and observe the slow movement of the eyes to the opposite side. By thus probing the fistula the circumscribed area of inflammation may be disturbed, and a diffuse induced serous or suppurative labyrinthitis excited. Under general anesthesia the quick component of the nystagmus will not occur, as the cortical centre from which the impulse arises is paralyzed by the anesthetic; hence, upon probing the eyes remain fixed in the position of the slow component. The cerebellar centrums (Deiters', Bechterew's and angular nuclei) belong to the lower order of reflex organs and are not paralyzed by the anesthetic; whereas the cortical reflex area, being a higher order of reflex centre, is paralyzed by the general anesthetic.

Treatment.—Do a radical mastoid operation, if required, and avoid meddlesome interference with the fistula and inner wall of the tympanum and antrum. Do not curette these areas. Do not remove granulations from the inner wall of the tympanum. Do not operate upon the labyrinth.

ACUTE DIFFUSE SEROUS LABYRINTHITIS

Acute diffuse serous labyrinthitis is a condition superimposed upon a preëxisting circumscribed labyrinthitis, or it occurs as a primary labyrinth disease secondary to acute aural disease, or to surgery of the mastoid. Some cases are therefore preceded by the vestibular attacks of circumscribed labyrinthitis, and intervals of freedom from vestibular symptoms (see Circumscribed Labyrinthitis). Others have a sudden onset with very severe vestibular disturbances, known as the "signs of destruction disharmony." These cases often closely resemble acute diffuse suppurative labyrinthitis, though some vestibular reaction can usually be elicited by the fistula or caloric test.

Etiology.—Diffuse serous labyrinthitis may be caused as follows:

1. It may be secondary to a circumscribed labyrinthitis, and it sometimes occurs without a known cause. That is, it appears to occur spontaneously, or following a cold in the head. It is sometimes classified as a secondary labyrinthitis because it is secondary to circumscribed labyrinthitis.

2. It is sometimes caused by a severe inflammatory reaction following the mastoid operation, the patient being previously affected by circumscribed labyrinthitis. When thus caused the symptoms appear at from the first to the fifth day after the mastoid operation. This is sometimes called diffuse serous induced labyrinthitis.

3. It may also follow circumscribed labyrinthitis, when a direct injury of the labyrinth is inflicted during a mastoid operation. When thus caused the symptoms appear at once, that is, when the patient awakens from the anesthetic he is giddy, nauseated, and has spontaneous nystagmus. The "signs of destruction disharmony" are present.

4. It sometimes follows acute otitis media in which the inflammation extends through the intact labyrinth wall.

5. Edema of the middle ear sometimes extends into the labyrinth.

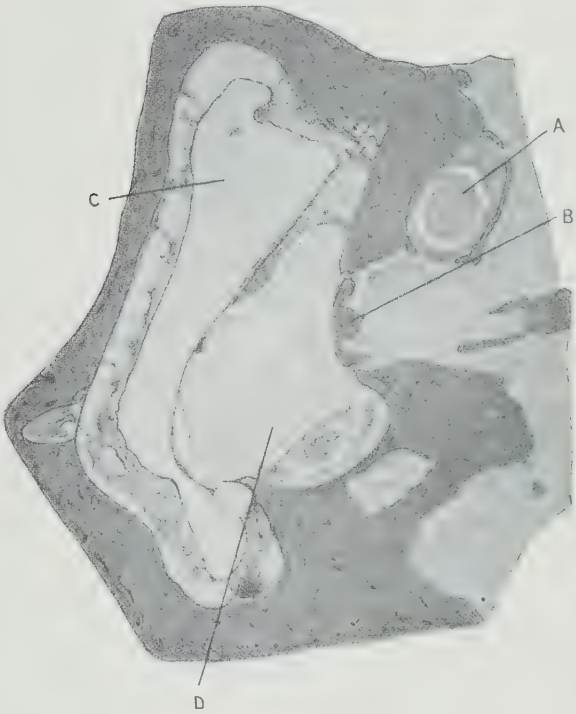
6. Chronic otitis media occasionally causes serous labyrinthitis by extension.

7. The absorption of the toxic products of the bacterial activity in the middle ear and mastoid cells is probably the most common cause of serous labyrinthitis (Fig. 495).

Symptoms.—When the diffuse serous exudate occurs there is a sudden arrest of function, *i. e.*, there is a rapid and marked diminution of hearing upon the affected side, and nystagmus to the sound side. Nausea, vomiting, vertigo, and disturbance of equilibrium are also present in varying degrees of severity. The patient involuntarily lies on his sound side, as this compels him to look toward the slow component of the nystagmus when the eyes are open; that is, he looks away from the pillow in the direction of the slow component of the spontaneous nystagmus, and this position of the eyes suppresses the vertiginous symptoms. These symptoms are often severe, lasting from three to

five days. The labyrinth is, however, not usually destroyed, as is shown by the subsequent restoration of its functions. The tonus of the affected static labyrinth is either greatly diminished or totally lost for a few days. Therefore the unaffected static labyrinth predominates in tonus impulses, and the spontaneous nystagmus is to the normal side (Plate XXV). As the serous exudate is absorbed the nystagmus and vertiginous attacks subside and finally disappear.

Fig. 495



Diffuse serous labyrinthitis. Section through the vestibule. *A*, facial nerve; *B*, stapes; *C*, utricle intact; *D*, exudate in the cysterna perilymphatica. (Ruttin, Annals of Otology, Rhinology, and Laryngology.)

Indeed, if the serous exudate persists for many days or a few weeks, extralabyrinthine compensation would cause the symptoms to disappear. The serous type may become suppurative and cause complete destruction of the labyrinth and eventuate in meningitis or abscess, and death. This termination is comparatively rare. I have observed several cases in the latent stage in which the hearing in the affected ear was completely destroyed, but in which some vestibular response to the caloric test remained. Such cases are generally classified as having been serous labyrinthitis of the fifth degree. It has also been stated that serous labyrinthitis has no latent stage. If these cases were serous in the acute stage, this disease has a stage of latency just

as truly as has the diffuse manifest suppurative type of labyrinthitis. For practical clinical purposes these cases will be classified as sequellæ of a diffuse serous labyrinthitis.

Severe vertigo, nausea, vomiting, and loss of equilibrium occur at the onset of the disease and gradually diminish in intensity. The hearing is the first function to disappear. The patient instinctively lies upon his sound side, as this causes him, when his eyes are open, to look away from the pillow toward the slow component of the nystagmus, a position of the eyes which inhibits the vestibular symptoms. No amount of persuasion will induce him to lie upon his affected side during the height of the vestibular symptoms, as this posture would, when the eyes are open, cause him to look toward the quick component of the nystagmus, and thus intensify the vestibular symptoms.

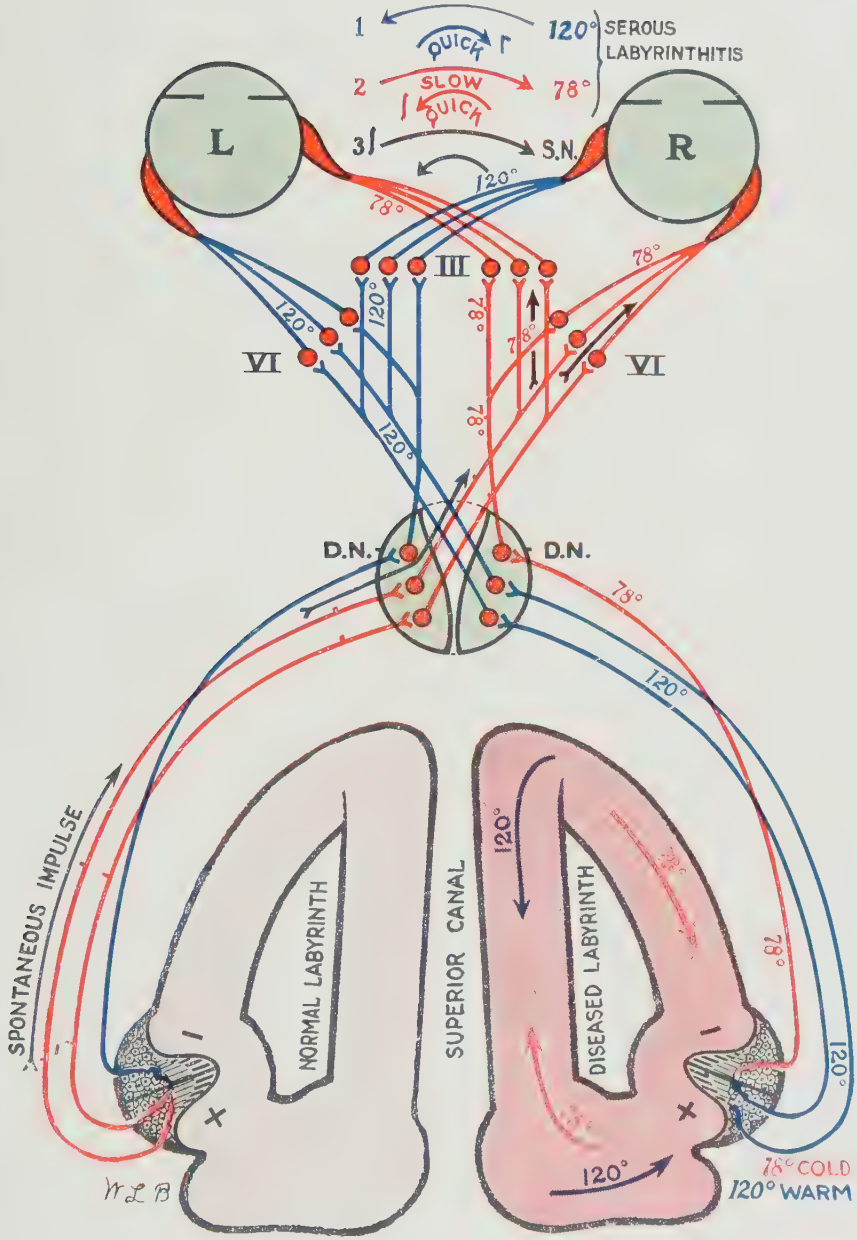
Should the patient attempt to stand with his heels approximated, head erect, and face forward, he would fall toward the diseased side, that is, toward the slow component of the nystagmus. With his face toward the right shoulder (assuming the right labyrinth to be diseased) he would fall backward in the direction of the slow component. With his face toward the left shoulder, he would fall forward, the direction of the slow component of the nystagmus, while the head is in this position.

Spontaneous nystagmus is rotatory and to the sound side (Plate XXVII). The hearing is more impaired than the vestibular function, which, according to Ruttin, is because the perilymph of the cochlea (functional element) is more superficially located than the endolymph (functional element) of the vestibular apparatus. The hearing is greatly impaired, and, indeed, in most cases is temporarily abolished in the affected ear. So long as the hearing is completely suppressed it is difficult to differentiate the disease from acute diffuse manifest suppurative labyrinthitis, in which the hearing is permanently destroyed. In diffuse serous labyrinthitis some static function is, however, usually more or less retained, and when this is the case the disease is readily distinguished from diffuse manifest suppurative labyrinthitis. In most cases the restoration of hearing is almost to normal. The reactions to the turning, fistula, and caloric tests are more or less modified, and in severe cases are temporarily abolished. When all reactions are abolished it becomes difficult, indeed, to differentiate it from acute diffuse manifest suppurative labyrinthitis, as the symptoms are identical. In the course of a few days, however, the hearing returns, and the reactions to the caloric, turning, and fistula tests are reëstablished except in very severe cases. This, of course, is never true of diffuse suppurative manifest labyrinthitis, as,

PLATE XXVII

There has been sudden and complete loss of hearing in the right ear. The warm caloric test induces nystagmus to the same (right) side. The cold caloric test induces nystagmus to the opposite (left) side, showing the disease to be serous labyrinthitis rather than diffuse suppurative labyrinthitis.

PLATE XXVII



The Caloric Tests in Serous Labyrinthitis.

in that disease, both the static and auditory labyrinths are completely destroyed. Whereas, in diffuse serous labyrinthitis the functions of the labyrinth are for a time partially or wholly suppressed, but the labyrinth is not usually destroyed, though complete destruction of the cochlea, and almost complete destruction of the static labyrinth may occur. In the subsequent course of such cases the patient may have recurrent attacks of giddiness. When the functions of the labyrinth are completely suppressed the case is liable to terminate in diffuse suppurative manifest labyrinthitis, or at least in complete destruction of the cochlea.

Every case of acute diffuse manifest labyrinthitis with apparent loss of cochlear and static function should be carefully tested before deciding upon operation, as it may be serous in type, and may recover with restitution of function. In severe diffuse serous labyrinthitis, one or more static reaction is usually, though not always, present. In diffuse manifest suppurative labyrinthitis all static and auditory functions are permanently lost. In order to detect the presence of possible remaining vestibular function, the tests should be methodically applied as follows:

1. Make the caloric test. If the reaction is negative the hearing, as a rule, is also destroyed (Plate XXVII). The static labyrinth may not be completely destroyed but may retain some function in which even the ratio of the nystagmus by the turning tests to the right and left will be more than 1 to 2 (see Plate XXV).

2. If there is no reaction to the caloric test, make the turning test (Plate XXVIII). This may or may not induce reaction. If the static labyrinth is totally destroyed, turning toward the sound ear will induce nystagmus of one-half the duration of that induced by turning toward the affected side.

3. If the turning test is negative make the fistula test, which arouses the strongest reaction of all the tests, and if fistula is present, and the case is one of diffuse serous labyrinthitis, there is a positive reaction, *i. e.*, nystagmus is elicited. If this and the other tests fail to induce nystagmus, and the patient is totally deaf in the affected ear, the case is probably one of diffuse suppurative labyrinthitis, though if fistula is absent the diagnosis cannot be thus positively made; it may be of the serous type.

Serous labyrinthitis, even when severe, rarely invades the intracranial content.

The loss of function usually occurs in the following order:

1. Hearing.
2. Caloric reaction.
3. Turning reaction.
4. Fistula reaction.

PLATE XXVIII

Sudden and complete loss of hearing. Turning to the right induces after-nystagmus to the left. Turning to the left induces after-nystagmus to the right; hence the disease is serous rather than diffuse suppurative labyrinthitis. Spontaneous nystagmus is to the opposite or healthy side.

When serous labyrinthitis is due to a reaction inflammation following a mastoid operation the "signs of destruction disharmony" follow in from twelve to seventy-two hours. When, however, it is due to direct injury of the labyrinth during the mastoid operation, the "signs of destruction disharmony" appear at once, *i. e.*, as soon as the patient recovers from the anesthetic.

The "signs of destruction disharmony" are nystagmus, nausea, vomiting, vertigo, and disturbance of equilibrium, and they are due to the sudden loss of function on one side, thus leaving a preponderance of nervous impulses from the sound labyrinth. If the right labyrinth is affected the impulses from this labyrinth are suddenly suppressed, and remain normal in the sound or left labyrinth. The left labyrinth, therefore, pulls the eyes (slow component) to the right, and the quick component to the left or sound side immediately follows. The nystagmus is to the sound side. The patient has a tendency to fall to the affected side, the direction of the slow component when looking straight ahead. When the face is turned over the right shoulder, the fall is backward. When the face is turned over the left shoulder the fall is forward.

In disease of the labyrinth it is practically universal to find the "signs of destruction disharmony" present (except in circumscribed labyrinthitis); whereas in testing normal ears, we elicit the "signs of stimulation disharmony."

Ruttin gives the following table of symptoms as typifying the five degrees of severity of this disease:

Table of Degrees of Severity in Serous Labyrinthitis.—Always with less severe degrees of serous inflammation, one or the other of the functions of the labyrinth is present. Loss of function is customary in the following order:

1. Hearing reaction
   Caloric reaction
   Turning reaction
   Fistula symptoms
   } Present.
2. Hearing — Absent.
   Caloric reaction
   Turning reaction
   Fistula symptoms
   } Present.
3. Hearing
   Caloric reaction
   } Absent.
   Turning reaction
   Fistula symptoms
   } Present.
4. Hearing
   Caloric reaction
   Turning reaction
   } Absent.
   Fistula symptoms — Present.

5. The fifth degree, namely, total loss of function is impossible to differentiate from suppurative labyrinthitis, except by waiting for the return of the cochlear and vestibular functions. If the functions of

the affected labyrinth do not return, the disease is probably acute diffuse manifest suppurative labyrinthitis. If they do return, it is a case of diffuse serous labyrinthitis. If, in a given case of circumscribed labyrinthitis, symptoms of diffuse labyrinthitis suddenly arise, the diagnosis of diffuse serous labyrinthitis depends upon the demonstration of at least one function of the labyrinth.

Indications.—The radical mastoid operation is indicated after the manifest symptoms disappear. The labyrinth should not be operated. The mastoid operation may be postponed until the acute symptoms of serous labyrinthitis have subsided as there is no particular danger of extension to the cranium.

The same symptoms prevail in the induced as in diffuse serous secondary labyrinthitis, though they are more severe in character. In the induced cases there is no history of previous vertiginous attacks, as in serous secondary labyrinthitis (secondary to injuries, etc.). The vestibular attack is sudden and severe and the vertigo lasts longer than in secondary serous labyrinthitis. This is due to the fact that compensation has not been previously established, as it has in the diffuse serous secondary labyrinthitis, in which there were numerous vestibular attacks preceding the serous exudate. The cases secondary to circumscribed labyrinthitis have become more or less immune, *i. e.*, compensation has been more or less established; whereas in the induced cases compensation has not occurred. As previously stated, the symptoms are somewhat in proportion to the suddenness and completeness of the suppression of function. The symptoms gradually abate in intensity and finally disappear in from one to four weeks.

Treatment.—The mastoid operation with rest in bed may be performed to relieve the mastoiditis and otorrhea. Curettage of the antrum and tympanic cavity should be studiously avoided. Do not operated upon the labyrinth.

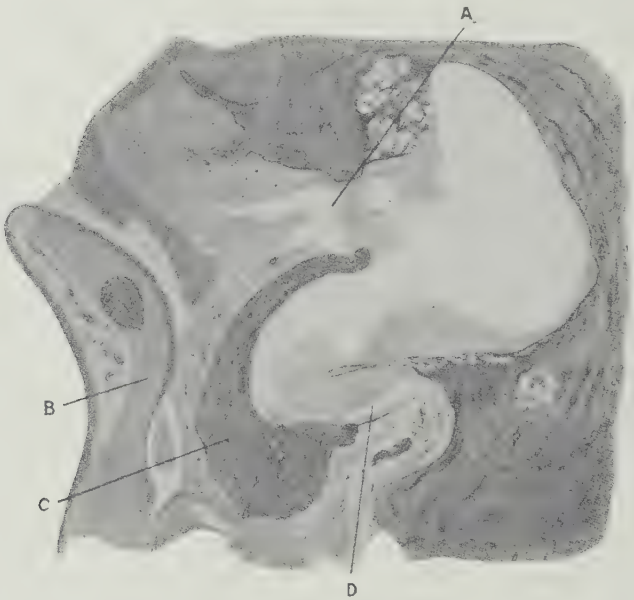
ACUTE DIFFUSE SUPPURATIVE MANIFEST LABYRINTHITIS.

Diffuse suppurative manifest labyrinthitis is characterized by sudden deafness in the affected ear, giddiness, nausea, vomiting, loss of equilibrium, and spontaneous nystagmus to the sound side (signs of destruction disharmony).

Etiology.—The causes of this form of labyrinthitis are acute mastoiditis, chronic mastoiditis, cholesteatoma, tuberculosis, syphilis, etc. It may follow circumscribed labyrinthitis, and diffuse serous secondary labyrinthitis. The causation is, however, more far-reaching than this; it often includes disease of the faucial and pharyngeal tonsils. I have had three cases which presented the following general history: (*a*) Acute tonsillitis, followed by (*b*) acute otitis media and mastoiditis; (*c*) during the acute stage of otitis media deafness, giddiness, nausea, and ataxia occurred, *i. e.*, acute diffuse suppurative labyrinthitis occurred; (*d*) a few days later meningitis developed; and finally (*e*) death supervened. The role of tonsillitis and adenoids in the

production of the labyrinth disease, though an indirect one, is of first importance, as disease of the tonsils and adenoids lies at the very foundation of aural disease, and as nearly all deaths of otitic origin are due to intracranial disease, we are, in the last analysis, driven to the deduction that indirectly disease of the tonsil and adenoids is the cause of labyrinthitis, meningitis, sinus thrombosis, and brain abscess, with their attendant high mortality (Fig. 496).

Fig. 496



Manifest purulent labyrinthitis. *A*, oval window, pus broken through; *B*, drum membrane· *C*, promontorium; *D*, round window broken through. (Ruttin, Annals of Otology, Rhinology, and Laryngology.)

Symptoms.—The symptoms of acute diffuse manifest suppurative labyrinthitis have their origin almost altogether in the sudden loss of balance or tonus between the two labyrinths. The suddenness of the loss is the determining factor. In exceptional cases the loss of tonus is so gradual that it is symptomless. In acute diffuse manifest suppurative disease of the labyrinth the whole labyrinth, cochlear and vestibular portions, are almost instantly destroyed. If the destruction should occur in both labyrinths simultaneously there would be no vestibular symptoms, as spontaneous nystagmus, ataxia, etc. As the destruction is usually limited to one side, or at least, does not often occur simultaneously on the two sides, there is a modified tonus in the affected labyrinth. The crutch (vestibular apparatus) being suddenly removed from one set of muscles, their antagonists act without their accustomed restraint, producing movements of the eyes, body, and extremities. A corrective reflex impulse is thereby excited

in a cortical reflex centre, and the eyes are quickly turned in the opposite direction, and incoördinate movements of the extremities occur. These movement-cycles are known as nystagmus and reaction movements. When spontaneous nystagmus occurs in disease of the ear, it is usually due to the sudden removal or diminution of the function of one vestibular apparatus. The labyrinth spaces are so small that the absorption of septic matter therefrom does not materially affect the temperature. The temperature present is, therefore, due to the absorption of toxic material from the middle ear and mastoid process rather than from the labyrinth; or if sinus thrombosis or meningitis is present it may be due to either of these conditions. In any event it is not a sign of progression within the labyrinth, but is a sign of continued infection extraneous to the labyrinth.

The symptoms of acute diffuse manifest labyrinthitis are generally referred to as the "signs of destruction disharmony;" whereas the symptoms of certain cases of circumscribed labyrinthitis and of congestion of the labyrinth are known as the "signs of stimulation disharmony."

The "Signs of Destruction Disharmony."—The "signs of destruction disharmony" are:

(*a*) Spontaneous nystagmus to the *sound* ear.

(*b*) Nausea and vomiting.

(*c*) Giddiness.

(*d*) The sense of external objects floating around the patient in the plane of the nystagmus, and in the direction of the slow component of the nystagmus.

(*e*) Ataxia or incoördinate movements of progression.

The foregoing are also the "signs of *stimulation* disharmony" with the following difference, *i. e.*, the spontaneous nystagmus is to the *diseased* side or ear. In diffuse suppurative labyrinthitis, say of the right ear, there is a total loss of static function in that ear; whereas there is normal static function in the left ear. Before the destruction the tonus was the same in both labyrinths; we will assume it to have been 20 potentialities. After the destruction of the right labyrint the tonus in the right labyrinth was 0, and in the left it remained normal, 20 potentialities. The nervous impulses from the left labyrinth being the stronger would therefore turn the eyes slowly to the right, according to Hoegyes' law, namely, Deiters' nucleus sends motor impulses to the adductors of the eyes of the same side, and to the abductors of the eye of the opposite side, thereby producing a conjugate movement of both eyes to the opposite side (slow component of the nystagmus). The cortical correction in the reverse direction immediately follows; hence spontaneous nystagmus to the healthy side occurs. If the nystagmus were due to stimulation of the cristæ of the affected labyrinth the nystagmus would be toward the affected side (Plate XX).

Deafness.—The deafness in this disease is usually sudden and complete in the affected ear. It occurs simultaneously with the "signs of

destruction disharmony," and is due to the same pathological process, *i. e.*, purulent inflammation extending throughout the cochlea. The deafness may not be obvious to either the patient or his companions, as the hearing in the other ear is not affected. Therefore, when the "signs of destruction disharmony" occur, the ears should be examined as soon as the condition of the patient permits. The signs of destruction disharmony at the onset are often very severe and compel the patient to go to bed. After several hours or a few days they usually subside sufficiently to allow the ears to be examined for vestibular function and hearing; indeed, the hearing may be tested earlier in the course of the disease. The test consists of the application of Barany's noise apparatus to the unaffected ear and addressing the affected ear with an increasingly louder voice, up to the shouting voice, or until it is ascertained whether or not hearing is present in that ear (see Functional Tests of Hearing). In this type of labyrinth disease the hearing is totally destroyed; hence, if the signs of destruction disharmony and total deafness are present, the disease is probably, though not certainly, acute diffuse manifest suppurative labyrinthitis. I say it is not certainly diffuse manifest suppurative labyrinthitis, because these symptoms may also be present in the fifth degree of diffuse serous labyrinthitis. Still another factor must be taken into account before a diagnosis can be made; indeed, two more factors must be considered, namely, (*a*) the presence or absence of vestibular reaction to artificial stimulation; (*b*) the subsequent restoration of hearing in some degree.

(*a*) If vestibular reaction in the affected ear is demonstrable the disease is not diffuse suppurative labyrinthitis but is serous labyrinthitis.

(*b*) If the hearing is restored at any future time the disease was not diffuse suppurative labyrinthitis but was serous labyrinthitis.

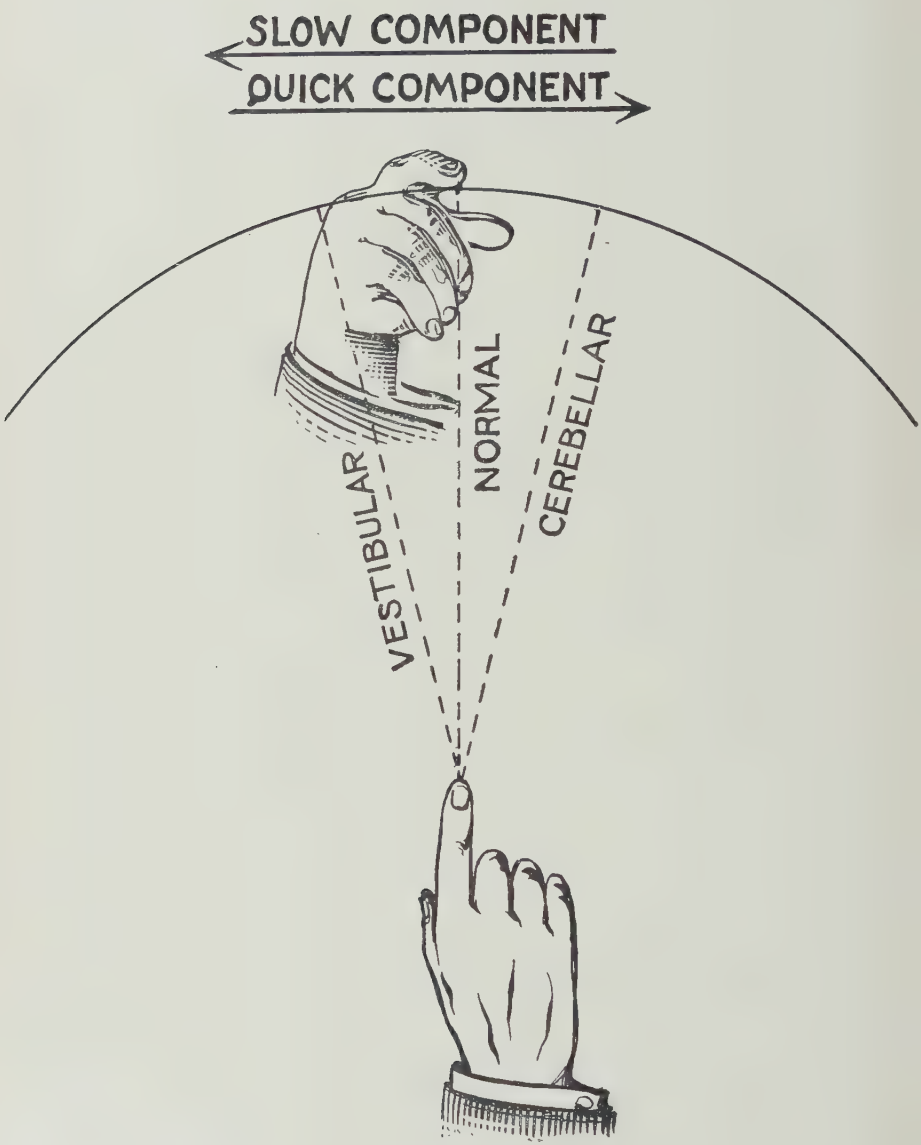
These conclusions are based upon the fact that diffuse manifest suppurative labyrinthitis is invariably attended by the complete destruction of the cochlear function, though it is conceded that some vestibular function may occasionally remain, as this organ has more resistance to degeneration from infection than the cochlea. If, therefore, the hearing never returns, and no vestige of vestibular function can be shown, the disease is diffuse suppurative in type; whereas, if any degree of hearing is restored the disease should be regarded as having been serous in character throughout its course.

Vestibular Reaction.—Vestibular reaction is usually abolished in the affected ear, though it may be present in exceptional cases for the reason expressed in the preceding paragraph. For clinical purposes all cases with total destruction of hearing in which the vestibular function is not totally destroyed may be classified as diffuse serous labyrinthitis of the fourth degree, though it is quite probable that some of them are suppurative in character. Thus classified such cases are to be treated non-surgically unless meningitis or cerebellar abscess subsequently develop. That the law in reference to the greater vulnerability

of the cochlea is not absolute was illustrated by a case reported recently by Dr. Ewing W. Day. This case had total destruction of the vestibular function and retained the cochlear function. The exceptional case does not, however, disprove the rule. Vestibular reaction is shown by the use of the caloric and fistula tests in the affected ear. As spontaneous nystagmus is already present, vestibular reaction can only be shown by increasing or diminishing the intensity of the spontaneous nystagmus. If some vestibular function remains, and the cold caloric test is applied to the affected (say right) ear, there will be a downward flow of endolymph in the superior canal, and the impact of the endolymph will be against the canal half of the crista ampullaris. This causes a nervous impulse to be given off which traverses the nerve tract (red) marked 78° in Plate XXVII, through which the impulses are carried to the adductors of the left eye and the abductors of the right eye, thereby causing a slow conjugate movement of both eyes to the right.[1] This is immediately followed by a corrective movement of both eyes to the left, the quick component of the induced nystagmus. The impulse cycles thus aroused augment those already being expressed in the spontaneous nystagmus, hence the nystagmus present is increased in intensity (see Plate XXVII). If the warm caloric test is applied to the affected ear the nervous impulses will traverse the paths shown by the two blue lines marked 120° in Plate XXVII, and will cause a slow conjugate movement of both eyes to the left. This is immediately followed by a corrective movement of both eyes to the right, the quick component of the induced nystagmus. The nystagmatic movements thus induced are antagonistic, *i. e.*, in the opposite direction, to the spontaneous nystagmus, hence it diminishes the intensity of the spontaneous nystagmus. Indeed, it may entirely suppress it for twenty to thirty seconds, or during the life of the induced antagonistic impulses. When vestibular reaction is shown by the caloric tests as described above, and the deafness is complete, there is some doubt as to the nature of the pathologic process present. It is probably diffuse serous labyrinthitis. If, during the subsequent course of the disease the hearing returns, the disease is surely of the serous variety, whereas, if the hearing is totally and permanently abolished the process was probably diffuse suppurative labyrinthitis. Whatever the nature of the pathologic process the prognosis is more favorable than if the caloric reactions were not induced. The indications for treatment would also be materially modified; that is, an operation would be contra-indicated. If, however, caloric reactions were not demonstrable, the prognosis would be more grave, and an immediate operation should, at least, be seriously considered. If for any reason a mastoid operation is required the inner wall of the tympanum and aditus ad antrum should be closely inspected for fistula, and if found the labyrinth should also be operated. If fistula is absent, the labyrinth may be left without operation, though according to some authorities

[1] This result may also be attributed to inhibition of the right crista, leaving balance of potentiality on the left side.

FIG. 497



Barany's pointing test.

the labyrinth should in such cases be operated if mastoid operation is performed. Only further experience and observation can settle this question.

Finally, it should be reiterated, that when caloric reaction is present and the deafness is complete the disease is in all probability

diffuse serous labyrinthitis rather than diffuse suppurative manifest labyrinthitis.

Temperature.—Elevation of temperature gives no information as to the progress of disease within the labyrinth. As previously stated, the labyrinth spaces are too small, and the osseous walls too dense to allow the absorption of enough septic material to affect the temperature. Whatever elevation of temperature may be present is due to the inflammation extraneous to the labyrinth. It is due to either otitis media, mastoiditis, sinus thrombosis, meningitis, abscess, etc., and not to the labyrinthitis. If this is true the elevation of temperature affords no information as to either the nature or progress of the labyrinthitis. It may, however, afford information as to the indications for surgical interference; that is, it may with the other symptoms present indicate either meningeal irritation or actual meningitis, and thus aid in determining the advisability of a labyrinth operation.

The Position of the Patient while in Bed.—During the initial period of the vertiginous attack, previously referred to as the "signs of destruction disharmony," the patient instinctively lies with his head so placed upon the pillow that when he opens his eyes he looks toward the slow component of the spontaneous nystagmus. With this position of the eyes the nystagmus and other vertiginous phenomena are either diminished in intensity, or altogether suppressed. The patient is thereby made more comfortable. If, however, he should lie so that when his eyes are open he is compelled to look toward the quick component of the nystagmus, the nystagmus and other accompanying vertiginous symptoms would be aggravated and the patient rendered most uncomfortable. He, therefore, voluntarily and insistently lies upon the side of the face toward which the quick component of the nystagmus is directed.

The Pointing Reaction.—The pointing reaction of Barany is based upon the phenomenon that, when an individual is affected with either pronounced induced or spontaneous vestibular nystagmus, he will, when the eyes are closed, point toward the slow component of the nystagmus with either hand. When, therefore, he points to the slow component it is normal for vestibular disease, *i. e.*, it indicates vestibular disease. If, however, he points in the opposite direction, or varies the direction of pointing in successive trials at the same sitting, it is abnormal for vestibular disease, and is suggestive of cerebellar disease. Furthermore, the pointing reaction in cerebellar disease only applies to the hand or arm of the diseased side (see Pointing Reaction Test and Fig. 497).

Headache.—Headache is usually present in acute diffuse manifest suppurative labyrinthitis, but it is not as severe or as localized as in cerebellar disease.

Tinnitus Aurium.—Tinnitus aurium of varying degrees of intensity is present in more than 30 per cent. of the cases. Occasionally it is very severe, even during the latent stage (see Diffuse Latent Labyrinthitis).

The course of acute diffuse manifest suppurative labyrinthitis is determined by the time required to establish extralabyrinthine compensa-

tion. This varies from one to four weeks. The establishment of extralabyrinthine compensation is signalized by the subsidence of the spontaneous nystagmus and the reaction movements of the body and extremities. The reaction movements consist of nausea and vomiting, and ataxia. When the spontaneous nystagmus and reaction movements cease to be manifest under ordinary conditions of life the disease is called *diffuse latent suppurative labyrinthitis*. There are, then, no manifest vestibular symptoms. They can only be aroused by special unaccustomed body movements, as in the turning test, sudden stooping, jarring movements, etc.

The *onset* of acute diffuse manifest suppurative labyrinthitis is characterized by a sudden and severe attack or seizure of spontaneous rotatory nystagmus to the unaffected side, nausea, vomiting, giddiness, the sense of surrounding objects whirling around the patient in the plane of the nystagmus, vestibular ataxia; and if he attempts to stand, he falls toward the slow component of the nystagmus. If the right labyrinth is destroyed he falls to the right when facing straight ahead. If he turns his face over his left shoulder he falls forward. If he turns his face over his right shoulder he falls backward. These reactions are known as the "signs of destruction disharmony," and they gradually abate in intensity and disappear altogether in from a few days to a few weeks. They are severe enough for a few hours or days to confine the patient to his bed. At the onset he instinctively lies upon the sound ear as this compels him, when the eyes are open, to look toward the slow component of the nystagmus, which diminishes the intensity of the nystagmus and reaction movements, and renders his condition much more tolerable.

The hearing in the affected ear is immediately, totally, and forever destroyed, as is also the physiological irritability of the vestibular apparatus. Total deafness in the affected ear can be shown by the use of Barany's noise apparatus (see Functional Tests of the Ear).

The caloric tests, if used in the affected ear, would show no vestibular reaction, as the vestibular apparatus is destroyed.

The fistula test would also yield a negative result for the same reason.

If, however, these tests are applied to the unaffected ear during the height of the spontaneous nystagmus they would yield positive reactions. The slow component of the spontaneous nystagmus (vestibular element) is rotatory and to the affected (right) side, and the quick component (cortical element) is rotatory and to the opposite, or unaffected left side. The application of the cold caloric test to the normal ear would induce a slow movement of the eyes to the unaffected side and a quick corrective movement to the affected side, and as each of these induced reactions is in the opposite direction to those of the spontaneous nystagmus, the intensity of the spontaneous nystagmus would be temporarily diminished or abolished. If the warm caloric test is applied the induced reaction would be in consonance with the spontaneous nystagmic movements, and the spontaneous nystagmus

would therefore be increased in intensity. The same tests may be applied to the affected ear, and if vestibular reaction is aroused the case is either one of serous labyrinthitis or cerebellar disease.

These tests need not be applied except when in doubt as to the diagnosis. It sometimes happens in cerebellar abscess or tumor that the nystagmus is to the opposite side as in acute destruction of the labyrinth, and the diagnosis becomes an important matter. Under these circumstances the tests may be made to clear the diagnosis.

If, however, acute cerebellar abscess is located on the right side, the side of the suppurative otitis media, and causes spontaneous nystagmus to the same side, and reaction movements, the warm caloric test, applied to the affected ear would induce nystagmus which would increase the spontaneous nystagmus already present, as shown in Plate XXVII. The nystagmus would be diminished in intensity if the cerebellar nystagmus were directed, contrary to rule, to the unaffected side. The same reactions would be obtained if the disease were diffuse serous labyrinthitis, with partial suppression of vestibular function on the affected (right) side, though the difference in intensity of the spontaneous nystagmus would be less pronounced, as the cristæ in this affection are inhibited, not normally sensitive, as they would be in cerebellar abscess. Another factor to be taken into consideration in the differential diagnosis between acute diffuse manifest serous and suppurative labyrinthitis and cerebellar abscess or tumor is the increasing or diminished intensity of the spontaneous nystagmus. In labyrinthitis it rapidly diminishes in intensity and disappears within a few days or weeks; whereas, in cerebellar disease it increases in intensity or continues indefinitely without recession. These difficulties of differentiation can but rarely arise, as total deafness is exceptional in abscess of the cerebellum, unless either diffuse serous or suppurative labyrinthitis is also present. The nystagmus of cerebellar origin may not be manifest until opaque glasses are applied.

The Elevation of Temperature.—The elevation of temperature is due to the inflammatory process outside of the labyrinth, and not to the process within the labyrinth, as the labyrinth spaces are too small and the osseous walls too dense to permit enough septic absorption to materially affect the temperature. Continued elevation of temperature, therefore, is not *per se* an indication of the progression of the infection through the labyrinth to the meninges and brain. It may, however, show a continued virulency of inflammation extraneous to the labyrinth, either extra- or intracranially. The prognosis is, however, thereby rendered correspondingly more grave. If the elevation of temperature continues, and is associated with severe headache, meningitis may be present; and if these symptoms are attended by a reversal in the direction of the spontaneous nystagmus, which is due to central stimulation rather than peripheral (labyrinthine) stimulation, meningitis is quite probable. Operation upon the labyrinth now becomes imperative and should be done at once.

Prognosis.—The prognosis of acute diffuse manifest suppurative labyrinthitis is quite grave, as meningitis or cerebellar abscess may

complicate it, or occur as a later phenomenon during the period of latency (diffuse latent suppurative labyrinthitis). Labyrinthitis is not of itself a serious disease. Death results from the complications and sequelæ.

Treatment.—The treatment consists in (*a*) preventing the extension of the infection from the labyrinth to the meninges and brain, and (*b*) the drainage of the infected suppurating area of the meninges and brain should they be present.

The Prevention of the Extension of the Infection to the Meninges and Brain.—If any one would satisfactorily determine the safest and surest way to protect the meninges and brain from infection during the course of acute diffuse manifest suppurative labyrinthitis he would render a service of the highest order to the human race. Various procedures have been advocated and some of them tried with more or less success, but none of them have been used in a sufficiently large number of cases to warrant their unqualified endorsement. Indeed, no one has presumed that any method of treatment can ever be universally successful. The hope is that some method of treatment will be found which will reduce the present mortality rate, and, perhaps, this has already been measurably accomplished. Of the methods of treatment which have been more or less successfully used for the prevention of the extension to the meninges and brain, the following are worthy of mention and discussion.

1. *Urotropin.*—Urotropin has for several years been advocated as of therapeutic value at the inception of meningitis, and more particularly as a prophylactic measure. That it is of great value, is, I think, hardly proved, though it has appeared to act favorably in some instances.

2. *Absolute Quiet by Fixation.*—Sheibe has advocated the fixation of the head by means of plaster bandages applied to the head and shoulders, though I understand some of his reported cures by this method of treatment afterward died and went to postmortem. Notwithstanding this, the idea is good and contains food for reflection. During the height of the attack the patient should be placed in bed and absolute physical quiet enforced, as even slight movements of the body and head might favor the spread of the infection to the subarachnoid spaces of the brain. For this reason no tests should be made that are not absolutely required to make the diagnosis.

Surgical.—The question as to whether surgery of the labyrinth prevents the extension of the infection and inflammation to the meninges and brain is still an open one. Some authors claim that if the proper surgical measures are adopted immediately after the hearing is totally destroyed, meningitis and brain abscess will rarely complicate or follow acute diffuse manifest suppurative labyrinthitis. In other words, they claim a much lower mortality rate may be obtained by prompt operation than by waiting for intracranial symptoms before operation.

Other authors of equal standing claim that to operate during the acute manifestations, or as soon as deafness is established, adds to

the liability of intracranial extension, and that the safer procedure is to watch the symptoms, and, when signs of meningeal irritation, as continued elevation of temperature, severe headache, etc., supervene, to operate the labyrinth. Furthermore, if the patient passes through the acute disease and emerges safely into the latent type, operation may then be performed with greater safety, etc.

If there is a question as to the disease being cerebellar abscess, and this cannot be determined, the labyrinth operation may be performed, and if after this the nystagmus and signs of destruction disharmony continue indefinitely the disease is undoubtedly cerebellar.

The foregoing is a brief summary of the two great schools of thought upon this subject. It is very difficult to determine which is the nearer correct in its position. Reasoning by analogy I should conclude that the first procedure will better conserve life, provided the proper type of labyrinth operation is performed, and the technique of the operation is skilfully executed. In the absence of a thorough knowledge of the various operative procedures and of a technical operator, the second or "waiting and watching" program would be the safer procedure. (See Indications and Surgery of the Labyrinth in the following chapter.)

DIFFUSE LATENT SUPPURATIVE LABYRINTHITIS

Etiology.—The etiology of the disease is identical with that of acute diffuse manifest suppurative labyrinthitis, as it is but the sequela or latent stage of that disease (see etiology of acute diffuse manifest suppurative labyrinthitis).

Symptoms.—The symptoms of this disease are due to the total destruction of both the vestibular and cochlear apparatuses in the affected ear and to the partial or complete compensation of vestibular function (Plate XXV). If compensation of extralabyrinthine vestibular function is complete the disease is, insofar as subjective phenomena are concerned, almost symptomless; that is, the patient does not suffer from either spontaneous nystagmus or the "signs of destruction disharmony." His only subjective symptoms are total deafness in the affected ear, and the phenomena incidental to the purulent otorrhea. If extralabyrinthine compensation is not complete sudden movements of the head and looking toward the opposite side will produce nystagmus.

The objective symptoms are those that may be induced by the various functional tests of the ear, *i. e.*, successive turnings to the right and then to the left would give vestibular reactions in the ratio of 2 to 1 (Plate XXV), and the caloric reactions would be negative in the affected labyrinth.

Deafness.—The hearing in the affected ear is completely and forever lost. The completeness of the deafness in the affected ear may be shown by placing Barany's noise apparatus in the unaffected ear, and, while it is in action, addressing the patient's affected ear with increasing loudness up to the shouting voice. If the instrument is

properly applied it will shut out all hearing from the sound ear; hence, if the loud voice of the examiner is not heard, the affected ear is shown to be totally deaf; whereas, if the patient hears the voice, the deafness is not complete (see Barany's Noise Apparatus Test).

Purulent Discharge.—The purulent discharge differs in no way from that of simple chronic otorrhea and mastoiditis. If a fistula of the promontorium is present the secretion may be observed discharging through it. Granulations may also be seen around the fistulous opening.

Fistula Symptom.—Fistula symptom, *i. e.*, reaction to the compression test, is absent (negative) even when a fistula is present. This is due to the total destruction of the cristæ of the affected vestibular apparatus. Should fistula symptom be present (positive) doubt is aroused as to the nature of the primary acute process preceding the latent stage, or disease, though it is quite conceivable that a diffuse suppurative process may not destroy all vestibular function, as this organ is more resistant than the cochlea. Positive fistula symptom should, however, lead the surgeon to regard the disease as probably being serous labyrinthitis.

Caloric Reaction.—Caloric reaction is absent or negative, as the cristæ of the vestibular apparatus are wholly destroyed; the possible exception being those cases in which the preceding acute diffuse suppurative process did not wholly destroy the vestibular apparatus. Total deafness in the involved ear would not, in the presence of caloric reaction, adequately support the assumption that these cases were primarily suppurative rather than serous in character.

Turning Reaction.—The turning reaction varies in diffuse latent suppurative labyrinthitis according to the period of time which has elapsed since the acute diffuse manifest suppurative stage. That is, it varies with the degree of compensation that has taken place. It is customary to say that compensation has occurred when, as a matter of fact, there is only partial compensation, that is, spontaneous nystagmus and the "signs of destruction disharmony" are no longer present under ordinary conditions of body movements, though they may be induced by slight extraneous stimulations, as sudden stooping movements, sudden jarring movements, etc. The fistula and caloric tests do not cause stimulation of the affected labyrinth in this disease, as they act upon a dead labyrinth. The turnings act upon the unaffected labyrinth, no matter what the direction of turnings may be, and measure with some degree of accuracy the amount of destruction and compensation present (Plate XXV).

We will assume the patient to be affected with diffuse latent suppurative labyrinthitis in the right ear, and that the turning test was made three months after the period of latency was established. He was placed in a revolving chair and turned ten times, with the head erect, to the right or toward the affected ear, and suddenly stopped. Horizontal induced after-nystagmus to the left or opposite side is observed over the opaque glasses. It continues for about twelve

seconds. After allowing the patient to rest for five or ten minutes he was turned ten times, with the head erect, to the left, and the eyes observed over the rims of the opaque glasses worn by the patient. Induced after-nystagmus of six seconds' duration was induced. While extralabyrinthine compensation has occurred, as shown by the absence of spontaneous nystagmus and "signs of destruction disharmony," actual vestibular compensation has not occurred. As time goes on actual vestibular compensation seems to take place, and as it occurs the extralabyrinthine compensation seems to be replaced by it (Plate XXVI). Extralabyrinthine compensation is relative rather than absolute. Vestibular compensation is almost absolute. Extralabyrinthine compensation is acquired in from one to four weeks, whereas, vestibular compensation is established only after the lapse of several years.

We will now assume that four years after the first test the same patient was subjected to the same tests, *i. e.*, turning to the right ten times, and turning to the left ten times, and the after-nystagmus was noted after each test as before. In this instance, after the turnings to the right, the after-nystagmus endured eleven seconds, and after the turning to the left it endured eight seconds, thus showing an approach toward equality of tonus in the two labyrinths.

We will now assume that eight years after the first, and four years after the second test, the patient was subjected to the same turning experiments, and that in this instance the after-nystagmus induced by turnings to the right endured twelve seconds, and after turnings to the left they endured eleven seconds. The tonus in the two opposing halves of the crista ampullaris of the horizontal canal of the unaffected ear is almost equal; indeed, it is as nearly equal as is found in persons with normal labyrinths, the difference being the shorter duration of the after-nystagmus after turning to either the right or the left (Plate XXVI). The extralabyrinthine compensation seems to have been gradually replaced by the vestibular or labyrinthine compensation. Whatever the explanation may be, it has been shown by Ruttin that, in cases of very long standing, almost complete vestibular compensation occurred, and that the extralabyrinthine compensation recedes as the vestibular compensation progresses.

Differential Diagnosis.—The following conditions may simulate diffuse latent suppurative labyrinthitis and should be carefully differentiated from it:

1. *Hysteria* sometimes simulates labyrinthitis though only in a superficial way. The nystagmus is not present in diffuse latent suppurative labyrinthitis though it may be in hysteria. If present in hysteria it is not of the vestibular type, *i. e.*, it does not have a quick and slow component, etc. The membrana tympani is either normal or it may be diseased. The hearing is either normal or, if ear disease is present, it may be impaired but not entirely lost, as in latent labyrinthitis. If disturbance of equilibrium is present the body may fall in any direction; there is neither rule nor certainty in what direction the patient will fall; whereas, in labyrinth disease he always falls in

the direction of the slow component of the nystagmus. If, with the face straight ahead, and the slow component to the right, the patient will fall to the right; with the face directed over the left shoulder the patient will fall forward; with the face over the right shoulder he will fall backward. In a hysterical case the direction of the face would exert no definite influence upon the direction of falling.

2. *Disease of the Eighth Nerve.*—Disease of the eighth nerve may develop independently of middle-ear disease, hence the vertiginous attacks due to a disturbance of the vestibular portion of the eighth nerve with an apparently normal middle ear should suggest disease of the eighth nerve. An important exception to this would be metastatic involvement of the labyrinth, especially for mumps. In disease of the eighth nerve (retrolabyrinthine disease involving the eighth nerve) only one division of the nerve is usually involved. That is, there is involvement of the vestibular branch alone, which is attended with vertigo, nausea and vomiting, loss of equilibrium, and nystagmus, while the hearing is unaffected; or the auditory division is affected and there is loss of hearing, but no vestibular symptoms are present. In true diffuse labyrinthitis both divisions of the eighth nerve are always affected, and deafness and vestibular symptoms attend the destructive process.

In these cases Ruttin has employed bilateral simultaneous caloric and galvanic tests, the caloric test giving the comparative susceptibility of the two static labyrinths to stimulation, while the galvanic test gives the comparative irritability of the two vestibular nerves and centrums.

3. *Polyneuritis (Syphilis) of the Eighth and Facial Nerves.*—This condition is characterized by an attack of vertigo, nausea, nystagmus to the affected side, loss of equilibrium, deafness, and facial paralysis. The ear may appear to be normal. This condition simulates sequestrum of the labyrinth in which the facial may be involved, though in sequestrum the vertiginous attacks are absent, but upon inquiry the fact of their existence some time previously may be elicited. The Wassermann test in these cases is usually positive.

4. *Cerebellar Disease.*—It sometimes happens that after the surgical removal of one labyrinth the nystagmus which at first was directed to the sound side became directed to the operated side. This is a sign of intracranial involvement, and the changed direction of the nystagmus is due to direct stimulation of Deiters' nucleus of the diseased side. Previous to this phenomenon the labyrinth on the affected side was inhibited or destroyed and nervous impulses did not emanate from that side. Upon the inception of the cerebellar disease the stimulous (toxic, congestion) is applied directly to Deiters' nucleus of the affected side and produces spontaneous nystagmus to the diseased side, or in a reverse direction to that which previously existed. The nystagmus due to the labyrinthitis was caused by the total suppression of nervous impulses from the diseased labyrinth, whereas the nystagmus due to the cerebellar disease was caused by the excessive nervous impulses from the diseased side.

CHAPTER LII

SURGERY OF THE LABYRINTH

Indications for the Labyrinth Operations.—1. There is at present no well-defined consensus of opinion as to the exact indications for the surgical drainage and exenteration of the labyrinth. Some hold that the total loss of hearing in *acute diffuse manifest suppurative labyrinthitis* is a positive indication for the labyrinth operation. Others contend that in such cases one should wait for indications of meningeal irritation, as severe headache, continued elevation of temperature, restlessness, etc., before doing a labyrinth operation, or, if these do not develop, that you should wait until the latent stage is established, as it is then safer to operate. In support of this view they cite cases that have recovered without the occurrence of meningitis, or cerebellar abscess. The opponents of these indications for the labyrinth operation in reply cite many cases that, while waiting, developed diffuse suppurative meningitis or cerebellar abscess and died. They also state that if meningitis develops about 98 per cent. of them die in spite of a labyrinth operation, and that of all cases, operated as soon as hearing is lost and before meningitis develops, a large per cent. of them recover. They claim the labyrinth operation in the hand of an otologist skilled in diagnosis and surgical technique does not of itself constitute a serious procedure. The seriousness attending such cases often lies in the failure to make a diagnosis sufficiently early or before meningitis sets in. A further danger in waiting for signs of impending meningitis lies in the fact that there are no well-recognized symptoms of "impending meningitis." When such so-called symptoms are present they not infrequently indicate the "actual presence" of diffuse suppurative meningitis. In these cases, therefore, it is often too late to do a labyrinth operation with any reasonable hope of saving the life of the patient. Another fact of great importance is that the further extension of the infection toward the meninges is not signalized by an increase in the vestibular symptoms as increased spontaneous nystagmus, vertigo, nausea, ataxia, etc. The vestibular apparatus is destroyed and incapable of being further destroyed, hence cannot again give rise to vestibular symptoms. In view of the foregoing considerations I am inclined to believe that the safety of the patient is better conserved in acute diffuse manifest suppurative labyrinthitis, with total destruction of the auditory and static labyrinths, by a labyrinth operation. If, however, there is a remnant of either the auditory or static functions remaining, it is safe to wait, as the further sudden destruction of these functions will be signalized by an increase in the

intensity of the vestibular symptoms, thus constituting a warning of impending danger in ample time to perform a labyrinthine operation.

2. *In diffuse latent suppurative labyrinthitis, i. e.,* the stage of quiescence following acute diffuse manifest suppurative labyrinthitis in which total destruction of hearing and static function occurred during the acute stage, a labyrinth operation may be more safely done than it can in the acute manifest form of the disease, that is, the mortality rate due to the operation *per se,* will be less than it is with operation in the acute stage of the disease. Indeed, the death rate should be almost *nil* after operation on this type of labyrinthitis. The object of the operation is, of course, to prevent the subsequent occurrence of meningitis and cerebellar abscess. Some authors advocate waiting until this stage to operate, as the operation will be much safer then. They apparently forget that, while waiting Macawber-like for something to "turn up," meningitis or brain abscess often develops, and the patient dies. It is to prevent these possible deaths that the labyrinth operation is recommended.

The patient, however, having been so fortunate as to pass through acute destructive suppurative labyrinthitis without serious consequences, is, nevertheless, still in grave danger of the occurrence of meningitis or other serious intracranial sequela.

In view of the almost total absence of danger from the labyrinth operation in latent diffuse labyrinthitis, and the danger that may occur if the operation is not performed, a labyrinth operation should be advised as an early remedial measure.

3. *In acute diffuse serous labyrinthitis* there is usually no likelihood that the cranial content will become involved, hence a labyrinth operation is contraindicated. Furthermore, the hearing is usually restored to nearly normal or as near normal as before the disease. This, of course, constitutes another contraindication to operation.

4. *In circumscribed labyrinthitis,* either with or without fistula symptom, the labyrinth operation is positively contraindicated, as the hearing is but slightly impaired, and static function is only disturbed at intervals, and warning will be given in the form of sudden deafness, ataxia, etc., of impending danger to the brain. Should sudden deafness and vestibular symptoms develop, a critical examination should be made at once, and if total destruction has occurred an immediate operation should be seriously considered. The disease is now converted into either an acute diffuse serous labyrinthitis, or an acute diffuse manifest suppurative labyrinthitis. If it is serous, operation is contraindicated. If acute diffuse manifest suppurative labyrinthitis, operation may be advised. If not performed the patient should be kept as nearly "fixed" in bed as possible, as even slight movements of the head might cause further extension of the pathologic process. Upon the appearance of headache or other signs of meningeal irritation advise a labyrinth operation, though it is probably too late to head off meningitis. It is just such "waiting," however, that may throw discredit upon the labyrinth operation.

The Merits of the Various Labyrinth Operations.—In arriving at conclusions as to the comparative merits of the various labyrinth operations we must constantly bear in mind the following facts:

1. The general purposes for which the various operations are performed.
2. The scope of each operation.
3. Whether the labyrinthitis is simple or complicated by some intracranial pathological process, or whether a sequestrum is present.
4. If in doing a mastoid operation a suppurative labyrinthitis is found (pus oozing from a perforation in the promontorium), the operation should be modified to meet the unexpected complication.

The general purposes for which the operations are performed are (*a*) drainage of the labyrinth spaces, and (*b*) the drainage of the meningeal or subarachnoid spaces.

(*a*) If only the drainage of the labyrinth spaces is necessary, the Hinsberg operation is admirable for the purpose. The Hinsberg operation in the University Hospital at Breslau has been attended by a death rate of only 4 per cent. This is certainly a good showing and speaks favorably for the operation, especially in view of the fact that it is the simplest operative procedure in vogue. In this operation the external limb and ampulla of the horizontal canal, the vestibule, and lower whorl of the cochlea are opened. In the Neumann and Richards operations all of the canals are either exenterated or laid open. As the canals communicate only with the vestibule, and the vestibule is freely opened in all types of labyrinth operation, it appears rational to expect no serious complication even though the canals are not exenterated or freely opened. We cannot, however, evade the issue that when meningitis is present none of the operations, excepting Neumann's, provides for the drainage of the meninges at the atrium of infection. It seems to me, therefore, that in choosing an operation this factor must be given due consideration. Neither the Hinsberg, Bourguet nor Richards operation drains the meningeal spaces. In all the vestibule and cochlea are opened, the vestibule very adequately, and the cochlea sufficiently to afford fair drainage. In Richards' operation the cochlea may be opened more freely than in either of the others, including Neumann's. Extensive exposure of the cochlea, however, is attended by considerable danger.

(*b*) If it is also necessary to drain the meninges, neither of these operations is suited for the purpose. Some operation must be chosen that will do all that these operations accomplish, and in addition must drain the meninges at the atrium of infection. As this is nearly always situated at the intracranial orifice of the internal auditory meatus which conveys the eighth or auditory-vestibular nerve to the labyrinth, or on the posterior wall of the pyramid, it is necessary to perform an operation that drains the areas of meninges at the internal auditory meatus and posterior wall of the pyramid. As the meninges envelop the eighth nerve throughout the entire length of the internal auditory meatus to the area cribrosa, it is doubly necessary to open the internal

auditory meatus its whole length, should the meningeal infection traverse this route. The only operation fulfilling all these conditions is Neumann's. When, therefore, it is either suspected or known that meningitis has developed, Neumann's operation is the only one available for this purpose.

Richards' operation is a fine piece of dissection of the vestibule, semicircular canals, and cochlea, and is a more thorough operation than the Hinsberg or the Bourguet operation. It is more difficult to perform, and is attended by the greater danger of injuring the facial nerve and modiolus of the cochlea, and is on this account a less desirable operation than the Hinsberg operation, which is, perhaps, the simplest labyrinth operation. The Bourguet operation accomplishes the same ends as the Hinsberg, though the technique is not so simple or well conceived.

(*c*) If a sequestrum of the cochlea or any other portion of the labyrinth is found, it should, of course, be removed. Some operators are content to extend the labyrinth operation under such circumstances only far enough to liberate the sequestrum, trusting to the protection of the granulation wall which has been formed.

(*d*) If the surgeon should chance to perform a mastoid operation upon a case affected by suppurative labyrinthitis without having previously diagnosed it as such, and should find pus exuding from a fistula of the labyrinth, he should extend the operation so as to include the labyrinth, perhaps preferably by the Hinsberg method. If, however, he suspects or knows that meningitis is also present, he should do the Neumann operation, or no operation.

In view of these facts it is apparent that the otologist should not only be conversant with the indications for an operation, but he should also be familiar with the indications for a particular type of operation. It should be said, however, that in spite of all that is known, it will be impossible to always correctly judge as to the absolute wisdom of doing a labyrinth operation, and, if an operation is really necessary, it is not always possible to correctly determine the scope of the operation required to give the best results.

It will doubtless require the accumulated experience and observations of many years to decide either of these questions. In the meantime each otologist should act in accordance with his best judgment and perfect a technique of diagnosis and operation, reporting his results for the benefit of other otologists. Only in this way will we be enabled to arrive at a satisfactory solution of this vexing problem. He should constantly bear in mind that he is dealing with a disease process which threatens life at its most vital centre, and should, therefore, carefully weigh all the phenomena and determine as nearly as he can the probable outcome in each case, with and without surgical intervention. He should also estimate the most favorable time in the course of the disease to operate. Furthermore, he should remember in formulating his indications for treatment of acute diffuse manifest suppurative labyrinthitis, that, though the death rate following opera-

tion in the subacute or chronic latent stage is lower than in the acute manifest stage, it does not necessarily follow that he should always wait until the latent stage to operate. This would be false logic, as while waiting for latency to develop the patient may develop meningitis and die; whereas it is possible, and even quite probable, that an operation in the acute manifest stage would, in properly selected cases, prevent the development of meningitis, and save the life of the patient.

I believe, therefore, that while an operation on the labyrinth should not always be done in acute diffuse manifest suppurative labyrinthitis, it should, however, always be most carefully considered in formulating the indications for treatment. I have endeavored in this section on labyrinthitis, to view the various phenomena and problems from a multitude of view-points so as to meet the many perplexities which confront the student of labyrinth disease.

THE JANSEN-NEUMANN LABYRINTH OPERATION

The Neumann operation is the same as the Jansen operation except that it goes one step farther and uncovers the eighth nerve (vestibulo-auditory nerve). That is, the internal auditory meatus is opened. The Neumann operation should be performed when meningitis is suspected or known to be present and has its atrium of infection through the internal auditory meatus, or through the posterior wall of the pyramid. The Hinsberg operation should be performed when the complicating meningitis is known not to be present. The Jansen and Neumann operations are essentially similar, with the aforesaid exception; hence they will be described together.

Preliminary Measures.—The radical mastoid operation should first be performed (if it has not been done previously) as a preliminary step in all labyrinth operations, and the labyrinth operation should immediately follow. The projecting margins of the mastoid wound should be removed to give ample space for the instruments in the labyrinth. The tympanic mouth of the Eustachian tube should be curetted to free it of bleeding granulations and congested mucous membrane. Adrenalin should then be applied to stop the bleeding, as it is necessary to keep the operative field clear in the subsequent steps of the operation.

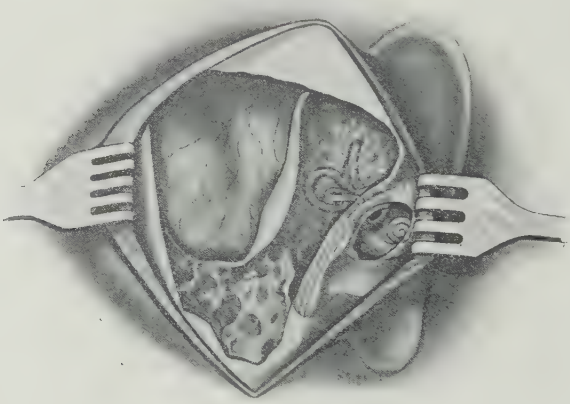
First Step.—The outer and anterior bony wall of the lateral sinus should be removed with either a broad gouge (Alexander's No. 14) or with special rongeur forceps The thin bone anterior and superior to the lateral sinus, known as Trautmann's triangle, is then removed. This triangular area is bounded above by the roof of the mastoid cavity, posterior-inferiorly by the sigmoid portion of the lateral sinus, and anteriorly by the facial ridge, or rather a line extending from the anterosuperior angle of the mastoid wound to the inferior margin of the lower end of the lateral sinus. In removing the thin bone from

the lateral sinus and Trautmann's triangle, the dura should first be gently elevated, and the bone then removed with a broad, flat gouge and mallet, or a special rongeur forceps. Great care should be taken to avoid tearing the dura, as such an injury might lead to meningitis or brain abscess. If the dura is torn, the tear should be enlarged at the close of the operation and tincture of iodine applied. A small rent does not afford good drainage, whereas a large one does. The dura should be thus respected throughout the subsequent steps of the operation.

In Fig. 498 I have shown a transparency of the labyrinth, which will act as a guide in exposing the canals and opening the vestibule in the subsequent steps of the operation.

Second Step.—The next step of the operation consists in elevating the dura from the posterior wall of the pyramid preliminary to the removal of the three semicircular canals which are embedded in the

FIG. 498

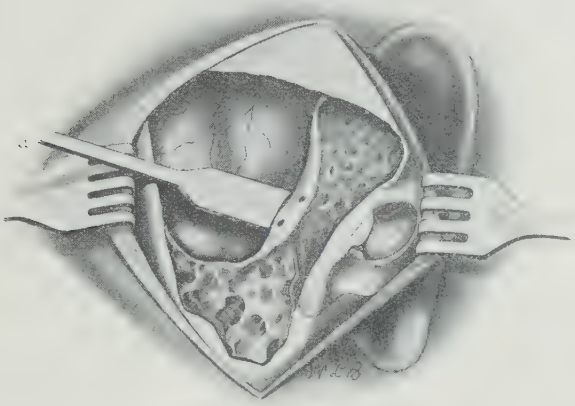


Transparency showing the labyrinth which, when compared with the following steps of the Neumann operation, explains the rationale of the operation.

pyramid or petrous portion of the temporal bone. The dura should be elevated from the posterior wall of the pyramid about one-half to one inch deeper than Trautmann's triangle. It should be elevated with a thin blunt-edged spatula or elevator down to the inner opening of the internal auditory meatus. As the saccus endolymphaticus is located in a slight depression on the posterior wall of the pyramid and is enveloped between reflections of the dura, there is some danger of tearing the dura at this point. To obviate this accident it may be necessary to cut the dural attachment of the sac to the bone with a scalpel. In other words, if the dura does not readily separate from the bone, the area of resistance should be scratched with the point of a scalpel, the point being directed against the bone rather than parallel with the posterior surface of it. Having thus freed the saccus endo-

lymphaticus, continue the elevation of the dura to the inner orifice of the internal auditory meatus.

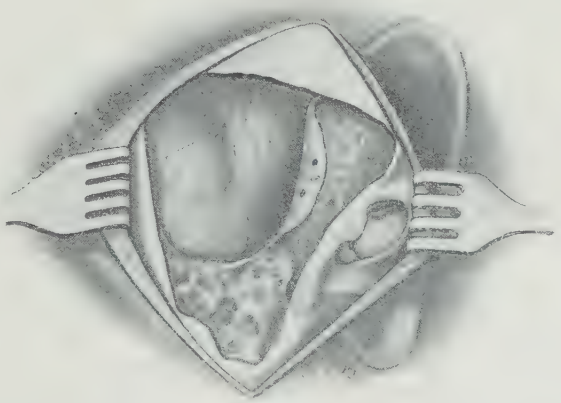
Fig. 499



First step. The lateral sinus and Trautmann's triangle uncovered, and the two openings of the posterior canal opened.

Third Step.—Having completed the elevation of the dura on the posterior wall of the pyramid, proceed to remove the semicircular

Fig. 500



Second step. Another chip of bone has been removed and the arch of the horizontal canal exposed, the three openings forming the angles of a triangle.

canals. This is accomplished with a No. 10 or 14 Alexander gouge, the gouge being directed parallel with the surface of the posterior

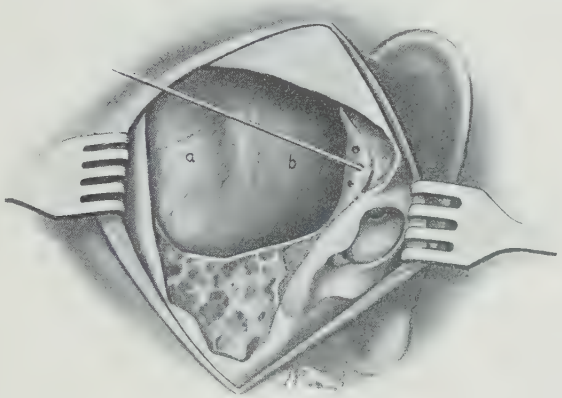
and superior walls of the pyramid. The posterior wall extends principally inward and somewhat forward. The superior margin slopes slightly downward and supports the superior petrosal sinus, hence the gouge should be directed inward, forward, and slightly downward, to avoid injuring this sinus, as otherwise troublesome hemorrhage would result. The operator should, of course, first establish a clear mental image of the relation of the parts by studying the bones *in situ* in a skull (Fig. 498). Having placed the gouge at the proper angle, with its concave surface facing posteriorly, the posterior wall of the pyramid should be removed in shavings or thin chips. The gouge should be very sharp, as it will otherwise pursue an uncertain course through the bone and fracture it. It should be driven with sharp blows of a leather, wooden, or leaden mallet, as the bone is very dense and resistant. The first chip of bone may be about $\frac{1}{2}$ by $\frac{1}{4}$ by $\frac{1}{16}$ of an inch in dimensions. When the chip is freely separated by the gouge it should be removed with stout dressing forceps. The dura having been previously separated from it, there should be little difficulty from this source. The rough edges of the fragment of bone may, however, catch against the roughened margins from which it is being detached and thus render its removal difficult. A little gentle manipulation, as twisting, etc., together with a firm pull will facilitate its removal. The chip should be inspected to see if the posterior canal has been cut through. If cut through the tip of its arch, an oblong groove will be present on its anterior surface. If the cut is more anteriorly the two limbs of the canal will be cut through and appear as two dark red oval or round openings (Fig. 499). These openings may also be seen in the posterior surface of the bony wound as well as on the chip of the bone. The openings serve as landmarks for the removal of the next chip of bone.

The gouge should again be placed in position as before and another thinner chip removed and so on until the common canal of the posterior and superior canals and the external limb of the horizontal canal are sectioned. The opening of the horizontal canal should be situated between the other two openings previously mentioned, and more superficially in relation to the side of the head, the three openings forming the three angles of a triangle (Fig. 500).

A small silver probe should then be introduced into the opening of the horizontal canal and passed into the vestibule. The direction taken by the probe should be noted, as the bone to be removed to open the vestibule envelops this limb of the horizontal canal (Fig. 501). A smaller Alexander gouge (No. 5 or 7) should be selected for the purpose and the bone removed. This stage of the operation brings the gouge in close proximity to the facial ridge and nerve, and great care must be exercised to avoid injuring the nerve. The gouge should undermine the nerve. Prying against the ridge should be studiously avoided. If the ridge is broken by prying or other traumatism the facial nerve will be injured and facial paralysis result. The location of the facial nerve should be carefully determined before opening

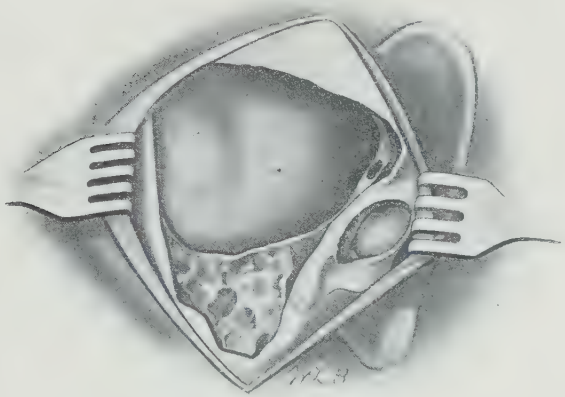
the vestibule and the gouge located posteriorly and superiorly to it to avoid injuring it. One or two small chips of bone along the course of the external limb of the horizontal canal, the course of which is as

FIG. 501



Third step. Still more bone removed and a silver probe passed through the external limb of the horizontal canal into the vestibule. This guides to the vestibule.

FIG. 502



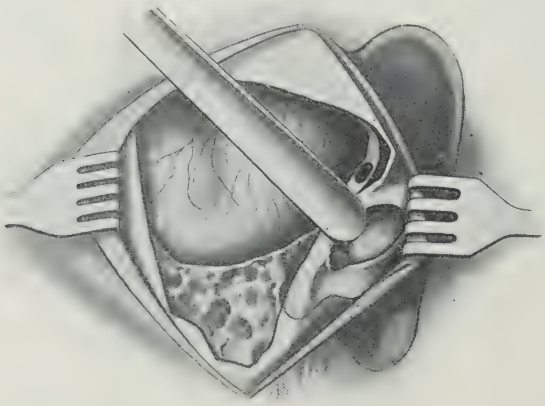
Fourth step. The vestibule exposed. The overhanging ledge of bone is removed.

indicated by probing it, should expose the vestibule on its posterior aspect (Fig. 502). A probe passed through the oval window should pass out through the vestibule into the bony wound.

Owing to their great vascularity, the canals when cut across appear as dark red or purple spots.

This technique completes the static labyrinth portion of the Jansen-Neumann operation. If incipient meningitis is present or suspected, the internal auditory meatus should also be opened by extending the

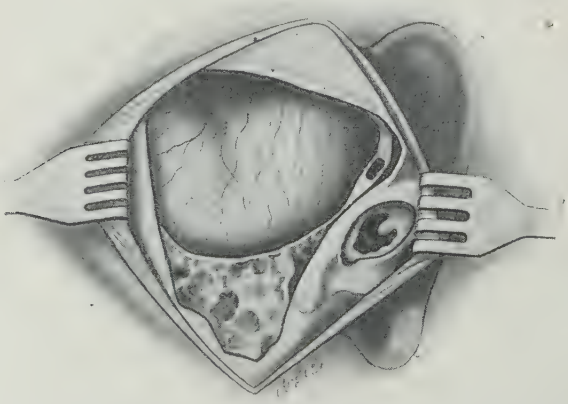
Fig. 503



The gouge in place is ready to remove the promontory.

bone wound deep enough to uncover the nerves in the internal auditory canal and thus establish drainage at the atrium of infection. This step is peculiar to Neumann's method of operating.

Fig. 504

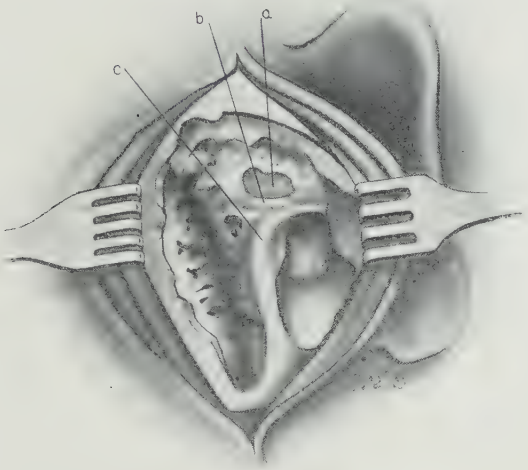


Fifth step. The promontory removed, exposing the lower whorl of the cochlea.

Fourth Step.—To expose the internal auditory canal which contains the eighth cranial nerve (vestibulo-auditory nerve) the Alexander gouges Nos. 7 to 10 are used. The posterior wall of the pyramid is

removed about one-quarter or three-eighths of an inch deeper than is done in the Jansen type of operation. The chips of bone should be about $\frac{1}{16}$ of an inch in thickness, or they may be thin shavings.

FIG. 505



First step. *a*, bone chiselled from above the horizontal canal. *b*, external arm of the horizontal canal. *c*, facial ridge.

FIG. 506

Second step. The arrow points to the open external arm of the horizontal canal. This was opened by chiselling downward, removing the superior bony wall of the canal.

If the dura is now retracted from the deeper portion of the wound the eighth nerve may be seen enveloped in a sheath composed of a reflection of the dura.

This completes the static-labyrinth portion of the operation. That is, the semicircular canals, vestibule, and internal auditory canal containing the auditory vestibular nerve have been exposed, and drainage of these parts and of the meninges at the possible atriums of infection has been established.

This field of operation lies posterior to the descending portion of the facial ridge. The remaining or cochlear portion of the operation lies anterior to the facial ridge and consists in exposing the cochlear spaces of the first or basal whorl of the cochlea.

Fifth Step.—In the fifth step of the operation one-half of the lower whorl of the cochlea is exposed by the removal of the promontory of the inner wall of the middle ear cavity. Preparatory to doing this the anterior margin of the facial ridge should be removed, as it overhangs the tympanic cavity and interferes with the removal of the promontory (compare Figs. 507 and 508). A probe bent at a right angle passed under the projecting margin will show how much is to be removed. If more bone than projects is removed the facial nerve will be injured. Before removal the posterior margin of the middle-ear cavity is ragged and somewhat straight in general direction. After the projecting ledge is removed the outline of the cavity is converted into a symmetrical oval.

The tympanic cavity being thus more fully exposed the tip of a flat No. 10 chisel or No. 10 Alexander gouge should be placed in the depression posterior to the promontory, *i. e.*, between the oval and round windows, and, with a light blow of the mallet the promontory shaved off (Fig. 503). The gouge at first stands almost perpendicular to the plane of the inner wall of the tympanic cavity, though it is inclined as much backward as the facial ridge will permit. As it is tapped its cutting edge is allowed to glide forward and the shank of the instrument to incline more and more backward. In this way the thin shell of bone forming the promontory is removed, and the lower half of the first whorl of the cochlea exposed (Fig. 503).

To attempt to more deeply expose the cochlea would endanger the modiolus, which, if fractured, would allow the cerebrospinal fluid to escape and would provide a dangerous avenue to intradural infection. This completes the Jansen-Neumann labyrinth operation, though the wound remains to be dressed and closed as after the radical mastoid operation.

The Closure of the Wound.—The plastic meatal flaps should be made as in the radical mastoid operation, but instead of using the deep anchor sutures as in the radical operation the meatal flaps should be sutured to the posterior surface of the auricular wound, having previously dissected away all the redundant tissue from this aspect of the auricle (see mastoid operation, Balance's plastic flap). This method of disposing of the meatal flaps should be adopted instead of the deep anchor sutures, as the mastoid wound should not be completely closed as in the radical mastoid operation.

The upper half or two-thirds of the mastoid incision should be sutured as in the radical mastoid operation, while the lower portion

should be left open for drainage and for inspection of the interior of the wound. If after five days no untoward symptoms have developed, the edges of the skin may be freshened under cocaine anesthesia and closed with two or three stitches.

THE HINSBERG OPERATION

This operation consists in opening the horizontal canal and vestibule (*a*) above and behind the knee of the facial ridge, (*b*) opening the vestibule superior to the facial ridge, and (*c*) opening one-half of the lower whorl of the cochlea. The meninges are not exposed as in the Jansen-Neumann operation. Inasmuch as it cannot always be determined whether meningitis is impending or already present, this operation would be inadequate in some cases. In those cases in which there is no meningeal complication it is an almost ideal surgical procedure.

The Hinsberg operation should be preceded by the radical mastoid operation, the curettage of the tympanic orifice of the Eustachian tube, the application of adrenalin, and the chipping off of the overhanging ledge of bone from the anterior margin of the facial ridge.

The stapes is then extracted and the oval window enlarged by breaking down the plate of bone between the oval and round windows.

First Step.—The first step of the operation consists in opening the horizontal canal, which communicates with the superior portion of the vestibule. The ampulla of the horizontal canal is situated directly above the oval window and above and behind the knee of the facial ridge, and may generally be located by a slight ridge or convexity on the inner wall of the antrum (Fig. 505, *b*). By means of a small probe bent at a right angle near its tip the roof of the vestibule may be delineated through the oval window. Having determined the limitations of the roof and the anterior and posterior walls of the vestibule, the opening of the ampulla of the horizontal canal should be begun. This is accomplished by chiselling away the spongy bone just above the superior aspect of the ampullary ridge (Fig. 505, *a*). The chisel should be flat, sharp, and small, as the bone should be removed in thin shavings. The upper part of the prominence of the horizontal canal should be removed until the ampullæ and canal are opened on their superior aspects. The lower part of the canal (*b*) should not be removed, as it is in close proximity to the facial nerve. Kerison advises a specially devised curette for this procedure. Having opened the ampulla and canal, a bent silver probe should be introduced through the enlarged oval window and through the ampullary opening in the roof, as shown in Fig. 507. The area of the vestibule should again be determined with the probe, and the ampullary opening just made should be enlarged, usually in a downward and forward direction, until the vestibule is uncovered from

above (Fig. 507). The vestibule should be examined for necrosed bone, granulations, etc., and if present they should be gently removed with a small curette.

Fig. 507

A bent probe is passed from the oval window upward through the exposed horizontal canal and vestibule.

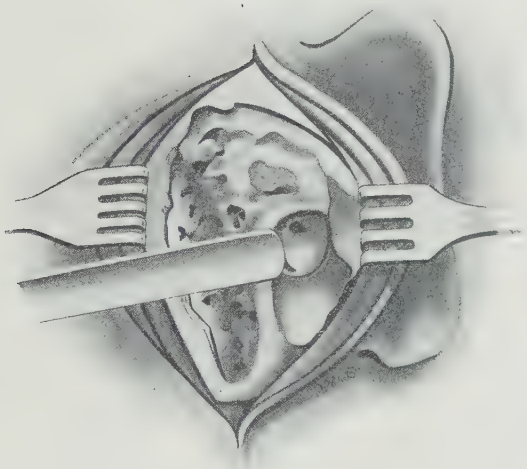
Fig. 508

Third step. *a*, the overhanging lip of bone, marked *x* in Fig. 507, has been removed to make it possible to use the gouge in removing promontorium. *b*, the promontorium.

Second Step.—The projecting ledge of bone (Figs. 507 and 508) should be removed with a No. 10 Alexander gouge to make room for the

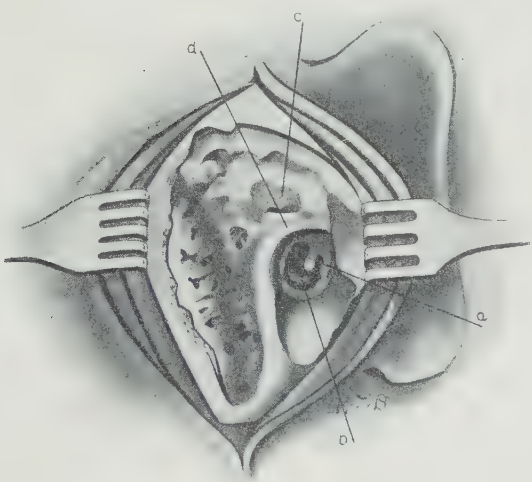
gouge in removing the promontorium in the cochlear portion of the operation.

FIG. 509



The gouge in place preparatory to removing the promontorium.

FIG. 510



Fourth step. *a*, the promontorium removed, exposing the cochlea. *b*, the cochlea exposed. *c*, the horizontal canal exposed. *d*, the facial ridge.

Third Step.—A No. 10 Alexander gouge is placed in position as shown in Fig. 509, engaging the posterior margin of the promontorium below the oval and round windows. The gouge at first stands almost per-

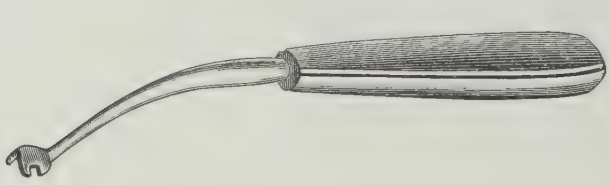
pendicularly, and upon tapping it lightly with a mallet it inclines backward, the tip advancing and shaving the promontorium from its attachment. The result of this step of the operation is the exposure of the lower half of the first whorl of the cochlea as shown in Fig. 510. Indeed, this figure shows the completed Hinsberg operation.

The after-treatment consists in dressing the wound in all respects as you would after a mastoid operation, except the posterior wound is left open for four or five days. or until the occurrence of meningitis is regarded as improbable.

THE BOURGUET OPERATION

Bourguet has devised an instrument (Fig. 511) for the protection of the facial nerve during the procedure for the opening of the canals. The instrument is provided with a semilunar plate 3 by 2 mm. in size. The convex border of the plate has a heel or toe projecting from it somewhat like the toe of a horseshoe. The

FIG. 511



Bourguet's guide and protector.

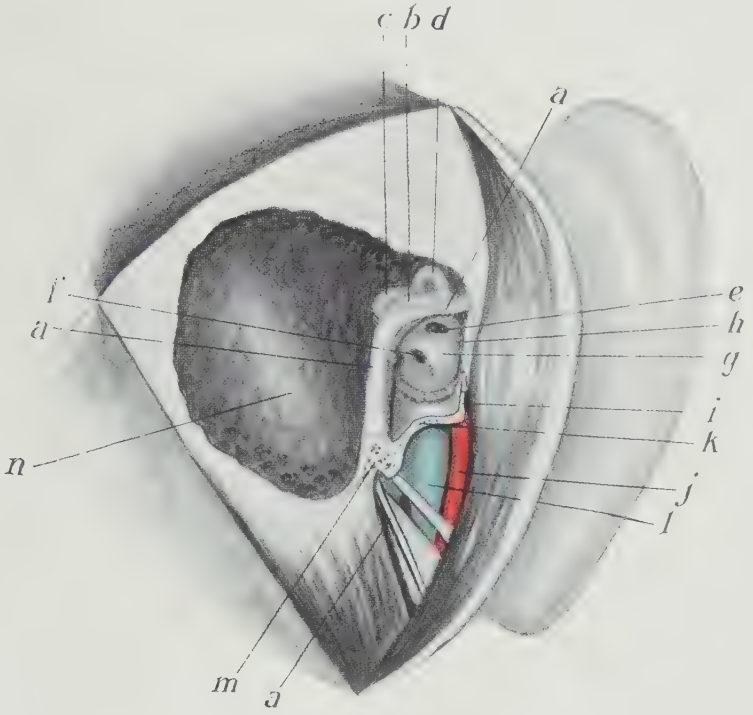
heel or toe is inserted into the oval window, while the convex border of the plate is directed upward. The body of the plate is thus located over the facial canal. When the instrument is thus adjusted the convexity in the plate is a guide to the junction of the horizontal and superior semicircular canals. A small sharp gouge is placed in the convexity of the plate, and with a few rotary motions it penetrates the bone and exposes the ampullary space beneath the angle. The external arm of the horizontal semicircular canal may then be exposed to its posterior limit, and, if necessary, the external arm of the superior canal may also be exposed by removing its outer wall upward from the primary opening at the petrous angle of the two canals (Fig. 513).

The Bourguet protector and guide is in position, protecting the facial ridge and guiding the gouge to the petrous angle at the junction of the two canals.

Technique.—(*a*) Perform the radical mastoid operation. Remove the portion of the zygomatic root and of the roof of the external auditory meatus, as shown in Plate XXIX to facilitate the use of the curette in removing the bony tissue surrounding the canals.

(*b*) Proceed to open the petrous angle of the horizontal and perpendicular canals as described in the Surgery of the Horizontal Semicircular Canal.

PLATE XXIX



Anatomical Dissection of the Ear and Surrounding Structures.

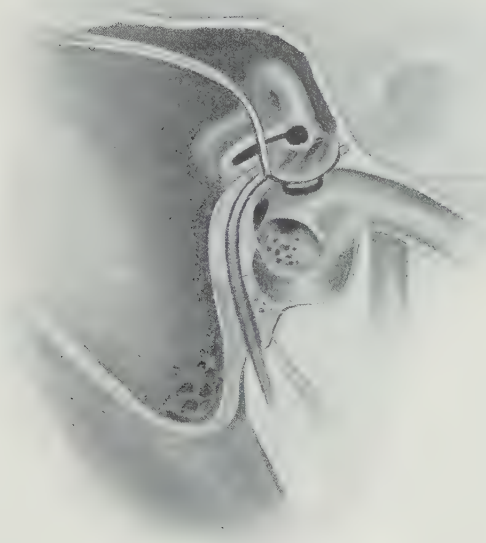
a, *a*, *a*, the facial ridge and nerve; *b*, the horizontal semicircular canal; *c*, the posterior vertical semicircular canal; *d*, the anterior vertical semicircular canal; *e*, the oval window; *f*, the round window; *g*, the promontory; *h*, the tympanic end of the Eustachian tube; *i*, the fragment of the anterior bony wall of the meatus; *j*, the internal carotid artery; *k*, the remaining portion of the floor of the meatus (the deeper portion of the floor of the meatus has been removed to expose the hypotympanum); *l*, the internal jugular vein and bulb; *m*, a section of the bone covering the facial nerve; *n*, the sigmoid portion of the lateral sinus.

(*c*) Extend the opening upward and backward, thus removing the outer walls of the horizontal and superior semicircular canals (Fig. 513).

(*d*) With a small curved gouge introduced above and beyond the outer limit of the horizontal canal (Fig. 513), remove the superior wall of the anterior vertical canal.

(*e*) Proceed to complete the opening of the horizontal and anterior vertical canals with a small curved gouge and a small thin chisel. The major portion of the work should be done with the gouge, a rotary or boring motion being used, as the blows of the mallet are liable to fracture the bone in unexpected directions and afford avenues of infection to the meninges.

FIG. 512



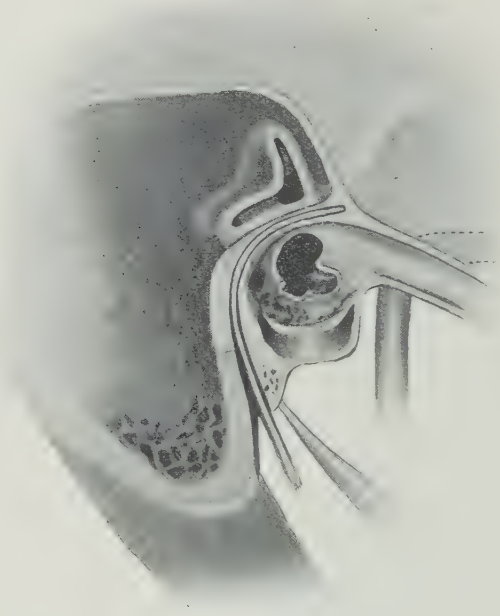
Schema showing Bourguet's operation upon the horizontal semicircular canal. The facial nerve is not actually exposed in the operation.

(*f*) Endeavor to open the upper portion of the vestibule, as this will insure better results; the semicircular canals open into it. This should be done with a small thin chisel curved on the flat. The petrous angle of the horizontal and anterior vertical canals, directly above the oval window, should first be opened and the gouge used to extend the opening downward to the vestibule. The force of the blows of the mallet should not be expended upon the facial ridge. That is, the gouge should be well above the facial ridge (not resting upon it), as to

use the facial ridge as a fulcrum in loosening the chips of bone might fracture it and cause facial paralysis (Richards).

Richards says that this route to the vestibule is safer than that *via* the inner wall of the cavum tympani, as there are no vulnerable points to be encountered except the facial ridge, whereas, in removing the bridge of bone between the oval and round windows and a portion of the promontory, the inner thin wall of the vestibule is more liable to injury, especially as the vestibule is shallow at this level and its inner wall very thin.

FIG. 513



Schema showing the Bourguet and Richards operation upon the semicircular canals, vestibule, and cochlea. The semicircular canals are opened, as shown in Fig. 512, with the protector and guide in position. The facial nerve is not exposed in the actual operation.

Then remove the bridge of bone between the oval and round windows with a thin sharp gouge, thus exposing the lower space of the vestibule. Enlarge the opening, if necessary, to expose a portion of the lower whorl of the cochlea (Fig. 513). (This figure also shows the horizontal and perpendicular semicircular canals opened.) Gently remove granulations from the vestibule, and bear in mind that the inner wall of the lower portion is thin and easily fractured.

THE RICHARDS OPERATION

Richards' labyrinth operation is essentially a careful and elaborate dissection of the semicircular canals, vestibule, and cochlea. The technique employed is admirable, though I doubt its practicability in acute suppurative labyrinthitis where the vitality of the patient is greatly lowered and the prolonged technique necessary would add greatly to the shock which always attends an extensive labyrinth operation.

Technique.—1. First perform the radical mastoid operation in the usual manner.

2. Having completed the radical mastoid operation, proceed as follows to establish as much room as possible for the execution of the labyrinth operation:

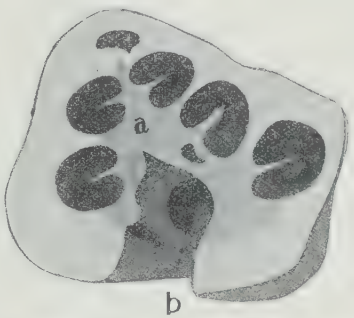
(*a*) Remove the posterior bony wall of the external meatus down to the Fallopian canal, which contains the facial nerve.

(*b*) The upper wall of the meatus is removed *almost* to the point of exposing the middle cranial fossa.

(*c*) The lower wall of the meatus is also chiselled away as much as possible. These procedures give access to the outer (lateral) aspect of the labyrinth, greatly facilitating the subsequent operation upon it.

(*d*) When the external auditory meatus is unusually convex at any portion the convexity should be reduced.

Fig. 514



Schema showing a cross-section through the cochlea from apex to base. The central shaded portion (*a*) is the modiolus. If more than the upper apical coil is removed, the internal auditory canal (*b*) at its base would be opened, thus exposing the patient to the dangers of meningitis.

(*e*) The tip of the mastoid process should be thoroughly removed without injuring the facial nerve as it makes its exit from the Fallopian canal.

(*f*) The Eustachian orifice should be fully exposed by removing the anterior wall of the meatus and the tensor tympani muscle.

(*g*) Curette the orifice of the Eustachian tube and apply adrenalin.

The foregoing steps are taken to give as free access to the labyrinth as possible. Operations upon the labyrinth are difficult under the most favorable conditions, and as they cause great shock to the patient it is important to render the parts as accessible as possible. The curettage of the Eustachian tube should be done not only to favor its closure but to prevent hemorrhage during the labyrinth operation.

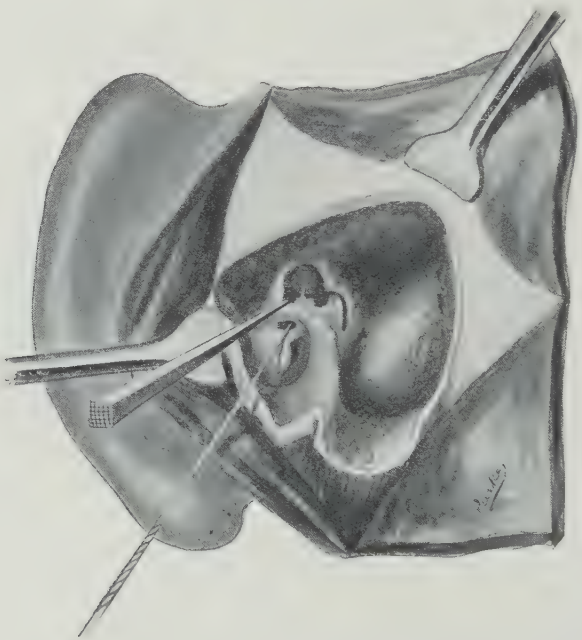
3. Remove the cancelous bone surrounding the semicircular canals (Plate XXIX). This preliminary step will enable the operator to accurately and speedily uncover the canals.

4. The horizontal semicircular canal is then opened at a point above

the oval window with a small gouge applied with a rotary boring movement. This will open it at the ampulla. The canal should then be opened, following its course posteriorly to its arch (Fig. 513). Next open the outer limb of the superior canal upward to its arch. During the opening of the external limb of the horizontal canal great care should be exercised to avoid injuring the facial nerve which lies in the Fallopian canal immediately below it and above the upper margin of the oval window.

5. The remaining portion of the anterior vertical canal is next opened, a small chisel bent at a slight angle being required for this purpose.

Fig. 515



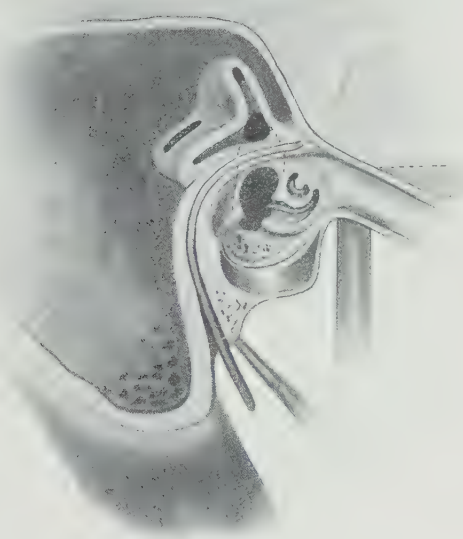
Richards' Labyrinth Operation. Showing the complete exposure of the three semicircular canals and the upper or suprafacial aspect of the vestibule. The curved probe introduced through the oval window extends upward through the opened roof of the vestibule. The tip of the chisel is at the ampulla of the external semicircular canal where the initial opening is made in Richards' operation. The posterior fossa of the cranium is not exposed in Richards' operation unless positive signs of meningitis are present.

6. The posterior (oblique) canal is then opened, thus completing the exposure of all the canals. Only about one-half of the bony walls of each canal should be removed, thus leaving small shallow grooves marking the position of the canals. When a canal is opened it presents a dark granular appearance which might be easily mistaken for clotted blood or granulation tissue.

7. The vestibule should now be opened through the solid angle of bone at the confluence of the canals where their ends communicate with the vestibule. To open the vestibule, the inner lip of the horizontal canal must be removed. The chisel must not rest upon the outer

lip or wall of the canal during this procedure, as the facial nerve lies immediately beneath it. Should the outer lip be fractured the facial nerve would be injured and facial paralysis follow. Richards urges that the outer lip of the canal be preserved intact as a protecting bridge to the facial nerve, and that the chisel be held perpendicular to the plane of cleavage during the maneuver. The vestibule is exposed as fully as possible from this point. It is subsequently more fully exposed (during the cochlear operation) by removing the bridge of bone between the oval and round windows. This completes the vestibular portion of the operation except that portion done in connection with the cochlear operation. Occasionally extensive necrosis around the facial nerve

FIG. 516

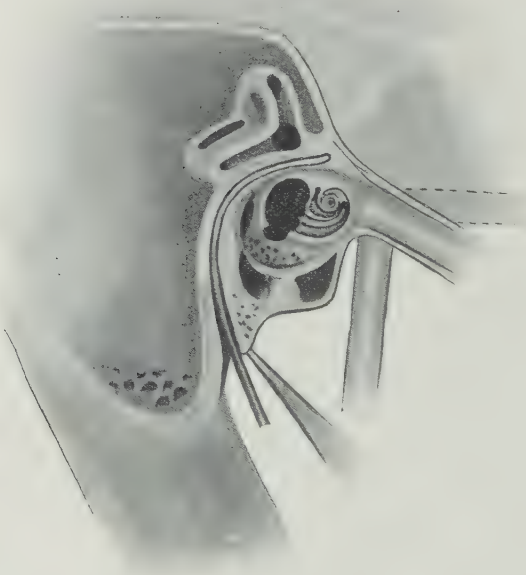


An extensive exposure of the canals and cochlea.

makes it necessary to remove the bone surrounding it. When this becomes necessary, the bone should be shaved off from above downward parallel with the nerve. When the nerve is thus exposed it lies in a bony gutter. It should then be gently lifted from this groove and the remaining portion of the bony support removed. In the downward course of the nerve, in the posterior wall of the meatus, it usually gives off a few filaments which should be cut and not torn. In removing the bony tissue supporting the horizontal portion of the nerve (parallel with and below the external semicircular canal), which corresponds to the upper and inner wall of the tympanic cavity, *the chisel should be directed from above downward, or from before backward, never from behind forward.*

8. The vestibule is still further exposed by removing the bridge of bone lying between the oval and round windows. This is accomplished by placing the point of a gouge on the bridge and tapping it with a mallet. The gouge should be exactly as wide as the bridge of bone. The force of the mallet blow should not be great enough to drive the gouge against the inner wall of the vestibule, which is not more than $\frac{1}{32}$ of an inch in thickness, and forms one of the walls of the internal auditory meatus. If this wall is fractured it may become the avenue

FIG. 517



Richards' radical operation upon the cochlea and canals. The cupola or apical whorl is removed, including the modiolus. This radical exposure of the cochlea should be performed only when meningitis is already present.

of meningeal infection. The direction of the gouge should be from above, downward and forward. The opening thus made is enlarged until the vestibule is fully exposed from this aspect.

9. The first or lower whorl of the cochlea is then exposed in a forward direction until the carotid eminence is reached (Fig. 513), a small sharp gouge of the width of the cochlear canal being used for the purpose. The gouge should be thin and have no shoulder or bevelled edge.

10. The apical whorl is next opened (Fig. 516). The apex of the

cochlea forms the most prominent point on the prominence of the inner tympanic wall, and is near the Eustachian orifice and the carotid eminence. The bone is shaved away until the dark outline of the cochlear canal is exposed through the thin lamella of bone. The chisel is directed from above downward and forward, or, in other words, in the direction of the apical whorl of the cochlea. This exposes the proximal half of the whorl.

11. The remaining half of the apical whorl is then exposed by gently chiselling away the shell of bone forming the apex down to where the first whorl or turn of the cochlea is completed. The apical whorl, as shown in Fig. 517, is thus completely removed. To attempt to penetrate more deeply into the middle whorl of the cochlea is to invite the occurrence of grave intracranial complications. The internal auditory canal, which carries the auditory and vestibular nerves, extends into the midst of the cochlea (Fig. 514), and if the modiolus or centre pin which supports the whorls is fractured by too deep extension of the operation, communication with the cranial cavity is established and infection of the meninges becomes probable.

Only when the meninges are already infected should the whole ($2\frac{1}{2}$ *whorls or turns) cochlea be removed.* This may be done in an endeavor to establish free drainage of the meningeal surface in meningitis of labyrinthine origin. The cochlea is already destroyed and meningeal infection is established, hence there can be no increase in the danger by this procedure; on the contrary, the increased facility for drainage from a point *at* the original site of meningeal infection increases the possibility of curing the meningitis.

INTRACTABLE AND UNBEARABLE VERTIGO

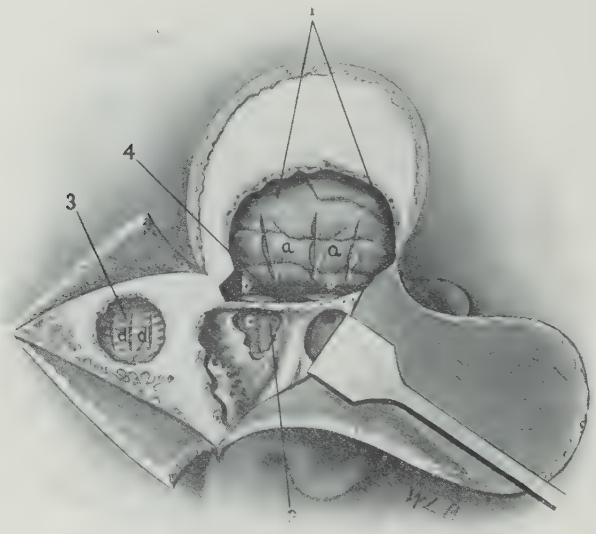
Mr. Richard Lake has devised an operation upon the vestibule and external limb of the horizontal canal for the relief of intractable and unbearable vertigo in cases of marked deafness of other origin than labyrinth infection. He does not advise the operation in cases with useful hearing, but only in those cases in which the hearing is of no practical value. Milligan had previously performed a similar though less extensive operation for the same purpose. Lake removes the stapes and opens the external limb of the horizontal canal and vestibule through its roof as in the Hinsberg operation. By this means he destroys the membranous vestibular and cochlear apparatuses, and at the same time provides free drainage against the possible suppurative inflammation which might follow the operation. Milligan only opened the upper aspect of the external limb of the horizontal canal, and thus effectually destroyed the membranous labyrinth as in the Lake operation. The advantage of Lake's operation is that freer drainage is provided. In both methods of operating the hearing is totally destroyed in the operated ear. This is, however, a negligible factor in properly selected cases, and does not have force as a contra-indication to the

operation. While the operation is not one often required, it is, nevertheless, of great economic importance to the few who are afflicted by this very annoying and distressing condition.

SURGERY OF BRAIN ABSCESS

The Surgery of Cerebral Abscess.—Abscess of that portion of the cerebrum embraced within the temporosphenoidal lobe may be opened through two routes, namely, (*a*) the tegmen tympani and antri, and (*b*) the squamous portion of the temporal bone. In some cases both routes should be employed, especially if the abscess is located high above the

FIG. 518



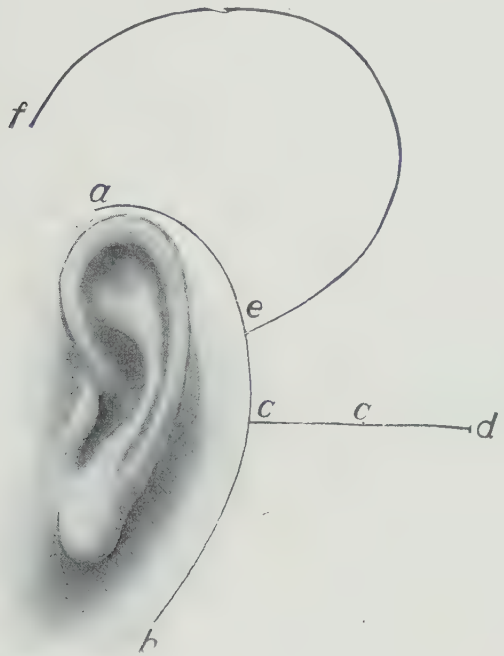
Avenues of approach to brain abscess: *a*, through the squamous plate to the temporosphenoidal lobe; *b*, through the tegmen tympani to the temporosphenoidal lobe; *c*, through the mastoid wound to the cerebellar fossa; *d*, through the cranial cortex (one and one-quarter inches posterior to the cavum tympani) to the cerebellar fossa.

tegmen tympani and contains large masses of debris and broken-down brain substance which cannot be removed through the perforation in the tegmen. In those cases in which the abscess is located near the tegmen tympani (roof of the cavum tympani) and in which the contents of the abscess are purulent or fluid, the route through the enlarged perforation in the tegmen may prove adequate for drainage.

Drainage through the Tegmen Tympani.—(*a*) A preliminary radical mastoid operation is first performed, not only to cure the mastoiditis and otitis media but to expose the tegmen or roof of the cavum tympani, the atrium of the brain infection.

(*b*) The middle-ear cavity (cavum tympani) is mopped with a cotton-wound applicator to free it of pus and blood, and if necessary adrenalin chloride solution should be applied to check the hemorrhage.

FIG. 519



The incisions for brain abscess: *a*, *b*, the primary mastoid incision; *c*, *c*, the secondary mastoid incision; *c*, *d*, an extension of the secondary incision for cerebellar abscess; *e*, *f*, the incision for abscess of the temporosphenoidal lobe of the cerebrum.

(*c*) The tegmen tympani should then be inspected under strong reflected light for oozing pus, and for the dehiscence or perforation resulting from necrosis. A probe may also be used to explore for rough and necrosed bone.

(*d*) Having located the point from which pus oozes, or where the granulations protrude from the necrosed area of the tegmen, it should be gently curetted to remove the granulations, and to expose the necrotic bone and the perforation through it. The opening should be enlarged by removing all the necrosed bone (Fig. 518, *b*), a dull curette being used for the purpose.

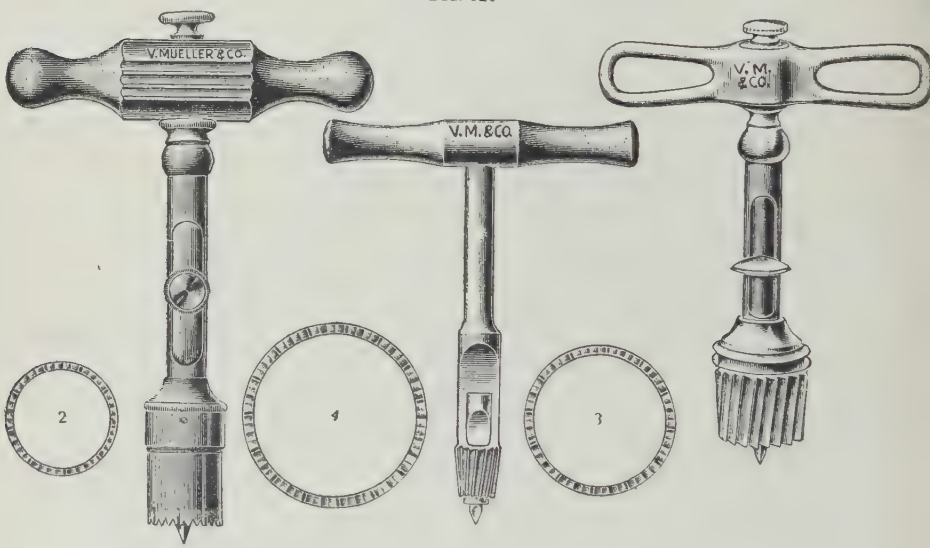
(*e*) If the abscess is located near the floor of the middle fossa immediately over the perforation in the tegmen tympani, it may be readily drained through this enlarged opening. The dura and brain substance may be incised to enlarge the channel of communication between the abscess cavity and the cavum tympani (Fig. 518). In one case coming under the author's observation the abscess cavity extended into the

brain substance for a distance of one and one-half inches, and communicated freely with the cavum tympani. Large cholesteatomatous masses were admixed with the pus, which were readily removed through the tegmen opening. In most cases in which the abscess is located as high as this, and in which large cholesteatomatous masses are present, it is impossible to evacuate the abscess through the tegmen.

(*f*) If the abscess is acute, simple drainage and irrigation are usually quickly followed by complete recovery. If the abscess is chronic, and the walls are lined with necrotic sloughs of brain substance, the healing process is much prolonged and requires careful after-treatment.

Drainage through the Squamous Plate.—The drainage of cerebral abscess through the squamous plate of the temporal bone is indicated when (*a*) the opening through the tegmen tympani is not large enough to insure adequate drainage; (*b*) when the abscess is located high in

FIG. 520



Circular trephine.

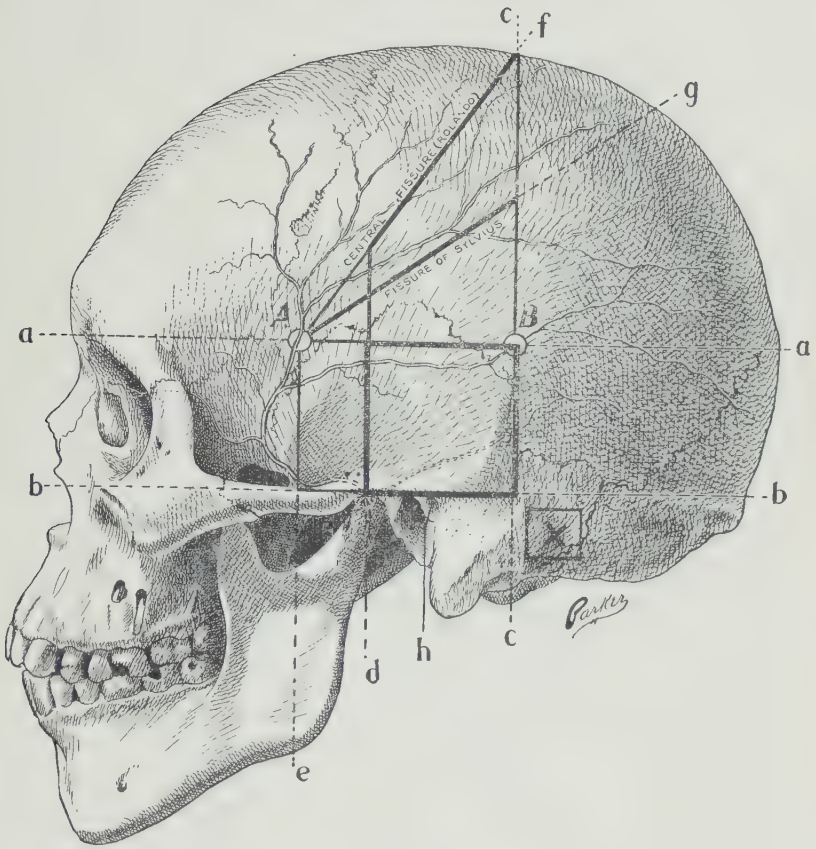
the brain substance, and only communicates with the perforation in the tegmen through a small fistulous tract; and (*c*) when the associated necrotic or cholesteatomatous masses are too large to escape through the tegmen opening, or are inaccessible through the tegmen tympani.

Technique.—(*a*) It is presumed, if the abscess is of otitic origin, that the radical mastoid operation has been performed. The skin incision should be extended from the postauricular mastoid incision in a curved direction backward, upward, and then forward, as shown in Fig. 519, *e*, *f*. The flaps are then elevated and retracted with the periosteum.

(*b*) A circular plate of bone one-half inch in diameter is then removed from the squamous portion of the temporal bone, with a circular trephine

(Fig. 520). The centre pin of the trephine should be located at a point one inch above the posterior wall of the meatus within the square area shown in Fig. 521. As the bone is of unequal thickness, one section of

FIG. 521



Kronlein's landmarks: *b*, *b*, the German horizontal line, or Read's base line, extending from the lower margin of the orbit to the occipital protuberance; *a*, *a*, the upper horizontal line, extending from the supra-orbital margin parallel with the German line; *A*, *e*, the anterior vertical line, extending upward from the middle of the zygoma at right angles to the German line *b*, *b*; *d*, the middle vertical line, passing through the condyle of the inferior maxilla at right angles to the German line *b*, *b*; *c*, *c*, the posterior vertical line, extending from the posterior margin of the mastoid process at right angles to the German line *b*, *b*; *A*, *f*, represents the location of the central fissure of Rolando; *A*, *g*, represents the fissure of Sylvius; *A*, *B*, represents the points for trephining to evacuate blood from a ruptured middle meningeal artery. Von Bergmann's area is inclosed within the square outlined by the heavy, black lines. Otitic abscess and abscess of the temporal lobe may be drained through this area. The upper line of the square represents the area for tapping the lateral ventricle. *c*, *B*, the sigmoid portion of the lateral sinus; *h*, the point for entering the antrum; *x* (in small square), area for trephining a cerebellar abscess.

the circle may be penetrated before the others. The centre pin should be set one-eighth of an inch flush with the plane of the teeth of the trephine, as this is the average thickness of the squamous plate in this

region. The trephine should be removed from time to time, and a small probe introduced into all parts of the circular cut to remove the bone dust, and to determine if the bone has been cut through at any given point. If it has, the trephine should be slightly tilted, so as to cut only at the intact portions. When the entire button of bone is severed from its attachments, a thin elevator or spatula should be inserted into the cut and the button gently lifted from the dura. The button of bone should be wrapped in a piece of sterile gauze and placed in a sterile or antiseptic solution ready for reinsertion should it be needed—that is, if pus is not found.

(*c*) Inspect the exposed dura for the following conditions: (1) The presence of pus from an associated meningitis. (2) The presence of congested and infiltrated membranes. (3) The presence of brain pulsation. Brain pulsation is usually present when the abscess is large and deeply located in the brain substance, or when the abscess is small and superficial. The absence of pulsation may, therefore, be taken to indicate a small deep-seated pus cavity or a large superficial one. Leptomeningitis with pachymeningitis may result in the fusion of the meningeal membranes, and thus obscure the pulsations which would otherwise be present.

(*d*) The dura should be incised layer by layer near the centre of the opening until its entire thickness is penetrated. It should then be seized with forceps, lifted from the underlying structures, and incised the whole diameter of the opening. If necessary, a cross-incision may be made to overcome the tension. The bloodvessels crossing the field should be cut one at a time, pinched with artery forceps, and ligated if necessary, as the blood might otherwise penetrate between the membranes and produce pressure, or carry infection to other parts.

(*e*) The exposed membranes, brain substance, and bone edges should be dusted with iodoform powder to protect them from the infected pus when the abscess is opened.

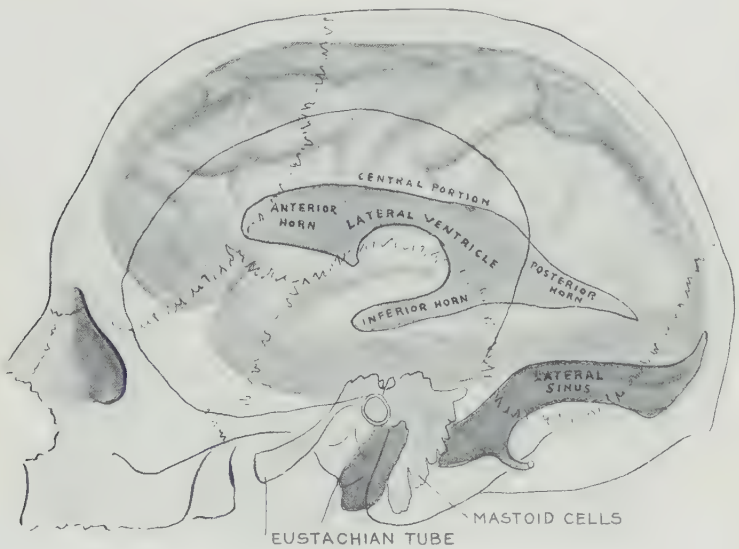
(*f*) The choice of an instrument for opening the abscess, or for exploring for it, is a matter of some importance. A hollow needle or cannula has commonly been chosen for this purpose. The late Christian Fenger preferred a long, slender-bladed scalpel, as it inflicted less damage to the brain substance, and at the same time was superior in locating and evacuating the pus. The needle and cannula are objectionable on account of the brain substance entering their lumen when suction is applied, thus interfering with the detection and withdrawal of the pus.

The knife should be passed a distance of one inch into the brain substance, then slightly rotated and lifted to open the channel for the discharge of the pus. If pus does not appear, it should be introduced a half inch deeper and similarly rotated and lifted. The knife should be passed to a greater depth than this with great caution, as the lateral ventricles (Fig. 522) may be opened and exposed to infection. If pus is not found, the knife should be withdrawn and reinserted in another plane, and if necessary in several planes, until the abscess is located and evacuated. If care is taken to keep the exposed area of the surface of the brain and the knife surgically clean, there is but slight danger from this

method of procedure, even when several punctures are made. The parts of the brain thus incised are not functionally injured, as the incision is clean-cut and the instrument is sterile.

(*g*) If the pus is too thick to flow readily through the incision, or the necrotic sloughs of brain substance are too large to pass through the incised channel, the encephaloscope designed by Whiting should be used. It should be introduced over the blade of the knife while it is still in the brain, the blade acting as a guide to the abscess. Through the opening thus obtained the pus escapes, and the sloughs may be removed

FIG. 522



A transparent skull showing the relation of the sutures, ventricles, Eustachian tube, tympanic cavity, mastoid cells, and lateral sinus of the left side of the head.

with forceps. When the abscess cavity is emptied its walls may be inspected by the aid of reflected light. If they are necrotic they should be curetted until healthy brain substance is exposed. Should such material be left in the cavity, the infection and inflammation will be much prolonged. Whiting's encephaloscope affords a means of treatment of great advantage that should be utilized whenever the conditions present warrant it.

(*h*) The abscess cavity should be irrigated with a warm antiseptic solution until the return flow is clear. With Whiting's encephaloscope or brain speculum the irrigation is a simple matter, as it allows the nozzle of the syringe to be introduced and at the same time allows the fluid to make its exit into the pus basin. If the encephaloscope is not used, a cannula should be introduced the lumen of which is larger than the one

attached to the syringe, as this allows a return flow of the pus and irrigation solution. This provision is necessary, because, if the outflow of the irrigating solution is blocked, the pressure of the retained fluid may cause it to extend beyond the walls of the abscess cavity to other parts of the brain.

(*i*) The first dressing should consist of a drainage wick of gauze, a protective covering of antiseptic powder, and an outer absorbent gauze pad. The drainage wick should be within the cavity and in contact with the external absorbent gauze pad. The proximal end of the gauze wick should be folded over the bony wound and dusted with a mixture of iodoform and boric acid (1 to 5), to prevent adhesion between the gauze wick and the outer absorbent gauze pad, as it may be necessary to leave the gauze wick in position for several days; whereas the outer gauze pad may, and in many instances should be removed daily. In acute cases the walls of the abscess cavity may collapse and heal in a day or two. Chronic cases require several days or weeks to heal. Macewen recommends that in some acute cases only the outer gauze pad be used, and if there is no temperature or pain, that it be left undisturbed for three weeks, the obvious purpose being to avoid the possibility of infecting the wound by removing the dressing. When, however, the discharge is sufficient to soil the outer gauze pad, it should be removed daily until healing is completed.

SURGERY OF CEREBELLAR ABSCESS

There are three routes available for evacuating abscess of the cerebellum, namely: (*a*) Through the mastoid wound *via* the recess at the angle of the sigmoid knee (Fig. 518, *c*), that is, through the recess between the inner wall of the antrum and the knee of the sigmoid sinus; (*b*) through the inner wall of the sigmoid sinus when the vessel is thrombosed and has been exenterated; (*c*) through the skull one and one-fourth inches posterior to the meatus and below the level of the lateral sinus (Fig. 521, *x*). The lower border of the lateral sinus may be determined by an imaginary line passing from the upper margin of the zygoma to the upper boundary of the external auditory meatus, and thence backward to the occipital protuberance (Fig. 521, *b*, *b*). Having constructed this line, trephine below it one and one-fourth inches posterior to the auditory meatus. This will open the skull below the lateral sinus and will afford the most available external route to the cerebellar abscess.

(*a*) If the abscess is immediately behind the petrous pyramid of the temporal bone it may be easily reached through the mastoid wound *via* the recess between the knee of the lateral sinus and the antrum.

(*b*) If the lateral sinus is thrombosed (and it is often the source of the cerebellar abscess) its walls should be carefully searched for necrotic areas, not alone as an avenue of approach to the abscess but as a means of tracing the location of the abscess through the fistulous tract leading

from the sinus to the abscess cavity. This route may be utilized to evacuate the abscess, though the subsequent treatment through this route is difficult to carry out on account of the restricted and deep situation of the opening in the mastoid wound. This is also true of the first (*a*) route.

(*c*) The external route through the skull (Figs. 518, *d*, and 521, *x*) is generally preferable on account of its accessibility.

The technique of the operation is otherwise similar to that described for cerebral abscess.

SURGICAL TREATMENT OF SEROUS MENINGITIS

Serous meningitis has no characteristic symptoms by which it may be positively diagnosticated from purulent meningitis. If, however, after completing the radical mastoid operation the tegmen tympani or antri is opened and serous fluid escapes and the meningeal symptoms subside, the diagnosis of serous meningitis may be made (Fig. 518, *b*, *c*).

The surgical treatment consists in doing a decompression operation, removing the tegmen tympani or the tegmen antri and allowing the serous effusion to escape. The after-treatment consists in the usual mastoid dressings.

Repeated lumbar punctures and the escape of the cerebrospinal fluid have been attended with brilliant success in some cases.

SURGICAL TREATMENT OF EXTRADURAL ABSCESS OR PACHYMENINGITIS CIRCUMSCRIPTA

Circumscribed pachymeningitis, or extradural abscess, located over the tegmen tympani or antri in the middle fossa of the skull, may be successfully treated in nearly all cases by first performing the radical mastoid operation, and then removing the roof of the cavum tympani and antri, and evacuating the purulent secretion. An extradural abscess is a localized meningitis, the circumference of which is walled off by a plastic exudate.

An early operation upon these cases prevents the spread of the infection in the form of a brain abscess and leptomeningitis, which are more serious affections. Leptomeningitis is usually fatal, though a few cases have recovered under surgical drainage.

SURGICAL TREATMENT OF THROMBOSIS OF THE LATERAL SINUS

An infective thrombus is more often found in the sigmoid portion of the lateral sinus than in any other of the intracranial sinuses. Early recognition and surgical treatment is of the greatest advantage to the patient, as many cases thus recognized and treated recover.

Indications for Lateral Sinus Operation.—Since the findings of Gruening, Libman, and Oppenheimer, as to the relation of bacteriemia (bacteria in the blood) to infective thrombosis of the intracranial sinuses, the indications for treatment are rendered possible at a much earlier stage than formerly, and with a correspondingly improved prognosis. Bacteriemia, usually a streptobacteriemia, is present in the earlier stages of thrombosis of the lateral or other sinuses before the distinctive septic symptoms, as a normal or subnormal temperature, followed by chills and rigors and a rapid and great rise of temperature appear. When, therefore, there is a mastoiditis attended by a constant elevation of temperature, and blood-cultures show streptococci present in the blood-stream (streptobacteriemia), the sinus should be operated. In such cases, uncomplicated by meningitis, the death rate after operation should not be more than 10 per cent. Indeed, it should be less than this, as shown by Dr. George L. Tobey in a series of cases in which the thrombosis of the sigmoid sinus was operated by ligation only with remarkable results. In mastoiditis with a continued high temperature, headache, etc., cultures of the blood should always be made, and if bacteria are found the sinus should be treated by some form of operative interference, as it is made obvious by the blood-cultures that one of them, usually the sigmoid, is involved by an infective thrombotic process.

The *earliest indication* of sinus thrombosis is, therefore, mastoiditis with continued elevation of temperature and bacteriemia.

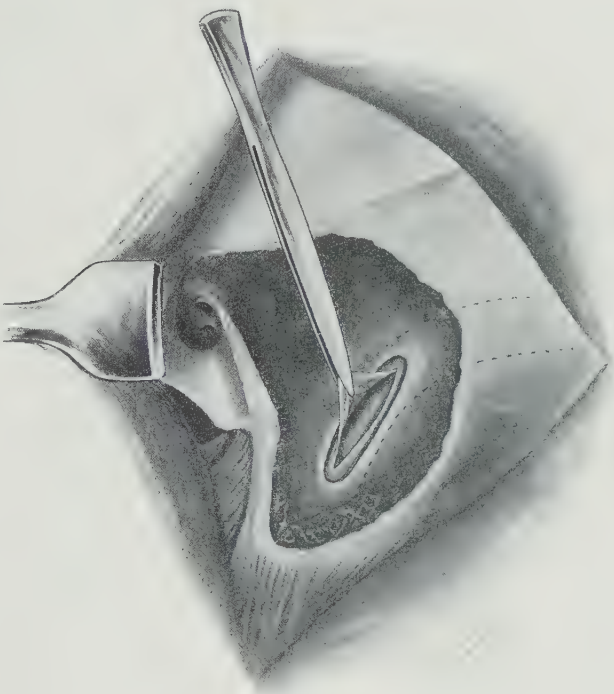
When the thrombic process has advanced to the stage of breaking down, and the blood-stream is periodically flooded with bacteria and septic material, the symptom complex is radically different from what it was in the earlier bacteriemic stage. The temperature now fluctuates from below or near normal up to 104° to 106° F. daily. The onset of the rise in temperature is attended by severe chills and rigors, or in infants and young children by convulsions.

A *later and more obvious indication* for the surgical treatment of sinus thrombosis is mastoiditis with a temperature fluctuating daily from near normal to 104° to 106° F., the rise in temperature being ushered in by chills and rigors, or by convulsions in infants and young children. While the prognosis is not so favorable in this stage of the disease it is nevertheless very favorably modified by the proper surgical treatment, *i. e.*, the exposure and evacuation of the sigmoid sinus and the ligation or excision of the internal jugular vein.

When the thrombus extends backward in the lateral sinus toward the torcular it has been recommended that the clot be removed with a curette. This procedure is liable to injure the intima of the vessel, thus creating favorable conditions for subsequent infective clot formations. A better plan is to continue the exposure of the lateral sinus toward the torcular, incising the membranous wall of the sinus with blunt-pointed scissors as it is exposed. Continue the exposure of the sinus to the torcular, if necessary, or until a free flow of blood is obtained. This method of procedure is safer and much more certain to establish a flow of blood than by curettage.

Technique.—(*a*) A preliminary mastoid operation is performed. If the mastoiditis and otitis are acute, the simple mastoid operation may be all that is necessary, the cavum tympani being unmolested; if, however, the mastoiditis and otitis are chronic, and the labyrinth is involved by the infective process, the radical mastoid operation should be performed. Richards reports 11 cases of labyrinthine disease upon which he operated, performing more or less extensive exenterations of the labyrinth, of which three were affected by thrombosis of the lateral sinus. This, as he says, points strongly to the labyrinth as a possible atrium of infection.

Fig. 523



Thrombus of the lateral sinus exposed.

(*b*) Remove the dense or necrosed bone covering the mastoid aspect of the lateral sinus as extensively as possible, thus exposing the membranous sinus to observation and operation. Determine whether a perisinus abscess (extradural abscess of the sinus) is present. Note the texture of the membranous sinus, whether velvety, covered with granulations at certain points, or necrosed. Palpate it with the finger to determine its resistance, whether doughy, hard, or fluid. Some surgeons recommend that the sinus be exposed in every mastoid operation, and that a portion of its contents be withdrawn with a hypodermic needle to

ascertain if pus is present. This is a reprehensible practice, as it is an unreliable method of determining the presence of pus, and exposes the sinus to the danger of infection. Whiting recommends that the tip of the finger be placed as near the jugular bulb as possible and then drawn upward toward the knee, noting whether the stripped sinus refills below the finger. If it does, the jugular bulb is open. The sinus should then be stripped from above downward toward the jugular bulb, and the same observation made of the upper portion of the sinus. If it refills, the sinus is open above; if it does not, it is closed by a thrombus. Having determined to open the membranous sheath of the sinus, see that iodoform and boric acid powder (1 to 5) and a strip of iodoform gauze (1 x 24 in.) are in readiness in case free hemorrhage occurs.

(*c*) Incise the whole length of the exposed portion of the membranous sinus (Fig. 523), and if the hemorrhage is free it should be closed by turning in the cut edges of the membrane and packing the bony opening with the strip of iodoform gauze. A few moments of hemorrhage should be allowed, as it may wash out the infective material and lead to recovery.

If the incision is not followed by hemorrhage, the thrombic clot, whether it be solid or undergoing disintegration, should be removed with a dull curette. The portion of the clot near the jugular bulb should be curetted until blood appears at the lower end of the opening. The curette should then be passed upward through the knee of the sinus, and the clot removed from this part of the sinus. The flow of blood from this end of the sinus is evidence that this portion has been cleared of the thrombus. Both ends of the sinus should give forth blood. The lower or jugular end should be kept closed with the finger while the upper end is being curetted, as too much blood might otherwise be lost, or the surgeon be impelled to work with undue haste. Having cleared the sinus of the clot, it should be filled with the iodoform boric acid powder, the edges of the membrane turned in and the bony aperture filled with iodoform gauze, and the usual mastoid drainage and absorbent dressings applied.

(*d*) The dressing may be removed at the end of from twenty-four to forty-eight hours, and the gauze removed from the bony aperture of the lateral sinus without danger of hemorrhage.

(*e*) The after-treatment consists in the usual mastoid dressings heretofore described.

Should pain, chills, and a rise of temperature occur, the dressing should be removed at once and the parts examined to determine the conditions which gave rise to the symptoms. If pus is present, endeavor to trace it to its source. It will usually be necessary to reopen the sinus and extend the curettement, as the sepsis is probably from within the sinus caused by fragments of the thrombus that were probably left at the time of the primary sinus operation. The sepsis may, however, have its origin from a perisinus abscess, and it may become necessary to resect the jugular vein and bulb.

RESECTION OF THE INTERNAL JUGULAR VEIN

The indications for the ligation and resection of the internal jugular vein have not been fully established. It is still a question as to when the resection increases the danger of spreading the infection, and when it prevents spreading the infection from a thrombosed lateral sinus. If the internal jugular vein is ligated and resected the anastomotic channels, of which there are many, will receive the venous blood current, provided there is a flow of blood through the sinus. If only the lower portion of the lateral sinus is closed by an infected thrombus, the blood may be forced into the superior petrosal sinus and cause thrombosis in it and the cavernous sinus, with which it communicates. If the entire sigmoid portion of the sinus is blocked by a thrombus, the blood current may be forced backward into the superior longitudinal sinus. If the thrombus is limited to the jugular bulb, the blood current may be forced into almost any or all of the intracranial sinuses. In ligating the internal jugular vein the effect upon the blood current is the same as that in jugular bulb thrombus. The question as to when the jugular vein should be ligated and removed from the neck resolves itself into the consideration of the foregoing facts, and may be stated as follows:

(*a*) It may be ligated and removed when the entire sigmoid sinus and jugular vein are thrombosed and should be obliterated by operative procedure. The jugular vein should be removed first, however, to obviate the danger of disseminating particles of the thrombus which may become detached during the exenteration of the sigmoid sinus.

(*b*) The internal jugular vein may be ligated and removed when the jugular bulb is thrombosed, the jugular bulb being removed after the resection of the vein, if the sigmoid sinus is obliterated at the same time, whether it is infected or not. If the sigmoid sinus is left open the infective material from the jugular bulb may be forced backward through this sinus, and thence through the petrosal to the cavernous sinuses.

(*c*) The internal jugular vein may be ligated and resected when it is thrombosed by extension from a similar condition in the sigmoid sinus and jugular bulb.

(*d*) The jugular vein should not be ligated and resected when there is a flow of blood through the sigmoid sinus.

(*e*) In a general way, it may be said that the jugular vein may be ligated and resected when the sigmoid sinus is completely blocked with an infected thrombus.

The object of the ligation and resection of the internal jugular vein is to prevent the dissemination of the infection to other parts of the body, as the lungs, spleen, liver, kidneys, intestines, etc. Statistics show more favorable results if this is done when there is complete blockage of the sigmoid sinus, and worse results when the sigmoid sinus has a current of blood passing through it.

Technique.—(*a*) Extend the mastoid incision downward along the anterior border of the sternomastoid muscle to the sternal notch (Plate XXX and Fig. 476).

(*b*) Retract the sternomastoid muscle backward and separate the fascia and other structures by blunt dissection until the internal jugular vein is exposed.

(*c*) The pneumogastric nerve runs between the internal jugular vein and the carotid artery and should be respected.

(*d*) Ligate the internal jugular vein just above the sternum and just below the floor of the external auditory meatus (Plate XXX).

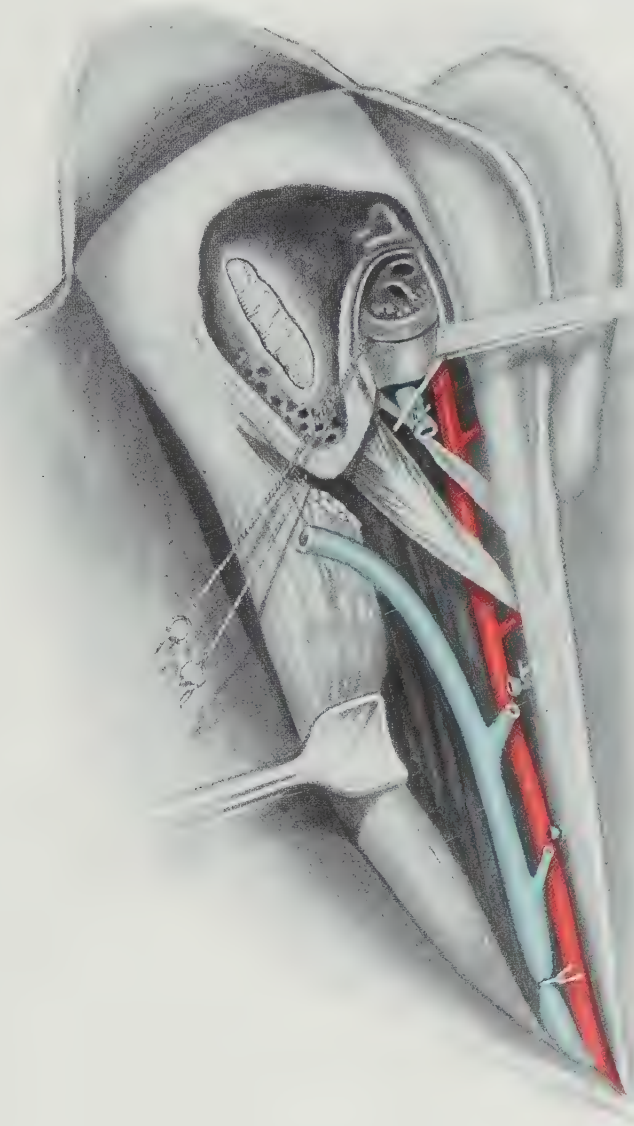
(*e*) Ligate all the branches of the vein given off between the upper and lower ligations of the jugular vein (Plate XXX).

(*f*) Sever the jugular vein just above the lower and just below the upper ligatures. Then sever all the branches close to the jugular vein and remove the vein from the neck. A gauze pad should be placed under the vein before resecting it to protect the tissues from infection.

(*g*) The sigmoid sinus is next opened and the thrombus removed as described in the preceding section. The danger of disseminating the disintegrating thrombus through the jugular vein is largely obviated by its removal, though the anastomotic communications are not altogether obliterated.

(*h*) The sigmoid sinus should be packed and obliterated (Plate XXX), and the mastoid wound dressed as previously described, with the exception that the lower half of the mastoid incision be left open so that the region of the exenterated sigmoid sinus may be subsequently inspected and dressed through it. The incision in the neck should be closed throughout its entire length, a secondary incision being made one inch posterior to the lower angle. This incision should be made to communicate with the primary neck wound by tunnelling beneath the skin. A spiral tube containing a small wick of gauze should be introduced into the secondary incision and extended beneath the skin to the lower portion of the primary neck wound, as shown in Fig. 476. The object of the secondary incision is to prevent an unsightly scar. As the primary wound was occupied by an infected and thrombosed vein, the tissues may have become contaminated. Under these circumstances, if the tube dressing were introduced into the wound through the primary incision, the tissues around the tube dressing would heal slowly and cause a retracted and disfiguring scar. The secondary incision, being removed from the region of infection, will, after the tube is discontinued, heal quickly with little scar and disfigurement.

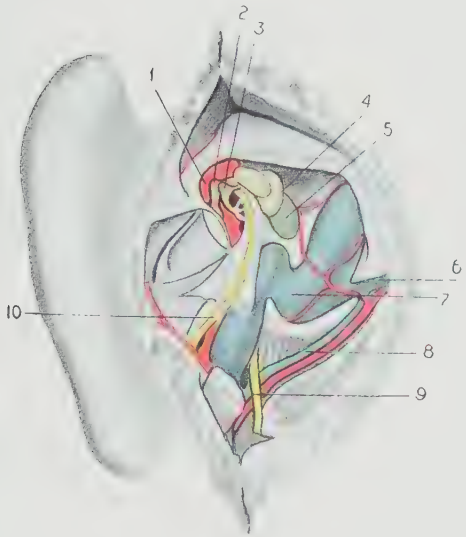
(*i*) The after-treatment, in so far as the wound in the neck is concerned, consists in the removal of the drainage-tube dressing at the end of the third day, or earlier if pain and temperature arise and persist. In those cases in which the neck wound is not infected, the tube dressing may be dispensed with after the first dressing, a small gauze wick being inserted only a little distance into the wound to carry away the excess of secretions. The channel occupied by the tube will quickly fill by granulation, and at the third dressing the gauze wick may be omitted to



Combined Operation for the Removal of a Thrombosed Sigmoid Sinus, Jugular Vein, and Jugular Bulb.

The sigmoid portion of the lateral sinus has been exenterated and packed with gauze. The jugular vein and its branches have been ligated and severed, and the floor of the meatus is being removed with a Gigli saw to expose the jugular bulb. The facial nerve has been exposed and retracted forward with a gauze tape to permit the bone which encloses it to be removed, as it is in the operator's pathway to the jugular bulb, though this was not necessary in this particular dissection.

PLATE XXXI



Anatomy of the Grunert-Panse Exposure of the Jugular Bulb. (After Bardeleben.)

Grunert removes the tip of the mastoid process and then proceeds toward the jugular foramen at the base of the skull. When the jugular foramen is reached he removes the outer and posterior portion of the bony ring encircling the vein. As shown in the drawing, the facial nerve lies in the way. Panse exposes it, removes it from its canal, displaces it forward, and proceeds to expose the jugular bulb.

1, tympanic cavity; 2, malleus; 3, incus; 4, posterior semicircular canal; 5, saccus endolymphaticus; 6, mastoid emissary vein; 7, lateral sinus; 8, occipital vein; 9, spinal accessory nerve; 10, facial nerve.

allow the cutaneous edges of the incision to approximate and unite. The scar resulting will be slight and the cosmetic effect good.

The sigmoid and mastoid wounds should be dressed as previously described.

SURGERY OF THE JUGULAR BULB

The indications for the removal of the jugular bulb are (*a*) extensive necrosis in the region of the bulb; (*b*) severe systemic infection from the disintegrating thrombic clots; and (*c*) the desire to remove every vestige of the foci of infection in order to give the patient the greatest chance of recovery.

Technique.—(*a*) The mastoid operation is first performed as previously described. The simple mastoid operation is performed if the case is acute and there are no special indications, as labyrinthine suppuration and necrosis, for opening the cavum tympani. Cerebral abscess with the atrium of infection through the tegmen tympani, and sigmoid sinus thrombosis with the atrium of infection through the labyrinth, etc., necessitates the performance of the radical mastoid operation.

Fig. 524

The first step in the Passow-Trautmann plastic operation for the closure of a persistent retro-auricular opening.

Fig. 525

The first and second steps in the Mosetig-Moorhof plastic operation.

(*b*) The internal jugular vein is next resected as described in the preceding section (Plate XXX).

(*c*) The sigmoid sinus is exposed, exenterated, and packed with gauze (Plate XXX).

(*d*) The floor of the external auditory meatus is removed, as it is in the pathway to the bulb (Plate XXX and XXXI).

(*e*) The facial nerve may be exposed, as recommended by Panse, when it lies in the pathway to the bulb. The nerve should be lifted

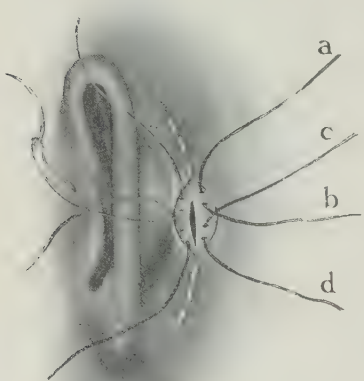
Fig. 526

The third step in the Mosetig-Moorhof plastic operation for the closure of a persistent retro-auricular opening.

Fig. 527

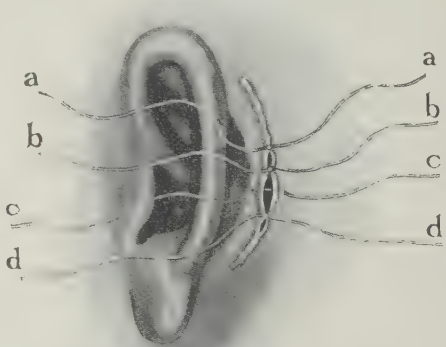
The fourth step in the Mosetig-Moorhof plastic operation for the closure of a persistent retro-auricular opening.

Fig. 528



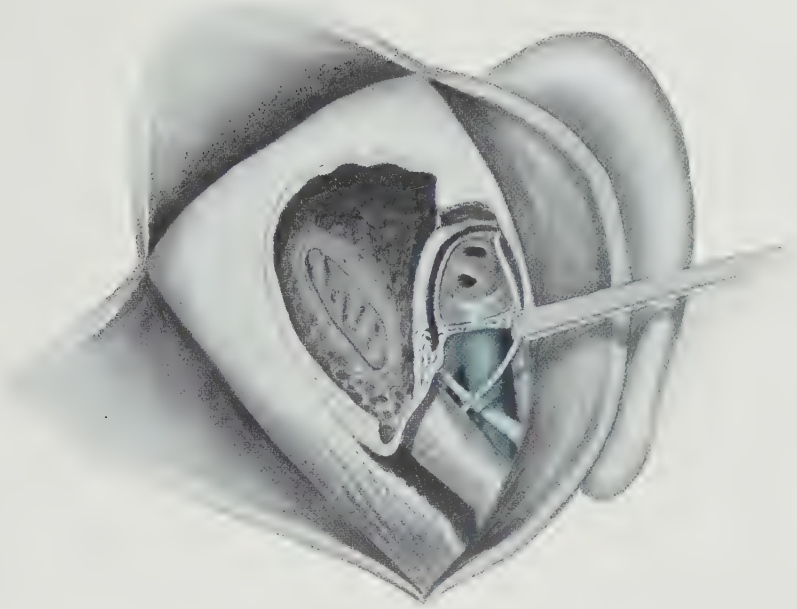
The second step in the Passow-Trautmann plastic operation for the closure of a persistent retro-auricular opening. The sutures *a b* and *c d* are to be tied to the opposite sutures to bring the periosteum together.

Fig. 529



The third step of the Passow-Trautmann plastic operation. Closing the skin.

PLATE XXXII



Exposure of the Jugular Bulb Completed, the Sigmoid Sinus Exenterated and Packed with Gauze and the Facial Nerve Lifted from its Canal and Retracted Anteriorly. The facial ridge is usually located more anteriorly over the jugular bulb than shown in the drawing.

from its exposed canal, a strip of gauze passed around it, with which it is retracted anteriorly, as shown in Plates XXX and XXXI.

(*f*) The styloid process, together with the lower portion of the bone which previously supported the facial nerve, and that portion of the mastoid tip which obstructs the path of the bulb, should be removed with a chisel, bone forceps, or a Gigli saw, as shown in Plate XXX. The saw should be placed in front of the fragment of the floor of the meatus, the anterior wall having been previously removed. One end should be passed backward beneath the tip of the mastoid process, the sternomastoid muscle being partially severed (Plate XXX), and the other backward and over it, and the bone, including the styloid attachment and the anterior portion of the mastoid tip, sawed through (Plates XXX and XXXI). The remaining portion of the bone, especially that lying beneath the floor of the meatus, may be removed with bone forceps.

(*g*) If the transverse process of the atlas projects outward into the field of operation, it should be removed, care being exercised to avoid injuring the vertebral artery (Bardeleben).

(*h*) The outer portion of the thin bone encircling the jugular bulb should be removed with bone forceps.

(*i*) The jugular bulb, being exposed to surgical interference, should be examined, and its condition noted for scientific purposes. As the sigmoid sinus above and the internal jugular vein below have already been obliterated and removed, there is no added danger in removing the bulb which forms the connecting link between them (Plate XXXII).

(*j*) The jugular bulb should be removed from the jugular fossa with a curette.

(*k*) The primary dressing should consist of a gauze wick, the distal end of which is inserted into the jugular fossa, and the proximal end in contact with the external absorbent dressing. The mastoid, sigmoid sinus, and neck wounds should also be drained by spiral tubes with a small gauze wick in each.

(*l*) The after-treatment consists in applying suitable internal drainage and external absorbent dressings until all suppuration ceases and the cavities have healed. The mastoid wound should heal by granulation, finally becoming covered with epidermis. Should exuberant granulations form, they should be reduced with caustic applications or with the electric cautery, though they will disappear in a few days if Emil Beck's bismuth paste (bismuth subnitrate, 1 part; vaseline, 2 parts) is used to fill the mastoid wound. The paste should be used daily and strands of catgut introduced to promote drainage. Should the mastoid bony surface fail to heal within from four to ten weeks, it should be freely exposed (the postauricular wound is left open at the time of the primary operation), curetted, the hemorrhage checked, and Thiersch grafts applied as previously described.

CLOSURE OF POSTAURICULAR FISTULA

The Mosetig-Moorhof Method.—This method is adapted to the closure of small openings and is performed as follows: (*a*) The edges of the fistulous openings are freshened; (*b*) a skin flap corresponding in size with the opening is made below it, a pedicled attachment being left at the upper portion of the flap; (*c*) the flap is then turned upward and placed in the fistulous opening with the skin surface inward; (*d*) it is then fixed in this position by four sutures; (*e*) finally, the freshened edges of the fistulous openings are brought together over the raw surface of the skin flap, thus forming an epithelial lining on the inside as well as on the outside of the fistulous opening (Figs. 525, 526, 527).

Passow-Trautmann Method.—(*a*) Make a circular incision about one-eighth of an inch or more (Trautmann) from the edge of the fistulous opening and separate the periosteum and skin; (*b*) unite the everted margins of the periosteum thus loosened with absorbable catgut sutures; (*c*) loosen the skin external to the incision and unite the edges over the first periosteal flaps with sutures (Figs. 524, 528, 529).

CHAPTER LIII

FACIAL PARALYSIS

The Plastic Surgery of the Facial and Hypoglossal Nerves.—The facial nerve is subject to the same diseases peculiar to other peripheral nerves, the most frequent affection being paresis or paralysis.

Paralysis is characterized by facial deformity, due to the immobility of the muscles supplied by the facial nerve. The manifestations are

FIG. 530

Facial paralysis of otitic origin. The patient is attempting to close both eyes and to draw the mouth on both sides; the right facial nerve being paralyzed, she is unable to close the right eye or to contract the right angle of the mouth.

inability to raise the eyebrow, the skin of the forehead, lip, and cheek, and to completely close the eye. The attempt to distend the buccal cavity is attended by the escape of air through the paralyzed side of the mouth. There is also inability to pucker the lips in whistling, because the angle of the mouth droops; this causes the patient a certain embarrassment in speech (Fig. 530).

Etiology.—1. Exposure to cold and wet, followed by neuritis and perineuritis of the facial nerve.

2. A neuritis due to toxemia, syphilis, rheumatism, diabetes, gout, leukemia, diphtheria, and other infectious diseases.

3. Tumors affecting any part of the course of the facial nerve, as intracranial, intra-osseous, and external neoplasms.

4. Traumatism, one of the most frequent causes of facial paralysis, and one which concerns the otologist. The facial paralysis may arise during suppuration of the middle and internal ear, especially chronic suppuration, or suppuration persisting after operative procedures for its cure.

Facial paralysis may also result from packing the mastoid wound too tightly after a mastoid operation. It is known to have been caused by the very means devised for the protection of the facial nerve during an operation, namely, Stacke's protector in the hands of an inexperienced assistant, who presses it too firmly against the facial canal or twists it while it is in the aditus ad antrum.

Curettage of the middle ear for granulations, where the facial nerve is not covered by bone, may injure the nerve and cause paralysis.

The vigorous cauterization of granulations in the middle ear with chromic or other caustic acids may also produce facial paralysis. One such case came under the author's observation.

Treatment.—The treatment is divided into:

1. Medical (local and expectant).
2. Surgical.

Paralysis of toxic origin, following exposure to cold or infectious diseases, is usually slight, recovery occurring in from one to six months by the natural process of repair. The usual treatment in such cases is elimination of the toxins by catharsis, the administration of strychnine and other tonics, facial massage, and electricity. These procedures are used principally to keep up the muscular tonicity while the nerve is regaining its normal function. Paralysis after a mastoid operation from too firm packing, or violent reaction, usually subsides within a short time after the cause is removed. When a tumor is pressing upon the facial nerve, or the nerve is injured in the removal of the tumor, the paralysis frequently disappears soon after the completion of the operation.

In all other conditions causing facial paralysis, wherein the continuity of structure of the nerve has been destroyed for a greater distance than the process of repair will bridge over, a surgical operation is required to effect a cure.

In order to understand the surgery of the facial nerve it is necessary to have a clear conception of its anatomy and physiology.

The facial nerve arises from a large group of cells situated in the upper portion of the medulla oblongata near the junction of the medulla and the pons.

From this nucleus it passes up to the fourth ventricle, forming a knee, to the nucleus of the sixth nerve, and comes out at the junction of the pons and medulla in connection with the sixth nerve. The fibers of the facial lie on the inner side of this composite nerve. From this point the nerve passes through the internal auditory meatus, through the

Fallopian canal, beneath the posterior and lower border of the annulus tympanicus, through the anterior border of the mastoid process, and then emerges from the stylomastoid foramen. From this point it passes forward into the substance of the parotid gland, within which it divides into three great branches, known as the *pes anserinus* (goose foot). One branch goes to the muscles of the forehead, the eyelid, and the upper portion of the malar zygomatic region. The second passes across the face, supplying the angle of the nose and the muscles that raise the upper lip. The third supplies the muscles at the angle of the mouth, the lower lip, the platysma, and the stylopharyngeus muscle.

At the exit of the nerve from the stylomastoid foramen one branch, the auricularis posterioris profunda, is given off, and goes to the muscles of the neck. The interosseous portion of the facial nerve gives off a number of small branches communicating with other nerves, as the fifth and the glossopharyngeal.

The function of the nerve is to supply the muscles of expression, as mentioned above, and it is, therefore, a motor nerve. However, a certain amount of sensitive fibers are contained within it, due to its gross association with the other intracranial nerves.

THE SURGERY OF THE FACIAL NERVE

The operative procedures for the cure of facial paralysis are:

1. Suture of the severed ends of the facial nerve.
2. Plastic operations.
   - (*a*) The union of the facial and hypoglossal nerves.
   - (*b*) The union of the facial and spinal accessory nerves.
   - (*c*) The union of the facial and the glossopharyngeal nerves.

The first procedure, that is, the suturing of the accidentally severed ends of the facial nerve, seems to be unnecessary, because if only moderate loss of substance between the two ends exists, the proximal ends of the nerve will regenerate and unite with the distal end without suturing.

In the plastic operations, the union between either the facial and spinal accessory (*b*) or the glossopharyngeal (*c*) gives rise to so many untoward symptoms following the procedures that they have been practically abandoned in favor of the union of the facial and hypoglossal nerves (*a*).

The Methods of Anastomosing the Facial and Hypoglossal Nerves.—

1. End to end.
2. End to side.
3. Side to side.

The easiest method is the end-to-end operation, and it is the most productive of success, but it necessitates paralysis of the muscles of the tongue. The end-to-side operation is to be preferred in all cases, as paralysis of the tongue is avoided. The side-to-side procedure has only been performed once, with a poor result.

Plastic Surgery of the Facial and Hypoglossal Nerves; Anastomosis of the Facial and Hopoglossal Nerves.—**Technique.**—(*a*) General anesthesia, the patient having been prepared as for any other major operation.

(*b*) An incision of the skin should be made, beginning at the tip of the mastoid process, near the lobe of the auricle, and extending downward and forward along the anterior border of the sternomastoid muscle to the level of the cricoid cartilage of the larynx.

(*c*) It should then be carried through the superficial fascia and the platysma muscle, thus exposing the sternomastoid muscle. The external jugular vein is usually sacrificed in this procedure, the severed ends being tied.

(*d*) The anterior border of the sternomastoid muscle and the internal jugular vein should be located, and retracted posteriorly, to expose the hypoglossal nerve, as shown in Plate XXXIII. The posterior belly of the digastric muscle is located more anteriorly and superiorly, as it extends from the mastoid tip to its pulley.

(*e*) The dimensions of the parotid gland, which is situated on the posterior border of the ramus of the inferior maxilla, should be determined, as the facial nerve divides into three branches within its substance. Having located the boundaries of the parotid gland, trace the facial nerve to it. The nerve may then be traced backward and upward to its exit from the stylomastoid foramen.

(*f*) The hypoglossal nerve should then be isolated from the tissues covering it. It crosses the external carotid artery just below the point where the occipital artery is given off. The nerve should be exposed by blunt dissection as far posteriorly as possible, to free it from the tissues. This allows the hypoglossal nerve to be brought toward the stump of the divided facial, with which it is to be anastomosed.

(*g*) The facial nerve should then be drawn from the Fallopian canal as far as possible, and severed at the stylomastoid foramen. If it is not thus drawn from the canal it will be too short to allow the anastomosis of the nerves.

J. C. Beck has devised a forceps for seizing the facial nerve as it comes from the styloid foramen. With this instrument it may be withdrawn a half-inch from the canal, which gives sufficient length for union with the hypoglossal nerve.

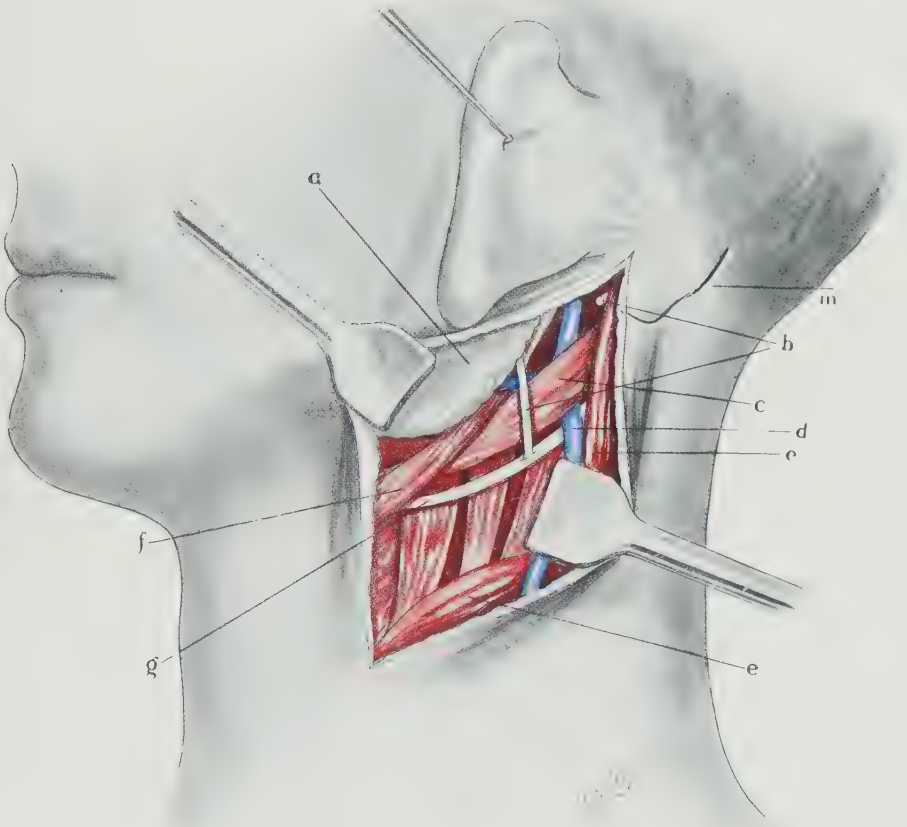
Having severed the facial nerve, the sheath covering its proximal stump should be removed with scissors to expose its axis cylinders (Fig. 531).

(*h*) Make an incision one-eighth inch long in the sheath of the hypoglossal nerve, in as close proximity to the stump of the facial nerve as possible (Plate XXXIII).

(*i*) The nerve fibers should then be separated with fine-pointed dissecting forceps, so that when the bared axis cylinders of the facial stump are inserted into the hypoglossal incision they will be in direct contact with those of the hypoglossal nerve.

(*j*) A fine silk thread with a small round needle on each end should then be passed through the sheath of the facial nerve from without

PLATE XXXIII

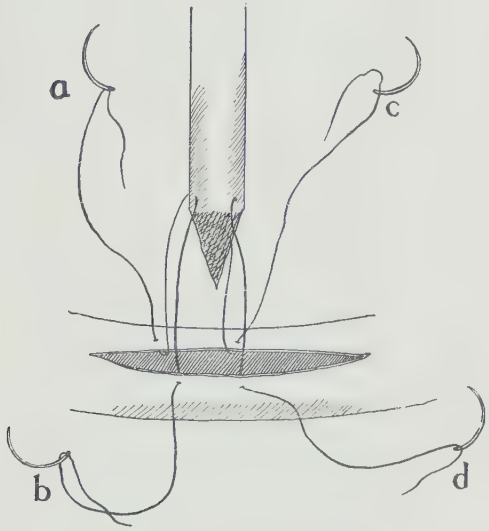


The Anastomosis of the Facial with the Hypoglossal Nerve.

a, the parotid gland; *b*, the stump of the facial and the facial anastomosed with (*g*) the hypoglossal nerve; *c*, the posterior belly of the digastric muscle; *d*, the external jugular vein; *e*, the sternomastoid muscle retracted to expose the hypoglossal nerve; *f*, the stylohyoid muscle; *g*, the hypoglossal nerve; *m*, the mastoid process.

inward, and each needle passed through the sheath of the hypoglossal nerve from within the incision outward. The same procedure is then carried out on the opposite side of the facial nerve, as shown in Fig. 531.

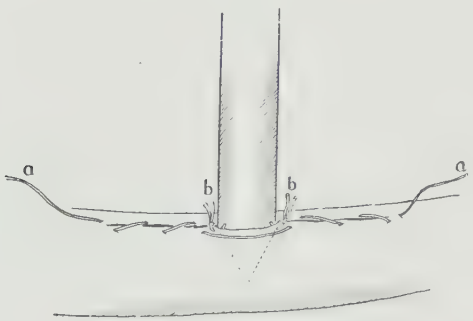
Fig. 531



Schema showing the method of suturing the fascia of the facial with the hypoglossal nerve. *a*, *b* and *c*, *d*, double-needled anchor sutures.

(*k*) The operator and the first assistant each handle one suture, and draw it tight, while the second assistant separates the lips of the incision

Fig. 532



b, *b*, anchor sutures holding the implanted facial nerve in position in the hypoglossal nerve; *a*, *a*, a loose running suture closing the longitudinal incision in the hypoglossal nerve.

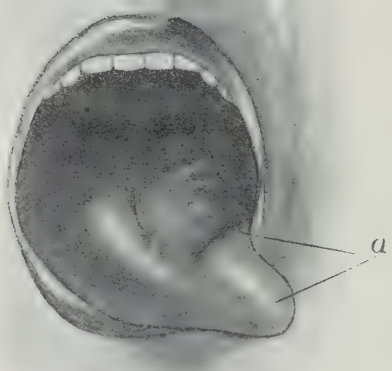
in the hypoglossal nerve, the third assistant guiding the pointed stump of the facial into the hypoglossal incision.

The anchor sutures (Fig. 531) are then tied and the axis cylinders of the two nerves are thus brought into direct contact.

The stump of the facial nerve should be directed toward the proximal end of the hypoglossal nerve so that stimuli from the brain, coming through the hypoglossal, will be more readily transmitted to the facial nerve and carried to the muscles of facial expression.

The sutures should be tied with the greatest care. If too great a number of the axis-cylinder fibers of the hypoglossal are caught in the suture there will be a certain amount of paralysis of the tongue (Fig. 533).

Fig. 533



Partial lingual paralysis shown upon protrusion of the tongue, due to the injury of a few of the fibers of the hypoglossus nerve at the time of the union of the facial and the hypoglossus nerves. *a*, the area paralyzed. (Dr. J. C. Beck's case.)

Too great tension of the hypoglossal nerve will also result in lingual paralysis, hence the necessity of drawing the facial from the Fallopian canal, and dissecting the hypoglossal nerve as far posteriorly as possible, to give it greater freedom of displacement toward the stump of the facial nerve.

(*l*) A secondary continuous suture should then be passed through the lips of the hypoglossal incision, as shown in Fig. 532, *a*, *a*. This suture should not be tied, but drawn tightly.

(*m*) The anastomosed nerves should be covered with a piece of cargile membrane, and the muscles of the neck replaced in their normal posi-

tions. The cargile membrane prevents the formation of scar tissue and adhesions, which would greatly interfere with the success of the operation.

(*n*) The final step of the operation consists in suturing the superficial fascia and skin, drainage being unnecessary, as the operator's field is aseptic.

After-treatment and Observations.—The skin stitches should be removed in from five to seven days, and as soon thereafter as possible massage, electric and tonic remedies should be instituted.

The earliest manifestations of the proper union of the nerves is the appearance of a certain amount of tonicity in the muscles of the paralyzed side of the face. This change is only an indication that anatomical union has occurred, and should not be construed as a beginning of functional activity. On the contrary, it may be weeks, months, or even a few years before functional activity is manifested.

The first sign of functional activity is a slight contraction of the muscles supplied by the lower of the three branches of the pes anserinus, namely, the muscles of the lower lip and the angle of the mouth. At a little later period, the muscles of the upper lip and the forehead show functional activity.

A still later development is the contraction of the facial muscles simultaneously with the act of deglutition. This gradually increases until the contraction on the paralyzed side is greater than on the unaffected side, which is very disagreeable to the patient.

The simultaneous contraction of the facial and hypoglossal muscles is very annoying and confusing. The patient soon learns, however, to disassociate the movements, and is able to swallow with a constantly decreasing degree of facial distortion, until finally the facial muscles remain quiet during the acts of deglutition.

The final and most desirable result is the voluntary contraction of the facial muscles independent of the act of swallowing.

The time required to obtain such a result varies greatly, depending upon the amount of muscle degeneration before the operation, the accurate apposition of the two nerves, and the general condition of the patient.

The reaction of the muscles supplied by the facial nerve should be tested with the electric current in long-standing cases, to determine whether they are still active. If contractions are not produced—that is, if complete atrophy of the muscle is present—it is useless to operate. The contraction of the masseter muscles should not be mistaken for the contraction of the facial muscles. One case of fourteen years' standing was successfully operated.

CHAPTER LIV

NON-SURGICAL DISEASES OF THE LABYRINTH

HYPEREMIA OF THE LABYRINTH

Etiology.—The etiology is generally associated with either congestion of the middle ear or the contents of the cranial cavity. It is rarely primary in the labyrinth. It is usually found in acute suppurative otitis media following scarlet fever, diphtheria, and typhoid fever. It may also be caused by the other exanthematous fevers, pneumonia, encephalitis, mumps, puerperal fever, meningitis, and tumors at the base of the brain. Thrombi in the sinuses contiguous to the petrous portion of the temporal bone and the internal jugular vein, goitre, angioneurotic congestion of the cranial vessels, intracranial affections of the trigeminus, diseases of the medulla oblongata, and the internal use of quinine, salicylic acid, and amyl nitrite may also cause it (Politzer).

Symptoms.—The symptoms are tinnitus, slight feeling of fulness in the head and ears, nausea, vomiting, spontaneous nystagmus to the affected side, and unsteady gait. The nystagmus is due to stimulation of the congested vestibular apparatus and is a typical "sign of stimulation disharmony." In destruction of the labyrinth the nystagmus is to the opposite side, and is a sign of destruction disharmony. The handle of the malleus may be injected, and, when present, denotes a general hyperemia of the organ of hearing. The face and auricle may in rare cases be red. If there is a sense of dazzling whiteness before the eyes, the hyperemia is probably of intracranial origin.

Treatment.—If the hyperemia is secondary to middle-ear inflammation special attention should be addressed to that disease, and with the subsidence of the middle-ear disease the labyrinthine symptoms will disappear. The patient should be put in bed, given laxatives, and have leeches applied to the nape of the neck and mastoid process. He should lie upon his affected ear, as this will cause him to look to the slow component when the eyes are open, and thus relieve the nystagmus and giddiness. If there is active inflammation in the middle ear and mastoid process the ice-bag or Leiter's coil should be applied to the mastoid region for one hour.

If the disease arises from an intracranial lesion, the treatment should be addressed to that condition, the ice-bag applied to the vertex, saline cathartics given, and alcoholic beverages and tobacco prohibited. In general, the habits should be well regulated, constipation prevented, and the beneficial effects of fresh air and sunshine should be taken advantage of by the patient.

ANEMIA OF THE LABYRINTH

Etiology.—The etiology is usually a coexisting general anemia. It may exist, however, as a local condition due to hemorrhage, the obstruction of the internal auditory artery from aneurysm of the basilar artery, neoplasms of the dura or brain extending into the internal auditory canal, embolism of the internal auditory artery, and atheromatous constriction of the internal auditory artery.

Symptoms.—In the angioneurotic and posthemorrhagic forms, the symptoms closely simulate those of seasickness: there is nausea, vomiting, severe tinnitus aurium, deafness, facial pallor, and dizziness. All these symptoms disappear with the return of the blood to the normal state. In the chronic form the tinnitus and deafness are the chief symptoms.

Treatment.—If the labyrinthine anemia is angioneurotic in origin, the neurosis should receive appropriate attention; perhaps a long sea voyage, residence in the mountains or at the seashore, primitive camp life, etc., might be beneficial. If the cause is an excessive hemorrhage, transfusions of normal saline solution should be given, or spontaneous relief may come after a more or less prolonged period of waiting. If it occurs in one who is subject to repeated severe hemorrhages, the duration of the ear symptoms is somewhat prolonged, and means to prevent the recurrences of the hemorrhages should be carefully considered in the treatment. In the angioneurotic type, the internal administration of the bromide of soda and the application of the galvanic current to the sympathetic nerves of the neck are indicated.

HEMORRHAGE INTO THE LABYRINTH

Small hemorrhages into the labyrinth may occur during the course of the exanthematous fevers, on account of the increased blood pressure and the rapid degenerative changes which sometimes characterize the progress of these diseases. The hemorrhages also occur in caisson workers and divers, and in prolonged suffocative seizures. Diabetes, nephritis, and sudden cessation of menstruation may also furnish the cause and atheromatous degeneration of the walls of the arteries predisposes to labyrinthine hemorrhage.

More extensive hemorrhages into the labyrinth occur in fractures of the skull, involving the petrous portion of the temporal bone; from severe contusions of the skull; from extension of carious processes in the temporal bone, and from primary and tuberculous meningitis (Politzer).

Course and Termination.—The course and termination of the hemorrhages into the labyrinth are obviously variable, according to their severity and origin. The blood clot persists in the labyrinth for a variable time, after which they may be absorbed, become organized, or the epi-

thelium, connective tissue, nerve elements, etc., involved by the pressure may become atrophied and degenerated. Politzer reports a case which ended in suppuration.

MÉNIÈRE'S DISEASE

This condition is characterized by sudden and complete loss of hearing, attended with tinnitus, nausea, vomiting, spontaneous nystagmus and vertigo, without a previous history of ear disease. It is supposed to be due to a hemorrhage into the labyrinth. The patient is usually robust, middle aged, and has never previously complained of deafness. At the onset of the attack he sometimes falls unconscious to the ground. In a case seen by the author, the attack came on at night. Upon attempting to rise in the morning he had severe dizziness (indeed, could not walk), nausea, vomiting, tinnitus, spontaneous nystagmus, and complete deafness. The history of the case showed that two years previously the left ear was similarly affected, the hearing remaining almost *nil* in that ear, the right being normal. It is now thirteen years since the last attack, and the hearing is unimproved.

The hearing by bone conduction is lost if the affection is bilateral, and when unilateral the sound of the tuning fork, when placed on the vertex, is lateralized to the unaffected side.

The *course* of Ménière's disease varies. The unconsciousness rapidly disappears, and the vomiting a little more slowly. The dizziness and staggering gait remain for several days. In the author's case the patient had a tendency to walk to the right for four or five days (direction of the slow component of the nystagmus), after the apoplectiform attack in the right ear, he was dazed, and thought slowly for some weeks. His handwriting was not tested. Guye and Politzer report that for a time the handwriting is like that of a tremulous old man. The unsteady gait may persist for years. Relapses usually occur, although there are exceptions to the rule.

Diagnosis. The diagnosis of Ménière's disease can only be made with certainty when the patient is examined immediately after the seizure. If the middle ear, drumhead, and Eustachian tubes are normal and the patient gives the clinical picture just described, and there is no paralysis of other cranial nerves, a diagnosis of Ménière's disease may be made.

This disease should be differentiated from Ménière's symptom complex, which is usually due to an intermittent closure of the Eustachian tubes. The rarefaction of the air in the tympanic cavity retracts the membrana tympani and forces the foot plate of the stapes into the oval window, thus increasing the tension of the labyrinthine fluids and giving rise to the symptoms of Ménière's disease. An examination of the drumheads and Eustachian tubes, however, shows retraction of the one and obstruction of the other. After inflation of the tympanic cavity the symptoms disappear and only return when the air in the tympanum

becomes rarefied. The history of the case shows repeated recurrences of deafness and Ménière's symptom complex.

Prognosis.—The prognosis is unfavorable, little improvement being reported in the cases thus far recorded.

Treatment.—The treatment is directed principally to the relief of the dizziness, nausea, and vomiting. The patient should be placed in bed with the head slightly raised, to avoid the necessity of changing the position in giving food and medicines, as the movements attending these acts increases the disorders present. This precaution should be observed for a few days while the symptoms are annoying. Cold compresses to the head, mustard plasters to the nape of the neck and calves of the legs, and the administration of purgatives may hasten the disappearance of the annoying symptoms. The tinnitus is often relieved by the administration of quinine and the iodide of potash, or, what is probably preferable, iodonucleoid, in which the iodine is united with nucleinic acid, thus rendering it readily digestible and easily and rapidly absorbed, without irritating the stomach. If the quinine causes mental excitement and increased tinnitus, its use should be discontinued (Charcot). It should be given in 2 grain to 5 grain doses three times daily for six or eight weeks. The iodide of potash (or iodonucleoid) may be given for three or four weeks.

To promote absorption of the blood clot and exudate, pilocarpine, in 2 per cent. solution, may be injected 4 to 10 drops daily; or it may be given internally for the same purpose. Its use should not be begun until about the third week, when the acute symptoms have subsided.

MÉNIÈRE'S SYMPTOM COMPLEX

This condition, while similar in its manifestations in many respects to Ménière's disease, should not be confounded with it. Ménière's symptom complex is characterized by dizziness, staggering gait, nausea, tinnitus, and more or less deafness, *with a distinct history of previous deafness and ear disease.* The deafness does not occur suddenly, and is not complete, nor are the profound disturbances found in true Ménière's disease present. The author once saw a case in consultation, in which nearly all the signs of Ménière's disease were present, the exceptions being: (*a*) There was a history of previous deafness and ear disease; (*b*) the deafness did not occur suddenly, nor was it profound; (*c*) inflation of the middle ear through the Eustachian catheter gave immediate and complete relief. The case was one of Eustachian catarrh, complicating a similar process in the epipharynx. The air in the middle ear became gradually rarefied by the absorption of the oxygen by the blood, the drumhead was retracted, and pushed the foot plate of the stapes inward, which compressed the intralabyrinthine fluids, and gave rise to the foregoing phenomena. The same phenomena may be due to chronic catarrhal adhesive processes. According to Politzer, a great majority of the cases are due to a temporary congestion of, or exudation into, the

labyrinth arising in the course of middle-ear infections, which bring about an irritation of the vestibular and ampullar nerves.

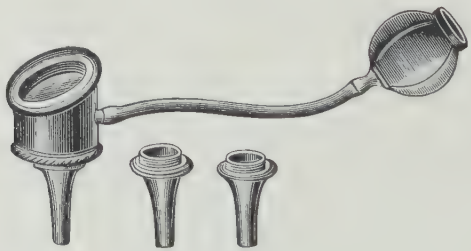
Dr. Geo. E. Shambaugh recently advanced the theory that the tinnitus attending this affection was due to a disturbance of the relation of the membrana tectoria to the hair cells of the organ of Corti. He holds that the membrana tectoria is the resonator of the perception apparatus, whereas, according to Helmholtz, the basilar membrane is the resonator. (See Physiology of the Labyrinth.)

The use of the tuning-forks enables the observer to differentiate between cases of middle-ear origin and those of labyrinthine origin. If with marked diminution of hearing there is positive Rinné, with hearing for low tones preserved, the lesion is in the labyrinth; if, on the contrary, there is a negative Rinné, with loss of hearing for low tones, the lesion is in the conduction portion of the temporal bone, *i. e.*, in the middle ear and Eustachian tube. If the disease is unilateral, the vibrating tuning-fork placed upon the vertex will, if the lesion is in the middle ear or Eustachian tube, lateralize toward the affected side; whereas, if it is in the labyrinth it will lateralize toward the normal or unaffected side.

Some cases reported by Urban Pritchard and Richard Lake were of an epileptiform type, with a tendency to fall toward the affected side. The room seemed to whirl, the face became pale, the eyes dull, the skin covered with cold perspiration, and the pulse small and often retarded.

The course of the symptoms is extremely variable, lasting from a few moments to several days or weeks.

Fig. 534



Siegle's otoscope.

Treatment.—In those cases due to hyperemia of and exudation into the labyrinth, the same treatment recommended under hyperemia of the labyrinth is of value. If the lesion is in the Eustachian tube or middle ear the remedies suited to the condition present should be used. Quinine is perhaps more valuable for the relief of the tinnitus than it is in Ménière's disease. Pneumomassage, especially rarefaction (suction) of the air in the external meatus, in either the middle ear or labyrinthine type, is beneficial in many cases. Its *rationale* in the middle-ear type is in the outward movement of the drumhead, which relieves the pressure upon the foot plate of the stapes, and in the labyrinthine type the lessened pressure in the middle ear relieves the labyrinthine congestion. Rare-

faction can be practised by means of a rubber tube with a metal tip, the patient supplying the suction power with his mouth at the other end of the tube, or Delstanche's rarefacteur or Siegle's otoscope (Fig. 534) may be used with equally good results.

ARTERIOSCLEROSIS OF THE LABYRINTH

According to J. J. Kyle, arteriosclerosis of the labyrinth may be local, or a part of a general sclerosis of the arterial and cellular structures of the body.

Etiology.—"The cause of arteriosclerosis of the labyrinth is the same as in any other part of the body, and may be syphilis, laborious occupation, alcoholism, lead poisoning, infectious fevers, auto-intoxication, vasomotor disease, and heredity."

Syphilis is probably the most important factor in the etiology of the disease in middle life.

The disease may be unilateral or bilateral, and is observed early or late in life.

Pathology.—"The affection probably begins as a structural change in the vasovasorum and is fibrous in character. The labyrinthine artery is the single artery of the labyrinth, and as soon as the nutrition of its wall is disturbed connective-tissue degeneration takes place in the media. Fatty degeneration soon follows in the intima with the deposit of calcareous salts. The vessels may sometimes become narrowed or obliterated.

"As soon as the nutrition of the basilar membrane and organ of Corti is partially or completely cut off, there is atrophy of the sensory auditory cells and connective-tissue proliferation of all the structures. The same change may be observed in the nerve endings of the vestibule and semicircular canals.

"The change in the brain structures varies according to the amount of nutrition carried to the parts. In endarteritis obliterans of the vessels supplying the centre of hearing and equilibration, there is, on account of the slow change in the arterial walls, degeneration and atrophy of the brain cells."

Symptoms.—"The symptoms of arteriosclerosis are both general and local. The general symptoms are increased arterial tension, increased tortuosity and prominence of the arteries of the temple, hypertrophy of the heart, and, if the last is present, there is generally a lowered vitality of the individual, a feeling of age, and tiring, as from overwork, followed by an appearance of aging. Analysis of the urine usually shows increase of the urates and long, thin hyaline casts, undergoing granular degeneration.

"The ear symptoms are unilateral or bilateral tinnitus, slight and progressive deafness, impairment of air and bone conduction, in some cases dizziness early in the disease, and in the later stages hallucinations of hearing may be present. The ear symptoms necessarily vary according to the extent of the sclerosis."

Diagnosis.—"The above symptoms, both general and local, should always direct the physician's attention to the possibility of arteriosclerosis. The early diagnostic symptoms are tinnitus, vertigo, nutritive change in the membrana tympani, and slight unilateral or bilateral deafness. If the general symptoms, as enumerated above, are present the diagnosis is usually complete.

"The location of the lesion, whether in the nuclear or labyrinthine endings of the nerve, may, according to Gradenigo, be shown by the tuning-forks. A diminution in bone conduction and the loss of high tones is indicative of labyrinthine deafness. In central deafness there is a pronounced loss of perception for both high and low tones.

"The disease should not be confounded with Ménière's disease, hyperemia of the auditory nerve, hysterical deafness, hemorrhagic extravasation in the labyrinth from a fall or blow upon the head, or nerve deafness from toxic absorption."

Prognosis.—"The prognosis is usually poor so far as the restoration of hearing or complete cure of the tinnitus is concerned. Under general treatment the symptoms may frequently be relieved and often brought to a standstill."

Treatment. "The treatment of arteriosclerosis of the ear is both general and local, depending somewhat upon the exciting cause. Cases with hereditary predisposing factors do not respond to treatment as well as those due to syphilis or acquired diseases. However, in both conditions, the iodide of potassium in from 2 to 5 grain doses, four or five times daily for long periods of time, is indicated."

LEUKEMIC DEAFNESS

Leukemic deafness is characterized by either sudden and complete deafness and Ménière's symptoms, or by moderate deafness which speedily grows worse, until within a few weeks or months it becomes complete. In acute leukemia the deafness and other ear symptoms occur in the early stage of the disease; whereas in chronic leukemia they usually appear in the later stages. The pathological changes consist of accumulations of lymphocytes, and hemorrhages into the labyrinth, followed by a reactionary inflammation of the endosteum and membranous labyrinth, which finally results in connective-tissue obliteration and partial ossification of the labyrinth (Politzer). The prognosis is obviously unfavorable.

OTITIS INTERNA PAROTITICA

(Diffuse Manifest Suppurative Labyrinthitis)

Mumps being an infectious disease, and the site of infection being anatomically in close proximity to the labyrinth, the infection may be

carried to it by metastasis, or it may be carried through the Glaserian fissure. Symptoms and treatment have been thoroughly discussed in the chapter on Diffuse Manifest Suppurative Labyrinthitis, and hence will not be considered here.

SYPHILIS OF THE INTERNAL EAR; SYPHILITIC OTITIS INTERNA

Syphilitic diseases of the labyrinth usually appear at the end of the secondary or at the beginning of the tertiary stage. Politzer, however, reports a case in which there was labyrinthine involvement seven days after the initial lesion. It may involve the labyrinth in common with the middle ear, or as one of the signs of a general infection, or it may be limited to the internal ear.

Pathology.—The pathology is but little known, as only a few cases have been carefully studied. From the examinations made it appears that there is present thickening of the periosteum of the vestibule (Toynbee, Moos), displacement and fixation of the foot plate of the stapes, small-cell infiltrations and hyperplasia of the connective tissue between the membranous and bony labyrinth; also infiltration of Corti's organ, of the ampullæ, and of the membranous semicircular canals (Moos). The canals and spaces of the labyrinth have also been found filled with new bony tissue. The acoustic nerve may or may not be affected. Adhesive bands, hornification, atrophy and destruction of the ganglionic cells, and syphilitic endarteritis (Baratoux and Virchner) have been reported.

Symptoms.—The symptoms are those of labyrinthine involvement in general, namely, loss of hearing by bone conduction, and for high tones and spontaneous nystagmus in the early acute stage when the vestibular apparatus is destroyed. If the affection is unilateral (rare), the Weber experiment will show lateralization of hearing to the normal side, and Rinné will be decidedly plus upon the affected side. The symptoms may appear suddenly, with tinnitus, deafness, dizziness, nystagmus, and staggering gait. The nystagmus is spontaneous during the acute stage, whereas in the latent period it only appears upon the use of the various tests if the destruction is partial or is negative when the destruction is complete. (See Functional Tests of the Vestibular Apparatus.) The deafness may become complete and permanent, the tinnitus increasing at the same time. The staggering gait and dizziness may disappear after a few days or weeks. Diplacusis and pain in the ear may be present, the pain being due to a periosteal growth in the labyrinth.[1]

Objectively, the signs of syphilis of the internal ear may be wanting. It is only when the middle ear, or Eustachian tube, and labyrinth are simultaneously involved that objective signs are found. There may then be the usual appearance of a catarrhal otitis media, or the characteristic swelling of the mucosa of the Eustachian tube. Syphilitic ozena of the nose and epipharynx may also be present.

[1] Moss and Steinbrugge, Zeits. f. Ohrenh., vol. xiv.

Course.—In most cases the deafness develops gradually for some weeks or months, remains stationary, and then, after a variable interval, suddenly becomes much worse. More rarely the deafness comes on suddenly. Slight exciting causes may bring on a rapid increase in the deafness. Concussions on the head, blows, etc., have been known to do the same thing. In rare cases improvement and recovery take place, and hearing by bone conduction gradually returns.

Diagnosis.—The differential diagnosis between syphilis, otosclerosis, and other forms of labyrinthine disease is not always easy, except when there are evidences of the secondary or tertiary manifestations of syphilis. Unfortunately, in many cases no such obvious signs are present. Politzer observes that "those forms of severe or total deafness which usually develop in both ears during childhood must be regarded as syphilitic affections of the labyrinth. Such cases were formerly supposed to be due to scrofula." The diagnosis of hereditary syphilis is aided by the presence of middle-ear catarrh, purulent otitis media, adhesive processes of the middle ear, and chronic interstitial keratitis (opacity of the cornea). When the syphilitic infection involves the middle ear, and the membrana tympani is perforated and discharges a purulent secretion, the disease is often mistakenly diagnosed as suppurative otitis media. If it persist in severe form syphilitic infection should be suspected and a Wassermann test made. Indeed, the Wassermann test should be made in all suspected cases.

Prognosis.—Recent cases offer a favorable prognosis, while older ones are quite unfavorable. The degree of deafness is not a safe guide in giving a prognosis, as totally deaf cases have been known to recover, while others, with mild deafness, have remained unimproved. General debilitating diseases render the prognosis more grave. The hereditary type, with opacity of the cornea, is unfavorable.

Treatment.—Mercurial injections, with the internal administration of iodonucleoid or iodide of potassium, are indicated. Pilocarpine injections, 4 to 12 drops daily, of a 2 per cent. solution, beginning with 4 drops and increasing to 12 drops, sometimes influences the case favorably (Politzer, Bacon, Gradenigo). The injection of solutions of the iodide of potassium into the middle ear through the Eustachian catheter, as recommended by Politzer, is not to be generally favored. The *technique* of such a procedure gives rise to the extreme liability of carrying infection into the middle ear. Under strict antiseptic precautions and a knowledge of the extremely small size of the tympanic cavity, and the technique of the whole procedure, the danger of infection disappears; and it is possible, though in the author's opinion not probable, that the injection of a solution of the iodide of potassium will affect the course of the disease favorably. The injections of iodoform, iodine vasogen, mercurial ointments etc., are more rational methods of treatment. It should not be forgotten, however, that the disease is essentially a systemic one.

Salvarsan in Syphilis of the Auditory Apparatus.—The use of salvarsan in syphilitic infection of either the sound conduction or the sound perception apparatus has, since Ehrlich introduced it, received both

laudatory and condemnatory criticism. It has been claimed that its use was the cause of neuritis of the auditory and other cranial nerves, and that deafness was often due to it. Indeed, the earlier results following its use seemed to warrant such a conclusion. A careful analysis of the cases in which the auditory nerve was involved has, however, shown rather conclusively that the neuritis and consequent deafness were in all probability due to the faulty preparation of the salvarsan and to the use of insufficiently large doses. At least since the salvarsan has been prepared under strict antiseptic precautions, and has been given in larger doses, the occurrence of neuritis of the auditory nerve, and the deafness resulting therefrom, have been markedly diminished. Nevertheless the question should be submitted to further observation and discussion before an ultimate estimate of the position salvarsan should occupy in the treatment of syphilis and non-syphilitic disease of the ear. In the meantime it is undoubtedly true that salvarsan is a most valuable therapeutic agent in the treatment of syphilis of the auditory apparatus. I will endeavor, therefore, in the following paragraphs of this section to give a summary of the facts of clinical significance, especially as they relate to the preparation and dosage of salvarsan.

Preparatory to the discussion of the use of the salvarsan I will briefly review some of the statistical data and opinions presented by various writers upon this subject.

J. Bernario has, perhaps, made the largest collection of cases of syphilis treated by salvarsan, and has deduced from his critique of the aggregation of cases certain facts of great interest to otologists. In his analysis of 14,000 cases of syphilis treated by salvarsan (606, arsenobenzol) he has formulated the following facts in relation to lesions of the cranial nerves following its administration:

1. Cranial nerve lesion occurred in 126 cases, 118 in the early and secondary stages of syphilis, and 8 in the tertiary stage. In other words, one cranial nerve lesion was present in every 111 cases of the 14,000 treated by salvarsan.

2. Auditory nerve lesion was found in 62 cases, or once in every 226 cases.

3. Of the 62 auditory nerve lesions 11 were accompanied by the involvement of other cranial nerves, especially the optic nerve. In 51 cases the auditory nerve alone was affected, and of these the cochlear branch alone in 29 cases, the vestibular branch alone in 5 cases, and both branches in 17 cases.

4. Of the 126 cases of cranial nerve involvement the lesion occurred in 96 per cent. of the cases within the first four months after the injections of salvarsan. Of the 96 per cent., 40 per cent. occurred during the second month.

As the nerve lesions followed the use of the salvarsan within a relatively short time it was at first thought they were due to the irritation of the arsenic in the salvarsan. Subsequent observations appear to have shown this hypothesis to have been wrong. Ehrlich explained

the occurrence of cranial nerve lesions as being due to insufficient dosage and the improper preparation of the salvarsan. He claimed that the spirochætæ within the sheath of the auditory and other cranial nerves were protected by their dense fibrous envelopes, thus preserving the integrity of the specific germs, whereas they were destroyed in other parts of the body. After the lapse of from two to four months the spirochætæ became actively destructive to the nerves protecting them, and deafness, blindness, etc., followed. While this theory does not adequately explain the clinical phenomena, it at least has the merit of explaining some of them. His suggestion that larger doses of salvarsan were required to destroy the spirochætæ within the sheaths of the cranial nerves than elsewhere in the body has been sustained by subsequent experience. His theory seems to convey the idea that insufficient dosage of salvarsan has a tendency to augment the virility of the spirochætæ protected in the sheaths of the cranial nerves in a percentage of cases, whereas this is probably not true.

Alexander, on the other hand, believes that the cranial nerve lesions are due to the action of the salvarsan and not to the spirochætæ, as claimed by Ehrlich. He says that (*a*) salvarsan is a dangerous remedy in the acute stages of all forms of labyrinth disease whether syphilitic or non-syphilitic; (*b*) in acute exacerbations of subacute labyrinth disease it is also a dangerous remedy, whereas (*c*) in chronic disease of the labyrinth and auditory nerve it is a safe and valuable remedy.

Brenario, however, has shown that in 10 cases of auditory nerve lesion (attended by deafness) following the mercurial treatment, 8 were relieved by injections of salvarsan, 2 of them partially, 6 completely, while the remaining 2 cases were not favorably influenced. It has also been demonstrated that cranial nerve involvement has been markedly diminished since larger doses of salvarsan have been administered, and since greater care has been exercised in its preparation.

Faulty technique of preparation and administration of salvarsan seem, therefore, to be the chief determining factors in the causation of cranial nerve lesions. It may, therefore, be given with safety in the acute stages of labyrinthine disease, provided a fresh sterile solution of salvarsan is employed in sufficiently large quantity to destroy the spirochætæ within the sheaths of the auditory, optic, and other cranial nerves.

INJURIES TO THE LABYRINTH; CONCUSSION OF THE LABYRINTH

Etiology.—The injury may be due to *direct* or to *indirect violence*, more commonly the latter. The violence may be transmitted through the bones of the head to the internal ear, or through the air and ossicles in the middle-ear cavity, when there is a *sudden condensation* of the atmosphere by a great explosion, or a *blow of the hand* over the ear. The bony capsule may be injured while the membranous labyrinth is unharmed, and *vice versa*. When a *fissure* of the skull passes through the labyrinth it usually extends to the middle ear and external auditory

meatus, hence the leakage of cerebrospinal fluid into the middle ear from which it escapes through the Eustachian tube or the ruptured membrana tympani. Great *violence* may produce pronounced aural disturbances without fracture of the bone. In these cases it is probable that the terminal nerve filaments of the labyrinth are irritated, and that small hemorrhages occur in the labyrinth.

Injuries to the labyrinth from powerful *compression of the atmosphere* by explosions, boxing the ears, etc., may or may not cause rupture of the drumhead. Should the drumhead rupture, however, the labyrinth is probably saved from some of the force of the concussion, as the air in the middle ear escapes through the rupture, thus relieving the tension which would otherwise expend itself upon the foot plate of the stapes in the oval window.

Detonations from heavy ordnance, or loud reports of guns in shooting galleries, produce a great deal of harm to the terminal nerve filaments of the labyrinth by irritation, and result in more or less deafness and tinnitus (Sexton).

Symptoms.—The symptoms vary with the severity of the concussion and the location and character of the lesion. If the concussion is powerful the individual may drop to the ground as though shot, and remain in an unconscious condition for several hours, after which consciousness returns, and he finds himself to be entirely deaf. Or, if the concussion is light, he may stagger, but not fall, and be stupid or dazed for a short time, with more or less tinnitus and deafness. There may also be nausea and vomiting, with more or less giddiness and nystagmus. (See Chapter XXXIII.) If the blow or concussion causes fracture through the cochlea or semicircular canals sudden and total deafness on the affected side, a staggering gait, and nystagmus will be the prominent symptoms. The nystagmus gradually subsides and altogether disappears in a few days or weeks.

The hearing for high tones is lost. Diplacusis and hyperesthesia acoustica are sometimes present. The sensibility of the skin of the auricle and meatus may be diminished.

According to Politzer, "a medicolegal decision as to the existence of concussion of the labyrinth can be given only in those cases in which there is a fissure of the temporal bone extending to the external meatus, and in which an injury of the labyrinth may be inferred, either from the discharge of cerebrospinal fluid or from complete deafness and the absence of perception through the cranial bones." In the cases due to compression of air in the external meatus no opinion can be given (Politzer). It should be said, however, that since the functional tests of the vestibular apparatus have been formulated, an opinion of some value is possible. (See Functional Tests of the Vestibular Apparatus.)

It may be of medicolegal importance to establish the degree of impairment of hearing, as the patient may seek redress in the courts. If he does so he will sometimes be tempted to magnify his auditory disability. By the use of a series of tuning-forks, whistles, and other functional tests of hearing a correct diagnosis may be made. It will also be

necessary to establish as nearly as possible the condition of his hearing apparatus before the injury. Lateralization of the sound in Weber's experiment to the injured ear signifies that the labyrinth is unaffected, whereas lateralization toward the sound ear is strongly suggestive of labyrinthine involvement in the injured ear. The loss of high tones in the affected ear also points to labyrinthine disease or injury. It is also necessary to prove or disprove the presence of labyrinthine disease before the date of the injury. This is not often easy to do. The Rinné test is of little value when there is complete deafness, but may prove of some value when there is only partial deafness. (See Barany's Test.)

Treatment.—Rest in bed consitutes the whole of the treatment in most cases, whether there is simple concussion or fracture through the labyrinth. Pain in the ear may be controlled with leeches applied to the mastoid region. Tinnitus of an aggravating character may be relieved by the administration of bromide of soda. After the acute symptoms have subsided iodonucleoid or iodide of potassium should be administered to hasten the absorption of the inflammatory exudate.

OCCUPATION DEAFNESS

For many years it has been recognized that among those who have been engaged in certain occupations for a long time, especially where continuous or frequently recurring sounds are heard, there is apt to be more or less deafness. The terminal nerve filaments of the labyrinth are continuously subjected to irritation, and undergo a degenerative change often amounting to complete atrophy, and consequent deafness. Occupation deafness has been observed among blacksmiths, locksmiths, telephone operators, boilermakers, certain machine-shop workers, weavers, and railroad employees. Among this class of workers it is probable that the continuous noise to which their ears are subjected causes an irritation of the acoustic nervous apparatus of the labyrinth and to the circulatory apparatus as well, which after a long time causes a disturbance of the nutrition of the parts, and finally leads to degeneration, atrophy, and paralysis. Both ears are usually affected.

There are other conditions, peculiar to certain occupations, which cause dulness of hearing, as exposure to damp, cold atmosphere, dust, and superheated air. Stokers and engineers are particularly exposed to atmospheric changes, heat, cold, dust, and noxious vapors. They are, therefore, subject to nasal and epipharyngeal catarrh, and its extension to the Eustachian tube and middle ear. Many, after from five to ten years' service on railroads, have well-marked dulness of hearing. Numerous observers have written on the subject, and their conclusions are as follows: (*a*) The deafness and tinnitus may be due to the constant vibratory movement of the locomotive, resulting in irritation to the terminal nerve filaments of the labyrinth; (*b*) constant straining of the ears to hear above the noise and roar of the train, is thought by some to

be a cause; (*c*) cold draughts of air and the heat from the furnace cause epipharyngeal and aural catarrh; and (*d*) the inhalation of the noxious gases and vapors cause irritation and catarrhal inflammation of the nose, pharynx, and middle ear.

The chief symptom of the catarrhal cases of occupation deafness are more or less dulness of hearing, tinnitus, and in some cases giddiness. Rinné may be positive or negative according to the degree of deafness present. Hearing by bone conduction is increased. If the labyrinth is also involved the tests are somewhat confused, especially as to the relative length of air and bone conduction, both of which are diminished. If there is also loss of hearing for high tones, the labyrinth may be safely said to be affected.

SIMULATED DEAFNESS

Various motives lead to simulation of ear disease. Hysterical individuals sometimes do it to excite attention or sympathy. Soldiers in the army and men drafted to fill the ranks, who desire to avoid duty, and those injured on railways, streets, and in shops, who wish to collect damages through the courts, sometimes exaggerate or assume deafness or artificially produce ear disease.

Tests for Simulated Deafness.—(*a*) First make a careful *objective examination* of the external ear, external auditory meatus, drumhead, and the Eustachian tube. It is a significant fact that in the army most cases of suspected simulated deafness are unilateral. This arises from the fact that a double deafness would have previously attracted attention, whereas a one-sided deafness might have existed without being discovered. In other words, it is easier to simulate one-sided deafness, hence its greater frequency among malingerers. The malingerer often artificially produces an obvious cause for the deafness he wishes to assume by dropping strong solutions of silver nitrate, carbolic acid, creosote, tincture of cantharides, etc., into the meatus. The skin and drumhead are thus cauterized and simulate in some degree suppurative otitis media. A careful examination will usually reveal the source of the inflammation. If silver is used, a dark brown stain will be seen; whereas if carbolic acid is used, the bleached skin will aid in arriving at a correct conclusion. A bandage placed over the ear and sealed, will in these cases lead to a speedy recovery, as the malingerer is unable to continue the caustic applications. Foreign bodies placed in the meatus to simulate deafness and ear disease may be detected by a careful examination.

(*b*) It is in cases in which there are no objective signs of ear disease that the real difficulty of detecting malingering arises. The would-be patient often studies the subjective signs of labyrinthine deafness so well that, if he is especially shrewd, it is well-nigh impossible to detect him. In making the examination of this class of cases the eyes of the suspect must be bandaged, thus rendering it somewhat difficult for him to judge distances in testing with the voice, acoumeter, or watch.

If he hears the instrument at greatly varying distances with the deaf ear (the other being tightly plugged) it is fair to presume he is malingering. If, on the other hand, during repeated short testings, he hears at about the same distance, it is fair to presume that he is really deaf.

(*c*) **Erhard's Test.**—When a normal ear is tightly closed a loud ticking watch (the Ingersoll watch) may be heard at three or four feet. The patient should have the supposed deaf ear tightly closed, and when the watch is within three or four feet of the normal ear, he should be commanded to count the beats, which he will, of course, readily do. The sound ear should then be closed, the supposed deaf one being open, and the same test made on the open deaf ear. If when the watch is within two or three feet of the ear he says he does not hear it, it is fair to presume that he is simulating the deafness, as at that distance he would hear the watch with the closed normal ear.

(*d*) **Chimani-Moos Test.**—In one-sided deafness a large vibrating c_2 fork is alternately held at an equal distance from each ear, until the suspected malingerer makes it plain to himself that he hears the fork loudest before the normal ear. The vibrating fork is then placed on the vertex, bridge of the nose, or median line of the incisor teeth, and the patient is asked in which ear he hears the fork the plainer. A patient with true unilateral middle-ear disease will, without hesitation, say that he hears it louder on the affected side; whereas a malingerer will hesitate, as he hears it equally well on both sides, or he may say he does not hear the fork at all in the suspected ear. The normal ear should now be tightly closed and the vibrating fork again placed on the median line of the skull, and the malingerer will probably say he does not hear it at all, or but faintly; whereas in true one-sided deafness the patient will say he hears the tone louder in the affected side. This only applies to disease, or simulated disease, of the middle ear. If disease of the labyrinth is being simulated, the problem becomes more difficult.

(*e*) A common stethoscope, having one tube closed with a wooden plug, may be used to detect simulated unilateral deafness. The stethoscope should be adjusted to the patient's ears, the open tube leading to the suspected ear, the closed one to the normal ear. The physician should now speak into the bell of the stethoscope, having the patient repeat what he hears. The instrument should then be removed, the normal ear tightly closed, and the same formula repeated to the patient. He will say he cannot hear, whereas he has already repeated after you, with the normal ear tightly closed with the plugged arm of the stethoscope. In other words, he heard with his suspected ear through the open tube of the stethoscope (the one leading to the normal ear being tightly closed), thinking, of course, that he would lead the examiner to believe he heard with the normal ear.

(*f*) The use of four ear specula, two open and two half filled with wax, may be used to detect malingering. The patient should sit with bandaged eyes facing the wall. The two open specula should be simultaneously introduced, one in each ear, and the examiner (behind the patient) should repeat certain words, or numerals, at varying distances, and

thus ascertain his hearing distance with both ears open. He should then change the specula, using one open and one closed, then two open, then two closed, and so on, noting the distances he hears with the varying combinations of the specula. In this way the patient will unwittingly reveal the true condition of his hearing apparatus.

Repeated examinations and the striking contradictions made by the malingerer during the various examinations will lead to a correct diagnosis in most cases.

Barany's Test.—Barany's noise apparatus may be used to detect malingering in one-sided deafness. The patient reads some selected paragraph or article aloud. So long as he hears his own voice it does not change in pitch or articulation. The noise apparatus is then applied to his sound ear while he continues reading. If he is actually deaf in the so-called affected ear his voice will become elevated in pitch and the articulation blurred. If he hears with that ear his voice will remain unaffected. This test may be made experimentally upon normal individuals by using two Barany apparatuses. At the beginning of the reading one is applied to the right ear. After a few sentences are read the other is applied to the left ear, thus rendering the patient totally deaf. His voice and articulation will be greatly modified.

PARESES AND PARALYSES

Angioneurotic Paralysis of the Auditory Nerve.—This is probably a rare affection, or, at least, it has been rarely recognized and described. It is characterized by a transitory facial pallor, nausea, dizziness, tinnitus, and deafness. The attack lasts but for a few minutes, and when it disappears, the hearing is perfectly normal. The attacks may occur at frequent intervals.

The treatment consists in the administration of sedatives, tonics, and the application of galvanism over the cervical sympathetics, which have an intimate anatomical connection with the terminal nerve endings in the labyrinth.

Rheumatic Paralysis of the Auditory Nerve.—This is an obscure affection and difficult to diagnosticate. The diagnosis must largely depend upon the history of rheumatism elsewhere in the body, and upon the involvement of other cranial nerves. It may, however, in rare instances involve the auditory nerve alone. Bing reports a case limited to the auditory nerve and the clinical picture was as follows: (*a*) Female, aged forty-seven years, exposed to a draught. (*b*) Complete deafness, and tinnitus in the right ear, the left being less affected. (*c*) Weber lateralized to the left ear. (*d*) Inflation of the middle ear did not increase the hearing distance. (*e*) The case ended in recovery in eight days from the internal administration of the iodide of potassium and the application of vesicants to the mastoid region. It should be remarked that in these cases there is an absence of the objective signs of middle-ear disease.

Symptoms.—The symptoms are those given above, with the addition of the history of rheumatism elsewhere in the body, the involvement of the facial or other cranial nerves, and the signs of labyrinthine involvement, as lessened, or loss of bone conduction. If the vestibular portion of the labyrinth is affected, there will be dizziness or a staggering gait and spontaneous nystagmus; whereas if the lesion is limited to the cochlear portion of the labyrinth, deafness and tinnitus will be the chief symptoms.

Hysterical Paralysis of the Auditory Nerve.—This form of ear disease is usually unilateral, and is characterized by unilateral deafness, with tactile hyperesthesia, hyposmia, contracted field of vision, and diminished sensibility of the skin on the affected side. The Eustachian tube, drumhead, external meatus, and auricle are occasionally hyperesthetic on the affected side. Weber experiment: tone lateralizes to the normal ear, bone conduction being diminished on the side of the paralysis. Whispered speech can often be heard at six or eight feet, while the tuning-fork may not be heard at all. This is considered by Hammerschlag as characteristic of hysterical paralysis. The same observer calls attention to the fact that a tuning-fork vibrating at its greatest intensity before the affected ear ceases to be heard, and then after a few seconds is heard again. This, he explains, is due to fatigue of the auditory nerve, which after a few moments' rest perceives the sound again (Politzer).

Slight aural lesions in hysterical individuals may give rise to marked disturbance of hearing. Tinnitus and dizziness, however, are signs of organic labyrinthine disease. In hysterical deafness the degree of deafness varies greatly at different times.

Treatment.—The treatment of hysterical deafness should embrace the relief of any middle ear disease found, no matter how slight in character, as great improvement, all out of proportion to the apparent lesion, often follows. The nervous and general systems should be built up by tonic and sedative remedies, outdoor life, bathing, etc. The iodonucleoid or the iodide of potash should be given in 3 to 6 grain doses three times daily. Galvanism of the ear and sympathetic system of the neck may also be used to some advantage.

NEUROSES OF THE AUDITORY APPARATUS; HYPERESTHESIA

1. **Hyperacuteness of Hearing.**—Oxyecoia is a rare form of hyperesthesia and is characterized by a temporary ability to hear music, or at least certain tones, at a much greater distance than others do with normal hearing. It is usually caused by alcoholic and tobacco poisoning, and is specially prone to occur in hysterical and neurasthenic persons.

2. **Paracusis.**—Paracusis may be due to a disorder of the nervous apparatus, the labyrinth, or to a disturbed tension of the drum-head and ossicles of the middle ear. In this condition there is a false interpretation of the pitch of a tone, often amounting to $\frac{1}{4}$ or $\frac{1}{2}$ interval.

Paracusis duplex, or diplacusis, is a variety of disturbed perception of pitch, and is characterized by the hearing of two tones for every sound produced, or in certain cases only for certain tones. It is due to certain unknown influences in the course of acute otitis media, serous middle-ear catarrh, chronic suppurative otitis media, and hyperostosis of the bony capsule of the labyrinth.

Paracusis Willisii is characterized by the ability to hear better in a noisy place, as on a railway train, street car, or in a machine shop. Its etiology is still a mooted question, although it is commonly present in sclerosis of the middle ear and in hyperostosis or spongifying of the bony capsule of the labyrinth. Some hold that the improved hearing in the presence of noise is due to the increased excitability of the terminal nerve filaments of the labyrinth, while others hold that it is due to the mechanical vibration of the bone and secondarily of the terminal nerve filaments, which increases their auditory power. Still others advance the theory that it is due to a shaking and loosening of the ossicles of the middle ear.

George McBean advances the theory that, owing to the large size of the membrana tympani as compared to foot-plate of the stapes, all vibrations of wide amplitude in the air are transmitted to the endolymph with greatly increased force though lessened amplitude. This force is spent on the membrane of the round window (which is five times more movable than the stapedial foot-plate, according to Politzer) and also in the ductus endolymphaticus.

As air vibrations are practically ever present, mass motions in the endolymph are also ever present, and the normal relation of the tectorial membrane to the hair cells of the organ of Corti must be in a moving liquid.

In otosclerosis the bony fixation of the stapes in the oval window prevents these mass motions in the endolymph, so that the fluid is comparatively at rest and the tectorial membrane becomes changed in its normal relation to the hair cells. (See Shambaugh's Theory of Sound Perception.)

In the presence of any heavy vibrations, as in riding on the cars, the endolymph is thrown into motion, which is permitted by the elastic membrane of the round window, so that artificially a mass motion is produced similar to the normal motion of the endolymph and the nerve terminations become more capable of responding to the normal stimuli of molecular vibrations.

If the round window becomes involved in the sclerotic process all mass motion must cease and paracusis Willisii would be absent.

The vibration of the cranial bones and the attending stimulation of the nervous apparatus and fluid contents of the labyrinth and cerebrospinal spaces seem to the author to be the most rational explanation. We know from personal observation that mechanical vibration applied to the spinal column and the head improves the hearing in some cases. Whether this is due to a stimulation of the nutritional centres or to a stimulation of the nervous apparatus of the labyrinth is still an open

question. We know also from personal observation that if these patients are placed in bed and given passive exercise (massage) and wholesome food for a few weeks their hearing will improve.

3. **Hyperesthesia Acoustica.**—This condition is characterized by a disagreeable sensation when musical tones or sounds are heard. The condition is usually present in anemic and hysterical individuals, and in those convalescent from severe illness. It may be present in certain forms of neuroses, as hemicrania and trigeminal neuralgia. It is also one of the manifestations attending the administration of quinine and salicylic acid.

4. **Tinnitus Aurium, or Subjective Noises.**—This is one of the commonest ear symptoms, and has been repeatedly referred to in this work in the descriptions of numerous ear diseases. Its exact etiology is obscure in spite of the large amount of literature on the subject. Various theories have been advanced, explaining its cause, the one by Shambaugh being the most lucid and satisfactory.

He advances the interesting and ingenious theory that: "In the first place, the character of tinnitus aurium is usually that of an indefinite sound, like the wind in the forest or the rushing of water, sounds made up of a great complexity of tones and with no definite pitch. Clinically, these subjective sounds arise from a variety of pathological conditions. One of the best known causes of tinnitus is pressure applied to the conducting apparatus, so as to push the foot plate of the stapes into the oval window. This results in tinnitus aurium of the indefinite character described above. What actually takes place when the stapes is thus forced into the oval window is an increase in the tension of the intralabyrinthine fluid. The result of this alteration in tension must be a disturbance of the membrana tectoria (see Anatomy and Physiology of the Labyrinth), which has apparently the same specific gravity as the endolymph when the latter is under normal pressure. The hairs from the hair cells, as have been shown, normally penetrate into the lower surface of the membrana tectoria. Any disturbance in this membrane, however slight, would, therefore, alter the normal relations existing between the membrane and the hair cells. It seems that such an alteration from the normal relation between the membrana tectoria and the hairs of the hair cells would constitute a stimulation of these cells. When the foot plate of the stapes is pushed into the oval window there would result a slight stimulation of perhaps all the hair cells in the cochlea. The result would be exactly what we meet with clinically, a tinnitus aurium of an indefinite character, like the wind in the forest or the roar of a sea-shell. When a sudden increase or decrease in the blood pressure results in tinnitus aurium, the cause is the same as when the stapes is pushed into the oval window. The explanation of the increase or decrease of the intralabyrinthine pressure is here quite evident. The tinnitus aurium arising from the administration of certain drugs is also plausibly explained in the same way as due to an alteration in the blood supply to the labyrinth with the resulting alteration in the presence of the intralabyrinthine fluid. The tinnitus occurring in Ménière's disease, where there has

been an escape of blood into the cochlea, is also similarly accounted for by this conception of the physiology of tone perception. The disturbances in the function of hearing arising from an injury produced by a shrill whistle, or an explosion near the ear, are also readily explained. In the first place, when a permanent disturbance in hearing is thus produced, it can be readily accounted for by a partial severance of the relation between the membrana tectoria and hair cells, so that the hairs from a greater or smaller number of these cells project free in the endolymph and do not come in contact with the membrana tectoria, and therefore cannot receive the stimulation from impulses passing through the endolymph. On the other hand, when there results from such an injury a permanent tinnitus aurium, this is explained by a partial, not complete, severance of the membrana tectoria from the hair cells over a certain area. This alteration of the relation existing normally between the hair cells and membrana tectoria may result, as we have repeatedly pointed out, in a stimulation of these cells. This explanation appears all the more rational since the pitch of the tinnitus is often approximately that of the whistle which originally produced the injury."

The external conditions which influence tinnitus are those which influence catarrhal diseases of the upper respiratory tract, namely, sudden changes in the weather and temperature, living in damp places, improper clothing, etc. Bodily conditions, as fatigue, exhaustion from heat or undue exposure to inclement weather, and bodily depression from overmental application, also aggravate the subjective noises.

The *character* of the noises is as various as noises themselves, the usual form being a singing, whistling, chirping, popping, crackling sound, or like the noise of a railway train in the distance. Many other noises are described by patients. They may be intermittent or continuous. The remissions usually occur while the patient's mind is engrossed with other matters, hence they are less troublesome in the daytime. Some patients are so distressed by the noises that they are driven to desperate measures, even to suicide.

In some cases the noises increase in proportion to the deafness, in others they cease with marked deafness, while in still others they continue to increase after the deafness is absolute. They may appear in persons who are not deaf, but who are nervous, or exhausted from overmental or physical exertion, or from grief.

THE HEARING OF VOICES AND MUSIC

Human voices and musical melodies are sometimes heard by persons who have some affection of the cortex of the brain, though rarely or never by subjects with an uncomplicated ear disease. An existing ear disease may aggravate the condition in the cortex of the brain; hence the cure of the ear disease is often attended by an improvement of the hallucinations. Some persons hear musical melodies repeated over and over which prove very annoying. The subjective hearing of human

voices is more serious, and often the forerunner of melancholia or progressive paralysis.

Prognosis.—The *prognosis and also the treatment of tinnitus* is embraced in the various diseases in which it occurs as a symptom. It may be said in general, however, to be comparatively good in cases of simple middle-ear and tubal catarrh, and generally unfavorable in hyperostosis and labyrinthine diseases, in noises of cerebral origin, and where the arterial noises have existed for a long time. Paracusis Willisii is usually taken to indicate a well-marked adhesive process in the middle ear or in hyperostosis of the bony capsule of the labyrinth, and the prognosis is unfavorable except when suitable remedial measures are used early. In cases in which human voices and musical melodies are heard the prognosis is very grave, except in rare cases in which the relief of the noises follows the cure of the middle-ear disease.

Treatment.—The treatment of subjective noises is as broad as the subject of ear and brain diseases, hence it will not be given further consideration.

WORD-DEAFNESS OR SENSORY APHASIA

This form of deafness is characterized by the ability to hear, with the loss of the power to distinguish words, and is thought to be due to a lesion of the cortex in a portion of the middle convolution of the left temporal lobe, or in the left gyrus of that lobe. It may be questioned, however, whether the auditory (acoustic) centre is so restricted in its distribution. When present, it is generally due to an encephalitis, an exudate following a hemorrhagic pachymeningitis, brain tubercle, or to an embolic softening of the brain.

Types of Word-deafness.—(*a*) *Amnesic aphasia* is characterized by the loss of memory for things, or by the application of wrong names to objects. (*b*) *Monophasia* consists in the naming of all objects to which the attention is directed by the same name. (*c*) *Amnesic agraphia* is the inability to write words that are spoken, or the names of surrounding objects, and (*d*) the inability to repeat what is heard and understood. (*e*) *Amusia* is a term introduced by Knoblauch to indicate deafness for musical tones. It occurs more frequently than word-deafness, and is probably due to a lesion of the first and second convolutions of the left temporal lobe in right-handed persons. Word-deafness and tone-deafness may exist at the same time. In tone-deafness the amusia varies in degree from absolute loss of hearing for musical tones to false interpretations of them.

DEFECTS OF HEARING DUE TO INTRACRANIAL TUMORS

Brain tumors, especially of basilar origin, may give rise to disturbances of hearing by pressure upon, or stretching of, the auditory nerve

fibers, and by causing an ascending neuritis of the auditory nerve. A lymph stasis at the origin of the auditory nerve may also cause aural disturbances (Gradenigo). This condition is similar to that which occurs in the optic papilla during an increase of intracranial pressure.

Symptoms.—The symptoms are unilateral tinnitus aurium, deafness, more or less complete, and dizziness. If the tumor involves the vestibular nerve, nystagmus to the opposite side will be produced. (See Chapter XXXIII.) Other symptoms not expressed through the auditory apparatus are a feeling of tightness in the head, glimmering or dull vision, pain or full feeling on the side of the head corresponding to the location of the tumor, slow pulse, choked disk, and motor and sensory paralyses over the areas supplied by the other cranial nerves, which are also usually more or less involved.

Diagnosis.—The diagnosis must be made chiefly by the disturbances arising through the lesions of the other cranial nerves, as the aural symptoms are not characteristic of this form of ear disease. An early diagnosis, therefore, cannot often be made. Facial paralysis and retained perception for the tuning-forks, watch, and acoumeter through the cranial bones, together with dizziness, tinnitus, and deafness, are significant symptoms. The perception of the forks, watch, etc., through the cranial bones exclude labyrinthine disease, even of a mild type. In some cases the perception for high tones often remains unaffected, and in others it is diminished. The age of the patient should be taken into account in connection with the tests of bone conduction and the hearing for high tones. If the patient is more than fifty years old there is a physiological diminution in the perception by bone conduction, as well as a restriction of the upper limit of hearing. (See Functional Tests of the Auditory (Cochlear) Apparatus.) Hence, in a case with the above aural symptoms, in which there is a suspicion of brain tumor, the presence of a slight diminution of hearing by bone conduction and the loss of hearing for the higher tones would not necessarily lead to the conclusion that the labyrinth was affected by a brain tumor. As first stated, the chief diagnostic guide is the pareses or paralyses of the other cranial nerves, the facial nerve usually affording the most direct and certain information. A slight paresis and anesthesia of the skin over the area of nerve distribution, when found in conjunction with deafness, tinnitus, and dizziness, usually points strongly to an ear disturbance having its origin in tumor of the brain.

NEOPLASMS OF THE INTERNAL EAR

Newgrowths in the internal ear may be primary (rare) or secondary. Primary growths at the root of the acoustic (auditory) nerve have been reported, but nearly all accurately reported cases have been secondary. Epitheliomata and malignant round-cell sarcomata may extend from the middle ear to the labyrinth, and destroy the cochlea, vestibule, or even the whole of the petrous portion of the temporal bone. Neuromata

of the auditory nerve have also been observed. Cavernous angiomata of the petrous portion of the temporal bone have been reported by Politzer but are extremely rare.

The symptoms vary with the location and size of the growths, and are deafness, tinnitus, dizziness, staggering gait, nausea, nystagmus and vomiting, together with other extraneous symptoms due to lesions of the other cranial nerves.

LOCOMOTOR ATAXIA DEAFNESS

Disturbances of hearing occurring in the course of locomotor ataxia are due to atrophy of the auditory nerve. The atrophy may affect the nervous apparatus anywhere from its cortical origin to its distribution in the labyrinth. According to various statistical reports, the hearing is affected in tabes dorsalis in from 1 to 80 per cent. of the cases recorded. The aural symptoms usually develop gradually. The tinnitus is always present and almost unbearable. The affection is usually bilateral, and dizziness is present in about 65 per cent. of the cases. The author recently examined a case in which there was deafness, intolerable tinnitus, and dizziness. The bone conduction and upper range of hearing were diminished, but not more than the age of the patient (sixty-five years) would account for. Rotating the head on its various axes with the eyes closed did not increase the dizziness or produce nystagmus. The appearance of the drumheads was normal. The hearing for low, deep-toned tuning-forks was normal, Rinné negative, and both ears were affected.

CHAPTER LV

DEAF-MUTISM

HOLGER MYGIND'S elaborate and classical treatise on deaf-mutism opens with the following paragraph:

Definition.—"*Deaf-mutism*, strictly speaking, signifies the abnormality which is characterized by the co-existence of deafness and dumbness. Various circumstances necessitate, however, a more limited definition. Deaf-mutism may, therefore, be defined as a pathological condition dependent upon an anomaly of the auditory organs, either congenital or acquired, in early childhood, causing so considerable a diminution of the power of hearing as to prevent the acquisition of speech; or, should speech have been acquired before the occurrence of the loss of hearing, it is preserved by the aid of hearing alone. Individuals exhibiting this pathological condition are described as *deaf-mutes*, even when speech has been acquired by a special system of instruction."

The foregoing definition will be observed in the consideration of this subject.

Historical.—It is interesting to know, as Mygind has shown, that deaf-mutism has been referred to in literature from the time Exodus (fourth chapter and second verse) was written. Herodotus, Hippocrates, Aristotle, Pliny, Gellius and others of the ancient period refer to it; and in the Middle Ages, Cananus, Pedro de Ponce, Andreas Laurentius, and Zachias.

A gradual change of opinion as to the relationship between hearing and speech took place. In the ancient period the idea prevailed that it was due to the inability to use the tongue (Hippocrates and Aristotle). Later, Pliny said, "The man who is born without the power of hearing is also deprived of the power of speech, and none are born deaf who are not also dumb."

During the Middle Ages the influence of Aristotle's writings was so potent that little progress, beyond the opinion expressed by him, was made. Cardanus, 1501 to 1576, first distinctly stated the true relationship, *i. e.*, that deafness is the principal and primary cause of deaf-mutism.

During the last century, the subject was placed upon a scientific basis, chiefly through the writings of Itard, Schmalz, Wild, Meissner, Toynbee, von Tröltsch, A. Hartman, Lemcke, and Mygind.

It is true that institutional work and statistical bureaus have aided very materially in the evolution of the subject. The classical work of Mygind probably represents the most advanced and correct statement on the subject that has been given, and it is chiefly from his work that

the author gleans the data for this chapter. Direct reference is also made to the works of von Tröltsch and Toynbee.

Classification.—Deaf-mutes may be best classified according to the degree of deafness as:

(*a*) *True deaf-mutes*, or those who are totally deaf to speech, and must depend entirely on the other senses to acquire its use.

(*b*) *Semi-deaf-mutes*, or those who have slight power of hearing, or who retain slight speech acquired before deafness supervened.

Some confuse those who, for other reasons than deafness, have lost the power of speech with deaf-mutism. *It should, therefore, be distinctly understood, without question, that deaf-mutism refers to those who have lost or failed to acquire speech on account of deafness.*

Another classification, which is perhaps better as a practical working basis, is that adopted by Mygind, namely:

(*a*) Congenital deaf-mutism.

(*b*) Acquired deaf-mutism.

The first class refers to those who are born with some defect of the organ of hearing, which, according to modern statistics, includes about 50 per cent. of all the cases. Mygind thinks this estimate too high, as many of the so-called congenital cases are, in all probability, due to some intercurrent disease of the ear which destroys the hearing before articulate speech is acquired. While the author's observations have been comparatively limited, they have nevertheless been sufficient to recognize the difficulties to be encountered in determining whether certain cases belong to the congenital or to the acquired class. The author is, therefore, inclined to agree with Mygind that 50 per cent. is too high an estimate to be placed upon the relative proportion of congenital as compared with the acquired types of deaf-mutism.

The *relative proportion* of deaf-mutes to the total population of the various countries in which statistics are to be found varies from 34 (Holland) to 245 (Switzerland) per 100,000 inhabitants. The average in European countries is 79, while in the United States it is 68 per 100,000 inhabitants.

Etiology. The great variation in the relative number of deaf-mutes in the different countries seems to point to certain localities as predisposing to it. *Old geological* (Escherich) *formations*, as found in the Alps, were formerly thought to be the cause, but more careful investigations have shown this to be incorrect. In Switzerland, where the rate is so high, it is due to the endemic cretinism so prevalent there. This phase of deaf-mutism is not included in the consideration of this subject.

Climate probably has no influence.

Unfavorable *social* and *hygienic* conditions play a very important part in the etiology of deaf-mutism.

H. Schmaltz emphasizes this in his work on *Deaf-mutism in Saxony*. In conclusion, he says: "The industrial population, and especially that part of it which is worse off from a pecuniary point of view—in fact, all who are in danger of degenerating both morally and physically on account of insufficient means, or poverty, and who, consequently, are

unable, or unwilling, to take the necessary care of their children—all such persons exhibit the highest percentage of deaf-mutes among their descendants. Finally, we found that when, in addition to all these unfavorable conditions under which children are born, they are brought up by a family which, for various reasons, is perhaps already more or less degenerated, and have to undergo all sorts of diseases in infancy without having sufficient power of resistance, then deaf-mutism is an only too common result."

Heredity undoubtedly influences the number of deaf-mutes. Mygind very tersely expresses the present status of our knowledge on this point in the following words: "Deaf-mutism is comparatively frequent among the relatives of the deaf-mutes; it is least frequent in the direct ascending line (grandparents, parents); more frequent in the collateral branches (great-uncles, great-aunts, uncles, aunts, cousins, parents' cousins, and second cousins); and most frequent by far among the brothers and sisters of the deaf-mutes. This is in exact accordance with the result of an investigation into the appearance of deaf-mutism among the relations of congenital deaf-mutes; from this and many of the facts above mentioned, we are justified in supposing that the manner in which deaf-mutism appears in different generations is a result of certain qualities appertaining to its congenital form."

It is not assumed that deaf-mutism *per se* is transmitted by hereditary influences, but that certain anatomical or nervous states are retained to some extent, and that these may result in deaf-mutism—that is, deaf-mutism is influenced by the transmission of a predisposition to certain ear diseases and to certain nervous disorders. These, in combination, tend to produce the affection.

Consanguineous marriages seem to influence the number of deaf-mutes, as is shown in the following table:

FORTY-SEVEN MARRIAGES BETWEEN BLOOD RELATIONS PRODUCED SEVENTY-TWO DEAF-MUTES

1	marriage	between	aunt and nephew	produced	3	deaf-mutes.
4	marriages	"	uncle and niece	"	11	"
26	"	"	first cousins	"	3	"
16	"	"	second cousins	"	20	"

Statistics prove that the influence of consanguineous marriages is entirely limited to congenital deaf-mutism.

Various diseases in parents, as alcoholism, syphilis, general debility, epilepsy, insanity, etc., are etiological factors in the production of deaf-mutism. The offspring of such parents do not receive *in utero* the vital energy necessary to resist the vicissitudes of life after birth. They are, therefore, more liable to be injured by infections and nervous diseases than the offspring of healthy parents. It may be said in this connection, however, that the parents of deaf-mutes are often remarkably healthy and robust individuals.

Hemophilia and deaf-mutism are rather commonly associated among the offspring of marriages producing a large number of children.

The death rate is higher among children in families in which there are deaf-mutes, probably on account of the stigmata of degeneracy, and because suppurative otitis media adds to the mortality rate.

Mygind cites statistics to show that first births produce more deaf-mutes than either the second, third, fourth, or fifth. Other weaknesses are also more common among the first born.

Maternal *impressions* do not appear to exert a marked influence in the production of deaf-mutism.

Immediate Causes of Deaf-mutism.—The *age* at which most cases of deafness occur in the acquired type is from the first to the fifth years, more occurring in the second and third years. In the United States the greater number occur in the third year.

Brain diseases, more particularly simple meningitis and epidemic cerebrospinal meningitis, are the chief causes of the acquired deaf-mutism. From 12 to 26 per cent. of the European cases have been attributed to epidemic cerebrospinal meningitis. Moos and Knapp were the first to call attention to this disease as one of the causes of deaf-mutism.

Deafness may occur during epidemic cerebrospinal meningitis resulting from middle-ear or labyrinthine lesions. The former occurs more often, but is not so pronounced nor so permanent as that due to the involvement of the labyrinth. Deafness of middle-ear origin does not so often produce deaf-mutism on this account. Labyrinthine involvement usually occurs about the second week of epidemic meningitis, although it may occur at a much later period (Knapp, Mygind). The deafness occurs suddenly, in contradistinction to that in middle-ear deafness. Postmortem examinations have shown most of them to be due to inflammation of the membranous labyrinth. "This process leads partly to the more or less complete destruction of the contents of the labyrinth, and partly to the deposit of new tissue. The new tissue may be either fibrous, calcareous, or osseous, and may fill the normal cavity of the labyrinth either completely or partially." (Mygind.)

The original cause of the disease is undoubtedly some microörganism which enters through the ear, nose, or epipharynx, although definite data is not yet at hand to confirm this statement.

The *equilibrium* is often disturbed in deafness due to brain disease, as pointed out by Moos. This is due to the involvement of the semicircular canals and other apparatus of the labyrinth. This may endure for years.

Other *acute infectious diseases*, as scarlet fever, measles, yphus and typhoid fevers, diphtheria, smallpox, vaccination, chickenpox, erysipelas, dysentery, influenza, malaria, whooping cough, mumps, croupous pneumonia, and rheumatic fever, directly or indirectly, cause infantile deafness. The inflammation first attacks the mucosa of the middle ear, which ulcerates, the bone beneath becomes carious, and the meninges and labyrinth are thus exposed to infection. The ossicles of the middle ear, being covered by the mucous membrane, undergo the same changes. If the destruction does not involve the labyrinth, the deafness is not

usually profound enough to cause deaf-mutism. If it involves the labyrinth, the same changes described under cerebrospinal meningitis take place and result in complete and permanent deafness. If this occurs before speech is acquired, the child becomes a deaf-mute.

In scarlet fever, measles, and kindred diseases, the infection enters the tympanum through the Eustachian tube. The labyrinth is usually invaded through either the oval or round windows, as has been shown in numerous autopsies by the scar on the membrane. In some cases, however, it appears that the middle ear is not involved, the drum membrane being normal. It is probable in these cases that the infection reached the labyrinth by metastasis.

Smallpox does not account for many cases of deaf-mutism in those countries where compulsory vaccination is in vogue. It is barely possible that vaccination may cause deaf-mutism.

Connor collected the literature of labyrinthine disease caused by mumps up to 1884, and found 33 cases, 9 of which were fifteen years of age or less.

Certain constitutional diseases, more particularly syphilis, scrofula, and rickets, are occasional causes of deaf-mutism. Inherited syphilis causes it more often than is shown by the statistics, as it is difficult to ascertain the data concerning this affection.

Fright, lightning-stroke, sunstroke, quinine poisoning, colds in the head, sudden immersion in water, and traumatisms occasionally cause deaf-mutism. A fuller knowledge of the causes of deaf-mutism should attain among physicians, as it is to them the parents will first appeal for information and relief. Many of the cases may be so educated as to make them useful members of society and a source of gratification to themselves and to their parents, if the needed advice or attention is given them at the proper time, *i. e.*, while their minds are still in the imaginative and perceptive stages of development. (See Lip Reading.)

Pathology.—Reliable postmortem examinations in 139 cases of deaf-mutism are on record. From these the following facts are gleaned (Mygind): (The changes in the external ear and the auditory meatus will not be considered, as they could have but little to do with the causation of deaf-mutism.) In the drumhead, perforations, calcareous deposits, adhesions, thickening, and entire absence have been found.

In the *middle ear* adhesive processes, calcifications, and ossification from intense inflammation have been found. The oval window is sometimes filled in with a mass of bony tissue (hyperostosis), while the round window is contracted in size. The membrane of the round window is sometimes thickened, or thinned, scarred, calcareous, or absent.

Osseous masses in the attic and other portions of the middle-ear cavity have been found. Caries of the bony walls of the middle ear from chronic suppurative inflammation are sometimes present.

The ossicles are ankylosed, bound down by adhesions, necrotic or entirely destroyed, from suppurative inflammatory processes, in a considerable number of cases. One or more of the ossicles may be absent, and the others present, the stapes alone being absent in a number of cases.

When atrophy of the *ossicula auditus* is present, it is probably of congenital origin.

Ankylosis of the ossicles is very often present.

Atrophy and *caseous degeneration* of the tensor tympani and stapedius muscles is often found. The chorda tympani nerve is sometimes absent.

The *mastoid process* is found to be affected, as elsewhere described under Suppurative Diseases of the Middle Ear and Mastoid Process. It is sometimes absent from arrested development.

The *Eustachian tubes* are sometimes obstructed by fibrous or osseous tissue, as a result of repeated inflammations.

The Labyrinth.—The most frequent pathological change found in the labyrinth is the deposit of osseous tissue from inflammatory processes. This is sometimes so extensive as to completely obliterate the labyrinthine canals (Mygind), and gives rise to the idea that there is congenital absence of the labyrinth from arrested development (Montain, Michel, Schwartze, Moos). Chalky pigment and fibrous deposits are also found.

Absence of the auditory nerve and labyrinth (partial or complete) are also reported. In one of Mygind's cases the labyrinth was completely filled with osseous tissue, except at certain portions where pus was present. It was due to a suppurative process following scarlet fever.

The membranous labyrinth may be congenitally absent, as shown by Nuhn.

The *vestibule* (excepting its aqueductus) is rarely involved, even in congenital cases. When an affection is present, the changes are inflammatory in origin. Pathological changes in the contents of the membranous vestibule have often been found.

The *aquæductus vestibuli* may be distended, in which case the cochlea is also affected (Ibsen) while the vestibule is not, thereby suggesting an intimate relation between the aquæductus and cochlea rather than the vestibule. Habermann explains the distention of the aquæductus vestibuli as being due to pressure in hydrocephalus, especially when the petrous portion of the temporal bone is rachitic.

The *semicircular canals* are quite commonly affected.

Symptoms.—*Deafness* may be partial or complete. If partial, there may be *hearing* for sounds, noises, voice, or speech. One child, for example, may hear a loud noise and not hear speech, or *vice versa;* or he may hear the voice and not hear articulate speech. Again, he may hear tones of a certain pitch and not hear those of another pitch.

As stated in the beginning of this chapter, the best classification is (*a*) true deaf-mutes, and (*b*) semi-deaf-mutes. In other words, those who have partial hearing and those who have total absence of hearing. It is often difficult to determine this point in young infants, for obvious reasons. In older ones it can be usually done by the use of bells, loud whistles, clapping hands, etc. The child will blink the eyes, or show by a change in its expression that it hears.

A more accurate method of testing older deaf-mutes may be made with tuning-forks and whistles. The hearing should be tested by both

air and bone conduction. Hearing by air conduction is tested by holding the vibrating fork near the external auditory meatus and noting the expression of the child; bone conduction is tested by placing the handle of the vibrating fork on the mastoid or the vertex of the head, the expression of the child being meanwhile watched for signs that it experiences a novel sensation. Other instruments, as the watch and the Politzer acoumeter, may be used if there is considerable hearing present. The voice, especially the articulate vowels, is a good test when spoken close to the patient's ears, care being exercised to prevent them seeing the movements of the lips. If they hear the vowels, consonants and words may also be utilized.

Semi-deaf-mutes hear better at certain times than at others, for the same reason that those with less pronounced middle-ear disease have variations in hearing.

The various reports as to the relative number of the totally deaf and partially deaf in the various statistical publications are not reliable, as different tests have been used to determine these facts. There are more cases of profound or total deafness among the acquired than the congenital cases, probably on account of the great severity of postnatal processes in the ear.

A very significant fact has been announced by Urbantschitsch, namely, that children who had previously reacted to no sound whatever, after certain acoustic exercises, were capable of hearing. This points to the fact that a sensory tract is developed by use. Its powers, or functions, may lie dormant for years, and then be aroused to activity and development. The fact that a child never has heard is not necessarily proof that it never will.

Mutism may be the result of the deafness, or it may be due to the same influences which caused the deafness. There may be an arrested or perverted development of the vocal organs, coincident with the disturbed development of the ear; or aphasia may be due to a congenital or acquired lesion of the brain. If the speech centres of the brain were injured at the time the ear was affected, the child can never be taught to speak clearly.

The age at which deafness must occur to produce mutism is not to be stated arbitrarily, as the capacity to learn speech varies greatly in different children. Hartmann says that if deafness occurs before the seventh year, mutism is apt to follow. The slight speech already acquired will gradually disappear unless special pains are taken to cultivate it.

The *speech of deaf-mutes* is peculiar, lacking in proper accentuation, which renders it monotonous. The respiratory act is deficient, and the voice feeble. The greater the deafness the more pronounced the peculiarities of the speech become. True deaf-mutes, as well as semi-deaf-mutes, may be taught articulate speech, which is known as "articulation." Deaf-mutes experience great difficulty in retaining "articulation" when they leave the school-room and mingle with those who can scarcely understand them. *Articulation* is quite different from ordinary speech, and it is only after hearing it used to a considerable

extent that one learns to understand it. This is one of the difficulties in the way of its more general use among deaf-mutes. Lip-reading is learned at the same time as articulation, but, as it requires close attention and good sight, it is also often abandoned when contact with the world at large is established.

Other ear symptoms, as tinnitus, giddiness, staggering gait, and otorrhea, are present in a certain number of deaf-mutes. Otorrhea is quite common, especially among the acquired cases.

Sequelæ.—An impairment of the mental faculties may or may not be present. When it is remembered that a deaf-mute is barred from many avocations, it is easy to understand that ambition is thereby hindered. The temptation to idleness and dependence upon those more fortunate often stultifies the mental and moral faculties. The morbid processes causing the deafness may also impair other portions of the brain, and thus impair the mental faculties. About 50 per cent. of those who are deaf-mutes are notably deficient in mental power.

The laryngeal muscles are slightly atrophied from non-use; otherwise the larynx is usually normal.

The *lungs* of deaf-mutes seem to be less resistant than those of other children, as shown by the fact that so many of them die of tuberculosis. This is still further shown by stethoscopic examinations. Their breathing is more superficial and less rhythmical than in normal children. This is also true of children with normal ears who have defects of speech, such as stammering.

Tuberculosis, *scrofula*, *sterility*, left-handedness, and diminution of muscular energy are commonly found among deaf-mutes.

The auricle is rarely malformed in deaf-mutes, as it develops independently of the internal ear. The external meatus and membrana tympani show such changes as are incident to middle-ear diseases in general. The same is true of the Eustachian tube and mastoid process.

Adenoids and catarrhal affections of the nose and epipharynx do not seem to be more common among deaf-mutes than other children. That there is a direct relation between infections which enter the middle ear through the epipharynx and Eustachian tubes there can be no doubt. The same irritation causes the adenoid tissue to enlarge, a fact which explains the apparent etiological relationship of adenoids to deaf-mutism.

Boucheron advances the ingenious theory that deaf-mutism may be caused by *otopiesis*, meaning thereby deafness by "producing exhaustion of the air in the middle ear as the result of the closing of the catarrhally affected Eustachian tubes, which process, again, causes overpressure in the inner ear, and consequently degeneration of the terminations of the auditory nerves." (Mygind.)

There are other abnormalities coincident with deaf-mutism, such as malformation of the cranium, the eye (retinitis pigmentosa, hemeralopia, "hen-blindness," panophthalmia, etc.), thyroid gland, nerves, and bones. They are largely the result of the same influences which primarily cause deaf-mutism.

The relationship between idiocy and deaf-mutism is not that of cause and effect, as they are both the result of the same primary influences. Deaf-mutism does not cause idiocy.

Insanity is estimated (Wines) to be four times as common among deaf-mutes as in individuals in general. Mygind shows that this is probably due to the isolated social position and mental depression which naturally attend the loss of one of the chief senses.

Diagnosis.—The diagnosis is easy in most cases, and is based on the following facts:

(*a*) Deafness so pronounced that speech cannot be heard.

(*b*) Deafness dates from birth or before the seventh year.

(*c*) Deafness and fragmentary speech (semi-deaf-mutes).

In infants it is difficult to make a diagnosis, as the child does not yet speak, and it is difficult to determine if it hears. Loud bells, clapping of hands, whistles, etc., should be used without letting the child see them, noting the blinking of the eyes or other signs that it recognizes the noises. A negative result is not, however, conclusive of deaf-mutism. Hartmann has called attention to the fact that some children do not have the organ of hearing fully developed at birth, the development being completed at the first year of extra-uterine life.

Simple mutism (aphasia) may be mistaken for deaf-mutism upon casual examination, although it is seldom congenital or acquired in infancy. Careful examination will show hearing present.

Simulation of deaf-mutism and hysterical deaf-mutism are rarely seen.

Prognosis.—A few well-authenticated cases are recorded in which the hearing was improved. The great majority, however, are not thus favorably affected. The number of cases reported by men of the highest standing, as being so much improved that they regained enough hearing to carry on conversation with their fellows, warrants the use of every means within our power to alleviate all ear affections, with the hope that those under our care may also be thus favorably influenced. Some cases undoubtedly improve spontaneously.

Speech will generally improve in proportion to the improvement in hearing.

Treatment.—The treatment should be such as would be given to similar ear affections in those who are not deaf-mutes. Suppurative disease should receive special attention, to prevent it spreading to neighboring organs. Postnasal adenoids and other diseased processes of the nose and throat should receive appropriate attention according to the methods described elsewhere in this work.

After having done all that can be done to improve the organ of hearing and the general system, the child should be sent to some institution of reputable standing, where he can receive suitable training in the acquirement of speech or other means of communication. Here he will also receive instruction in useful knowledge and manual training, which will fit him for a place in social and economic life.

The prevailing methods of instruction are known as the German and French methods. The first is probably the best for a majority of deaf-

mutes, as it teaches them articulate speech. There seems to be no doubt that the use of the vocal organs stimulates the development of the brain and motor tracts. Makuen has called attention to this fact. (See Defects of Speech.) The French method teaches communication by means of signs. This is probably well adapted to some cases. The question of methods should, however, be left to those who are more intimately concerned to decide. It is not the physician's province to train these unfortunate children. His duty is to relieve the physical condition as nearly as possible and then recommend the parents to send the child to some reputable institution for deaf-mutes, assuring them that only in this way will he be fitted for a useful place in society.

LIP READING

Deaf-mutes, and persons so deaf as to understand conversation with difficulty, should be taught lip reading whenever possible. It has long been known that persons partially deaf watch the face of the one addressing them, and by combining what they imperfectly hear with the movements of the lips, the facial expression, and the gestures of the speaker, they are enabled to understand what is being said. This suggested the advisability of reducing lip reading to a scientific basis, and schools for this purpose are now established in nearly all large cities.

The acquirement of facility in lip reading necessitates the closest application on the part of the student, and the most painstaking and persistent effort on the part of the teacher; hence, there is little hope of success outside of a special institution for the purpose. The physician cannot give adequate attention to such patients, and he should recommend that they be sent to a school at as early an age as possible, as otherwise the patient will be greatly handicapped in the pursuit of his business in later life. As there are many charlatan schools advertising to give such instruction, the physician should first make diligent inquiry as to which are conducted upon scientific lines before making any recommendation.

Lip reading may also be profitably studied by adult deaf persons whose early education in this respect was neglected.

INDEX OF AUTHORS

A

Abels, Hans, 897
Adami, 111, 127
Alexander, 882, 891
Allport, 841
Andrews, A. H., 51, 194, 616, 714
Aristotle, 1023
Arnold, 448
Asch, 67, 68, 77, 78, 79
Atkinson, 442

B

Babinski, 488
Bacon, 1008
Ball, 349, 350, 402, 405, 408
Ballance, 601, 733, 840, 853, 854, 855
Barany, 627, 628, 881, 882, 891, 906, 908, 909, 910, 911
Bardeleben, 991
Baron, 268
Baumgarten, 324
Beard, F., 376
Beck, Emil, 98
Beck, Joseph C., 43, 101, 155, 158, 219, 220, 314, 328, 329, 996
Berard, 326
Berens, T. Passmore, 609
Bezold, 616, 617, 622, 627, 735
Bickel, 331
Bier, 127
Bikeles, 479
Bing, 628
Blake, Clarence, 680, 681, 741
Bland-Sutton, 369
Boenhaupt, 279
Bono, 33, 34
Bostoc, 548
Bostroem, 326
Bosworth, 57, 68, 70, 160, 442, 444, 553
Boubland, 551
Bouche, 367
Boyce, John W., 582
Brandegee, 341
Brauers, 558
Braun, 903
Brawley, 127, 196
Briggs, 485
Brindel, 333
Broca, 766
Brown, J. S., 652
Brown, R. H., 95
Browne, Lennox, 308, 310, 349, 350, 354, 365, 368, 370, 373, 503, 512, 545
Bruce, 400
Bruhl, 763
Brunk, Thomas H., 684, 688
Brunnung, 891
Bruns, 370
Bryant, J. D., 444
Bryant, W. S., 684
Buchanan, T. Drysdale, 589
Burnet, 635
Burnett, 446, 644
Burns, B. V., 548
Buck, 320
Buck, A. H., 724

C

Campbell, J. T., 376
Cananus, 1023
Canfield, 218, 236
Cardanus, 1023
Carel, 339
Carter, 88
Casselberry, 282, 358, 424
Celsus, 637
Charcot, 1003
Charsley, 268
Chiari, 329, 470, 500
Chimani, 1014
Church, J. F., 635
Clark, C., 547
Clark, J. Payson, 547, 548, 549
Coakley, C. G., 162, 473
Cobb, C. M., 197
Cobb, F. C., 25
Coffin, 164
Coghill, 309
Cohen, J. Solis, 309, 508
Cohen, R., 537
Cohen, S. Solis, 261
Colburn, J. E., 187
Conitzen, 369
Connor, 1027
Coolidge, Frederick, 25, 347
Cooper, Astley, 194, 230, 733
Corlin, 462
Corwin, 227
Cosolini, 443
Crisp, 448
Cunes, 551
Cunningham, 563, 569, 589
Curtis, 470, 471, 527, 530

D

Dabney, 127, 196
Daly, William, 26
Dawbarn, 377, 378, 558
Dawson, 381
Day, Ewing W., 943
de Reske, Jean, 23
Dehio, 309
Delevan, 442, 548
Delstanche, 760
Demme, 309
Dench, E. B., 762, 767, 793, 854
Denker, 233, 735, 737
Deprès, 320
di Mendoza, Suarez, 690
Dieulafoy, 380
Duchemin, 533
Duel, A. B., 689, 742, 743, 815
Dunbar, 262
Dupuy, 680, 766, 767

E

Edinger, 514
Eisenlohr, 515
Escat, 351
Escherich, 1024
Esmarch, 360
Evans, 533
Ewald, 874

F

Farlow, 373, 442
Farrel, 400
Farrington, 255
Faurd, 547
Fauvel, 545
Ferreri, 546
Fish, 164
Fish, H. M., 185, 186
Flatau, 442
Flemming, 384
Fletcher, J. R., 87, 891
Fleurens, 874
Foster, Hal, 289
Foucher, 351
Fraenkel, E., 309
Fränkel, B., 266, 556, 560
Frazier, 809
Fredet, 448
Freeman, 57, 58
Freer, 82, 85, 87, 100
Freisner, 903
Freudenthal, 313
Frey, 891
Friedreich, 26, 379
Frisco, 33, 34
Funke, John, 785, 786

G

Gallagher, 313
Gautier, 328
Gellius, 1023
Gerhardt, 544, 554
Getchell, 512
Gibb, 446
Glass, 288
Gleason, 67, 75
Gleitsmann, 309, 313
Gluck, 557
Goldsmith, P. C., 99, 109
Goldstein, 316, 772
Goldstein, Max A., 54, 104, 141, 289, 730
Goodale, 44, 285, 309, 380, 385, 386, 396
Gottstein, 514, 661
Gradenigo, 758, 1006, 1008, 1021
Gradle, 344, 758, 787, 790
Grant, Dundas, 98
Green, 551
Grober, 384, 388, 390
Grosvenor, 443
Gruber, 268, 672
Grunert, 815
Grünwald, 155, 156, 181
Guerard, 485
Gulland, G. L., 384
Guns, 155
Guye, 302, 337, 538, 541
Guyon, 373
Guyot, 695

H

Habermann, 735, 761, 1028
Hack, 268
Hajek, 84, 90, 161, 447, 470
Halle, Max, 210
Halstead, T. H., 400, 575
Hammerschlag, 891, 1016
Hannebert, 906
Hansen, 322
Harpy, 312
Hartly, Frank, 558
Hartman, A., 1029
Hartz, 741
Hasslauer, 815
Hawley, George F., 44
Hazletine, 105
Heath, 44, 779, 851, 853
Heath, Charles J., 833
Heath, Clarence, 756
Heinze, 309
Helmholtz, 613, 614, 1004
Herodotus, 1023
Hertz, Henry J., 735
Heuth, 853
Heysinger, 646
Hezener, 736
Hibbard, 480
Hillis, 497
Himly, 733
Hippocrates, 1023
Hirsch, 235
Hoegyes, 874
Hollander, 306
Hollinger, 403, 767
Holmes, 145, 187, 634, 766, 767
Hoople, Heber Nelson, 34, 35
Hopkins, F. E., 414
Horsley, 514, 518
Hotz, 756, 782
Hovell, 651, 674, 703, 714, 772
Hubbard, Thomas, 701
Huizinga, J. G., 187
Hurd, L. M., 95

I

Ibsen, 1028
Ingals, E. Fletcher, 214, 442, 581
Itard, 1023

J

Jack, 741
Jackson, Chevalier, 505, 515, 555, 557, 577, 579, 581, 586, 587, 589, 597, 599
Jansen, 610, 834, 853, 904
Jarvis, 57, 58, 59
Johnson, 313

K

Kahn, Harry, 278
Kalisko, 479
Kanasugi, 308
Kaposi, 287
Katz, 735, 738
Kauffmann, 54, 391, 403, 408
Keen, W. W., 568
Keimer, 442
Killian, Gustav, 19, 69, 84, 90, 99, 182, 219, 220, 221, 222, 223, 224, 251, 260, 264, 385, 547, 578, 580, 581, 585, 586, 587
Kirschner, 854
Klebs, 476
Kleishaber, 558
Knapp, 793, 1026
Knight, 309
Knoblauch, 1020
Kobrak, 815
Kocher, 381, 557
Koerner, 601
Körner, 833
Kowalzig, 276
Kramer, 695
Kraus, 514, 518
Krönlein, 979
Kuhnt, 217
Kuster, 218, 231
Kyle, D. Braden, 68, 69, 80, 154, 155, 254, 270, 278, 307, 327, 414, 454
Kyle, J. J., 1005

L

Lack, Lambert, 302, 303
Lake, Richard, 644, 975, 1004
Lancereaux, 309
Landois, 383
Lange, 902
Laurentius, Andreas, 1023
Lehart, 815
Lemcke, 1023
Lermoyez, 217, 339
Leutert, 603, 754, 755, 815
Levy, Robert, 36, 270, 313, 315
Lewin, G., 350
Liebreich, 262
Lobe, 262
Lockard, 313
Loeb, H. W., 162, 269
Loeffler, 476
Loewy, 57

Lohnberg, 314
Louis, 448
Lowenberg, 637, 696
Lubet-Barbon, 766

M

McAuliff, G., 649
McBean, 1017
McBride, 264, 333, 335, 338, 723
McKenzie, 434
McKernon, J. F., 197
MacDonald, 157
Macdonald, Greville, 368
Macewen, 766, 767, 770 771, 772, 787, 811, 819, 982
MacKenzie, E., 516
Mackenzie, G. Hunter, 549
Mackenzie, G. W., 872, 881, 882
Mackenzie, John, 317
Mackenzie, Morrell, 414, 530, 531, 545, 547
Makuen, 536, 540, 541, 543, 1032
Malherbe, 678, 733
Manasse, 736
Manicatide, 479
Martin, 481
Maschziker, 646
Masini, 392
Massei, 507
Maxwell, George T., 416
Mayer, 77, 78, 79, 641
Mayer, Emil, 286, 304
Mayo, 310
Meissner, 154, 1023
Mendel, F., 34
Metchnikoff, 386
Meyer, William, 26
Meyjer, 448
Michel, 447, 1028
Miller, 470
Milligan, A. W., 314
Minot, 646
Miot, 699
Mojocchi, 328, 329
Moll, 500
Montain, 1028
Monti, 480, 484
Moorhof, 992
Moos, 786, 1007, 1014, 1026, 1028
Morgani, 57, 58, 59
Morrissy, 480
Mosetig, 992
Mosher, 57, 247
Moss, Robert E., 551, 735
Mount-Bleyer, J., 533
Moure, 81, 250, 344, 547
Müller, J., 761
Mygind, H., 306, 1023, 1024, 1027, 1028, 1030
Myles, 225, 238

N

Nagel, 197
Neumann, 87, 88, 678, 874, 880, 882, 884, 891, 911, 912
Neurenberg, 815
Newkirk, 345
Northrup, 477, 479, 480, 483, 490

O

O'Dwyer, 492, 493
Ollier, 282
Oppenheimer, 814, 815
Orth, 309, 736
Osler, 322, 323
Ostrum, 223

P

Packard, 380
Page, Lafayette, 541
Panse, 601, 990
Parker, 352
Passow, 990
Patterson, 387
Paul of Ægina, 639
Payson, J., 547
Pean, 559
Pedro de Ponce, 1023
Peltesohn, Felix, 331
Phillips, 442
Pierce, Norval H., 54, 141 262, 758, 767, 848
Piotrawski, 351
Pischel, 72
Pliny, 927
Politzer, 616, 619, 620, 646, 651, 656, 659, 660, 661 667, 673, 676, 687, 694, 696, 699, 701, 702, 724, 725, 727, 728, 729, 730, 733, 738, 740, 744, 746, 747, 761, 762, 763, 766, 767, 902, 1000, 1001, 1002, 1003, 1006, 1008, 1011, 1016, 1022
Posey, W. C., 186
Poucet, 326
Powers, 557
Price-Brown, 67, 69, 81
Pritchard, Urban, 1004
Proust, 515
Pynchon, 127, 196, 207, 403, 408, 415, 429, 634, 698, 710

Q

Quénu, 351

R

Rach, 69
Raugi, 369
Rebinski, 309
Reik, 197
Reinhard, 853
Reininger, 155
Reszke, Jean de, 527
Retzius, 541
Ribbert, 786
Richards, 985
Richards, George L., 344, 441
Richards, John, 972, 974
Richardson, Charles, 441
Rist, 786
Riverias, 637
Robertson, 310, 428
Roe, 68, 69, 70, 77
Roosa, St. John, 767
Root, A. G., 524
Rosenback, 519
Rosenberg, 548
Roux, 476, 481
Rubenstein, 304
Rumbold, T. M., 632
Ruttin, 873, 901, 933, 936, 938

S

Saissy, 646, 695, 696
Sajous, 507
Santorini, 601
Schadle, 207, 255, 260, 345
Schaus, 58, 59
Scheibe, 735
Schilling, 735
Schmidt, Moritz, 266, 373, 544, 545, 546, 547
Schmalz, 1023, 1024
Schnitzler, 547
Schroetter, 505, 547, 554
Schwalbe, 541
Schwartze, 646, 723, 726, 766, 767, 771, 1028
Seifert, 350
Seiss, 350
Semon, Felix, 262, 414, 514, 516, 519, 544, 546, 553, 554, 555, 557
Senator, 515
Senn, 326
Sexton, 1011
Sexton, Samuel, 639
Shambaugh, 613, 739, 861, 1004
Sheppard, 766

Shurley, 309, 548
Siebenmann, 601, 738
Simon, 486
Skilleren, 148, 269
Sluder, Greenfield, 67, 69, 74, 192, 430, 431, 433
Smith, Harmon, 547, 548
Sondermann, 127, 196
Spencer, 758
Stacke, 829, 833
Stahl, 645
Steele, J. S., 289
Stein, O. J., 19, 262, 263, 279, 317, 555
Stirling, 383
Stoerk, 574
Stöhr, 381, 382
Strassmann, 379
Stubbs, 343
Stucky, 115, 164, 353, 449, 563, 569, 733, 767
Suker, G. F., 187, 647, 732
Swain, H. L., 261, 348, 549
Sydacker, 763

T

Talbot, Eugene S., 57, 58, 59, 646
Terry, W. J., 556
Theisen, F., 447, 448
Theobald, 756
Thomer, 304
Thompson, 265, 266, 345, 1024
Thorner, Max, 306
Tilley, 230
Tompkins, 448
Toynbee, 726, 735, 1007, 1023
Trautmann, 992
Trendelenburg, 57, 58
Trousseau, 490
Türck, 314
Turner, 192, 333, 335, 338
Turner, Logan, 164, 547, 723

U

Uffenorde, 148, 269
Urbantschitsch, 640, 689

V

Vail, 145, 226
Valsalva, 673, 694, 701
Verneuil, 351
Vieussens, 155
Villar, 370
Virchner, 1007
Virchow, 443, 504, 554, 646
Voltolini, 641
von Babes, 381
von Bergman, 555
von Esmarch, 276
von Langenbeck, 276, 277, 445
von Stein, 926
von Tröltsch, 639, 640, 669, 726, 761, 1023, 1024

W

Wade, 376
Wale, 610
Wales, 204
Walsh, 474
Walsham, 302, 381
Watson, 67, 68, 69, 73, 268, 558
Waxam, F. E., 492, 493
Weber, 1016
Weiss, 751
Welcker, 59, 61
Werckmeister, 557
Whalan, 512
White, 557
Whiting, 767, 816, 825, 826, 829, 836, 981
Wild, 1023
Wilde, 770, 824
Williams, Watson, 20, 267, 268, 380, 469
Williston, 636
Wilson, 414, 522, 596
Wingrave, Wyatt, 786
Woakes, 115, 449
Wood, 164
Wood, C. A., 187
Wood, G. B., 387, 408, 410, 414, 415, 416
Woodruff, 259
Wreden, 644
Wright, Jonathan, 44, 127, 380, 381, 385, 386, 391, 396, 544, 546, 547, 815

Y

Yankauer, Sidney, 90, 715, 724, 731, 734
Yersin, 476

Z

Zaufal, 603, 637, 703, 754, 1023
Zeim, 164
Ziemssen, 367, 553
Zuckerkandl, 57, 103, 351

GENERAL INDEX

A

Abscess of brain, 799
surgery of, 976
peritonsillar, 404. *See* also Peritonsillitis.
retropharyngeal, 359
subperiosteal mastoid, 770
Acoumeter, Politzer's, 620
test for hearing, 620
Actinomycosis of nose, 325
etiology of, 325
pathology of, 326
of pharynx, 326
diagnosis of, 327
from carcinoma, 328
from sarcoma, 327
from syphilis, 328
from tuberculous disease, 328
electrolysis in, 328
iodonucleoid in, 328
potassium iodide in, 328
symptoms of, 326
treatment of, 328
of tonsils, 326
Adductor spasm of larynx, 509. *See* also Laryngismus stridulus.
Adenectomy, anesthesia in, 340
Richards' method of, 344
Adenoids, 333
aprosexia in, 337
auditory apparatus and, 346
aural complications in, 338
blood in, 347
bones and, 347
chronic suppurative otitis media and, 757
moist catarrhal otitis media and, 723
club foot and, 347
deaf-mutism and, 1030, 1031
diffused hypertrophic laryngitis and, 465, 467
diagnosis of, 338
from granulomata, syphilitic, 339
tuberculous, 339
from tumors, fibrous, 338
malignant, 339
epipharyngeal catarrh in, 337
inflammation and, 345
in epipharynx, 337
Adenoids, epithelium in, 334
etiology of, 333
Eustachian tubes and, 346
"face," 339, 345
fibrous tissues in, 335
forceps, Brandegee's, 339, 341
"Gothic" arch and, 345
hard palate and, 345
hearing in, defective, 337, 633, 634
heredity and, 333
lymphoid tissues in, 335
membrana tympani in, 346
mentality in, 337
mouth breathing in, 337
nasal inflammation and, 115
operations for, Ballenger's, 339
Boeckmann-Stubbs' curette in, 341
Brandegee's adenoid forceps in, 339, 341
Ferguson-Pynchon's mouthgag in, 340
Meyer's ring curette in, 344, 349
pharyngeal scissors in, 347
position of patient in, 340, 342
Pynchon-Golding-Bird's curette in, 344
Quinlan's forceps in, 344
Shutz's adenotome in, 344
Stubbs', 343
otitis media and, 338
pathology of, 333
pharyngeal, 333
"pigeon chest" and, 347
prognosis of, 339
respiration in, 336, 343, 344
sequelæ of, 345
singing voice and, 526
speech in, defective, 337, 539
suppurative otitis media and, 346
symptoms of, 336, 337
teeth and, 345
Thornwaldt's disease in, 347
treatment of, 339
tubal catarrh and, 684, 685
vegetations, 333
Adenoma of nose, 278
Adenopathy, diphtheria and, 485
Adentome, La Force, 339
Adhesive processes in middle ear, 724
Aditus ad antrum, obstruction of, 767, 779, 780
relations of, 609

Aditus ad antrum, relations of, to facial nerve, 834
Adrenalin in epistaxis, 285
Agglutination methods for removal of foreign bodies in ear, 637, 638
Agraphia, amnesic, 1020
Air pressure, negative, treatment of sinuitis by, 196
Akinesis of pharynx, 365
Alæ nasi, collapse of, 302
etiology of, 302
operation for, Lack's, 303
Walsham's, 302
symptoms of, 302
treatment of, 302
Albuminuria in diphtheria, 482, 485
diphtheria antitoxin and, 489
hemorrhagic laryngitis and, 472
Alcohol, 51
diffused hypertrophic laryngitis and, 465, 467
hyperacuteness of hearing and, 1016
in hyperemia of labyrinth, 1000
injections for hyperesthetic rhinitis, 263
tinnitus aurium and, 720, 727
treatment of chronic suppurative otitis media, 760, 781
of diphtheria, 468
Alcoholism, arteriosclerosis of labyrinth and, 1005
deaf-mutism and, 1025
Alimentary canal in diphtheria, 480
Allen's nasal speculum, 97
Allport's bone-crushing forceps, 858
divulsion forceps, 841
mallet, 828
mastoid retractor, 858
Alveolar operation upon maxillary sinus, 238
process, lavage of maxillary sinus through, 194
American method of inflation of tympanic cavity, 700
Amnesic agraphia, 1020
aphasia, 1020
Amusia, 1020
Amyl nitrite, hyperemia of labyrinth from, 1000
Anastomosis of facial nerve with hypoglossal nerve, 995–999
of hypoglossal nerve with facial nerve, 995–999
Andrews' cannula, 195
Anemia of labyrinth, 1001
nose and, 28
Anesthesia in adenectomy, 340
cocaine, in indirect laryngoscopy, 549
in operations on tonsils, 418, 420, 429
for direct laryngoscopy, 587
nitrous oxide, in tonsillotomy, 436
of pharynx, 364
rectal, for laryngofissure, 563
Anesthesia in submucous resection of nasal septum, 82
in tonsillectomy, 417
for tracheoscopy, 581
for upper tracheobronchoscopy, 589
Anesthetic leprosy, 323
Aneurysm of arch of aorta, laryngeal paralysis and, 517, 520
diagnosis of, from retropharyngeal abscess, 359
laryngeal paralysis and, 522
of subclavian, laryngeal paralysis and, 517, 521
Aneurysmal cough in laryngeal paralysis, 519
Angina in diphtheria, 482
epiglottidea anterior, 447
lacunaris of pharyngeal tonsil, 331
laryngis, diagnosis of, from chronic laryngitis, 474
pseudomembranous, 400
Vincent's, 400
diagnosis of, 401
etiology of, 400
pathology of, 400
prognosis of, 401
symptoms of, 401
treatment of, 401
Angioma of auricle, 650
diagnosis of, 651
from othematoma, 651
electrolysis for, 651
Paquelin's cautery for, 651
symptoms of, 650
treatment of, 651
of nose, 278
galvanocautery for, 279
symptoms of, 278
treatment of, 278
of pharynx, 373
electrolysis in, 373
treatment of, 373
of tonsil, 442
electrolysis in, 442
Angioneurotic paralysis of auditory nerve, 1015
Ankylosis of ossicles in adhesive processes of middle ear, 728, 729
in deaf-mutism, 1028
defective hearing from, 631
tuberculous, of arytenoid cartilages, 505
Annulus tympanicus, 610
relation of, to facial nerve, 835
Anosmia, 253
obstruction of olfactory fissure and, 253
in sinuitis, 186
Antrum chisels, Corwin's, 227
of Highmore. *See* Sinus, maxillary.
Antrum saw, Vail's, 226
Aphasia, amnesic, 1020
Aphonia in acute catarrhal laryngitis, 449, 453
in laryngeal diphtheria, 483

Aphonia in laryngeal paralysis, 516, 519, 521
in membranous laryngitis, 460
in papilloma of larynx, 547
spastica, 509
in tuberculosis of larynx, 311
Apoplexy, laryngeal, 511
etiology of, 512
neurasthenia and, 512
symptoms of, 512
treatment of, 512
Aprosexia in adenoids, 337
defects of speech and, 538, 539
in fibroma of pharynx, 370
Aquæductus vestibuli in deaf-mutism, 1028
Aquiline or hump nose, 295
Arch, Gothic, 57
Arsenic paste for lupus, 305, 306
Arteriosclerosis, epistaxis and, 285
of labyrinth, 1005
Arthritis, catarrhal laryngitis and, 450
Articulation in deaf-mutism, 1029
Arytenoid cartilages, tuberculous ankylosis of, 505
"Ascending croup," 484
Asch-Mayer's operation for deviation of nasal septum, 77, 78, 79
Asch's septum forceps, 78
Aspergillus niger, 664
Asphyxia in edema of larynx, 462
in laryngeal diphtheria, 483
in operations for laryngeal carcinoma, 575
Asthenopia in disease of sinuses, 205
due to enlargement of middle turbinated bone, 35
Asthma, miller's, 455, 509. *See* also Laryngismus stridulus.
of nasal origin, 267
in obstructive deviations of nasal septum, 65
rachiticum, 509. *See* also Laryngismus stridulus.
thymic, 509. *See* also Laryngismus stridulus.
Ataxia, locomotor, laryngeal paralysis and, 521
Atrium of ear, outer wall of, removal of, in radical mastoid operation, 838
suppuration of, 781
Atrophic laryngitis, 471
rhinitis, 154
Attic of ear, caries of, 773
divisions of, 611, 612
external, acute inflammation of, 717, 718
outer wall of, removal of, in radical mastoid operation, 838
suppuration of, 607, 678, 781, 782
of nose, 254
Auditory apparatus, functional tests of, 618
Auricle, angioma of, 650
chondritis of, 601
cysts of, 652
dermatitis of, 656
epithelioma of, 652
fibroma of, 651
herpes of, 655
zoster of, 656
lesions of, defective hearing from, 630
malformations of, 645
neoplasms of, 648
nodular enlargements on, 652
perichondritis of, 601, 654
sarcoma of, 652
Aurophone in hyperostosis of bony capsule of labyrinth, 741
Auscultation, aural, 712
of tympanic cavity, 696, 697, 702
"Aussatz," 321. *See* Leprosy.
Autophony, 689
in chronic moist catarrhal otitis media, 720

B

Bacilli in upper respiratory tract, 113
Bacillus lepræ, 321
mallei, 323
pyocyaneus in membranous laryngitis, 460
of rhinoscleroma, 287
Bacteremia, thrombosis of lateral sinus and, 814, 815
Bacteriological diphtheria, 480
Ballance flap method in radical mastoid operation, 840, 854
incision in radical mastoid operation, 841, 843
Ballenger-Foster septum speculum, 97
Ballenger's complete operation on tonsil with right-angle knife and ecraseur, 417
curette, 246
ethmoid knives, 246
mastoid periosteal elevator, 826
modification of radical mastoid operation, 848
mucosa knife, 96
swivel knife, 105
nasal septum clamp, 293
operation for adenoids, 339
for complete exenteration of ethmoidal cells and middle turbinal, 242
upon ethmoidal sinuses, 239, 242
for fibromata of pharynx, 372
intranasal, for aquiline or hump nose, 296
upon frontal sinus, 207
for long or drooping nose, 297
for peritonsillitis, 407
for removal of ethmoidal cells, 239
upon sphenoidal sinuses, 251

Ballenger's operation on tonsils with scalpel, 423
for twisted or crooked nose, 291, 292, 293
reverse chisel, 297
right-angle knife, 417
septum gouge, 96
swivel cartilage knife, 96
turbinotome, 146
tonsil ecraseur, 421
forceps, 418
knife, 424
syringe, 441
turbinal knives, 240
turbinotome, 150, 152
vulsellum forceps, 418
Bane-Allport gauze packer, 758
Barany's fixation apparatus in vestibular nystagmus, 903
noise apparatus, 627
Beck's double osteoplastic operation upon frontal sinus, 219
mercury massage, 43
paraffin syringe, 299
Beckmann's serrated scissors, 145
Beck-West method of enucleation of tonsils, 426
Bellocq's postnasal tampon cannula, 145
Benzoin, compound tincture of, in treatment of chronic suppurative otitis media, 782
Bezold-Edelmann tuning forks, 617
Bezold's mastoiditis, 857, 859, 860
Bier's hyperemic treatment, 127
indications for, 128
in mastoiditis, 768, 769
technique of, 128
Bing's test for hearing, 628
Binnafont method of Eustachian catheterization, 695, 696
Birmingham nasal douche, 274
Bishop's trephine, 229
Bismuth, subnitrate of, powder, 50
Black diphtheria, 285, 483
Blennorrhea, Stoerk's, 286, 468
Blood count in thrombosis of lateral sinus, 814
cultures in thrombosis of lateral sinus, 813
spitting of, 472
Boeckmann-Stubbs' curette, 341
Boetcher's tonsil hemostat, 422
Bone marrow in diphtheria, 480
Bony capsule of labyrinth, hyperplasia of, 735
Boric acid powder in treatment of chronic suppurative otitis media, 781
Bosworth's operation for deviation of nasal septum, 70, 71, 72
saw, 71
Bougies in obstruction of Eustachian tube, 689, 690, 731
Bourguet's labyrinth operation, 968
Boyce's position for tracheoscopy, 581
Bradycardia in diphtheria, 481
Brain, abscess of, 799
acute suppurative otitis media and, 748
cholesteatoma and, 763
chronic otorrhea and, 752, 805
exploration of, indications for, 805
head pain in, 805
incisions for, 977
infection of tympanic cavity and, 608
irritability in, 805
prognosis of, 806
stages of, 801, 802, 804
stupor in, 805,
surgery of, 976
drainage through squamous plate in, 978
through tegmen tympani in, 976
symptoms of, 801
tegmen tympani in, perforation of, 801
treatment of, 806
vertigo in, 805
diseases of, deaf-mutism and, 1026
tumor of, deafness from, 1021
laryngeal paralysis and, 521
paralysis from, 1021
spasm of pharynx and, 367
Brandegee's adenoid forceps, 339, 341
"Bräune, häutige," 459
Breathing, inferior costal type of, 528
methods of, 527
Bright's disease, edema of larynx and, 462
Bronchi, diphtheria of, 484
foreign bodies in, 577
Bronchial diphtheria, 484
syncope, 511
Bronchitis, diphtheria and, 486, 487
Bronchopneumonia, diphtheria and, 487
Bronchoscopy, 581. *See* also Tracheoscopy.
Brophy vaporizer, 589
Buchanan's anesthetizing dosimetric attachment, 590
Bulb, jugular, surgery of, 989, 990, 991
Bulbar paralysis of pharynx, 365, 366
Bulla ethmoidalis, obstruction of nose due to, 119
Buttles-Pynchon inhaler, 686

C

"Cadaveric" position of vocal cords in laryngeal paralysis, 519, 521
Caisson workers, hemorrhage into labyrinth in, 1001
Calomel in acute laryngitis in children, 457
fumigations in membranous laryngitis, 462
Calcareous deposits in membrana tympani, 679, 788

Calculus of tonsils, 403
Caldwell-Luc's operation upon maxillary sinus, 231
Caloric nystagmus, 874
reactions in diffuse latent suppurative labyrinthitis, 950
test in acute diffuse serous labyrinthitis, 937
of vestibular apparatus, 881
Camphoroxol, treatment of chronic suppurative otitis media with, 782
Canal, frontonasal, 191, 192
Canaliculus carototympanici, 610
Cancer. *See* Carcinoma.
Canfield-Ballenger's antrum operation upon maxillary sinus, 233
Cannula, Andrews', 195
Bellocq's postnasal tampon, 145
Well's trocar, 229
Capsule, bony, of labyrinth, otosclerosis of, 735
hyperplasia of, 735
Capsulitis labyrinthii, 735
Carcinoma, inoperable, of upper respiratory tract, 377
laryngeal, 533, 553
age and, 554
cough in, 555
diagnosis of, 557
early, 553
from benign neoplasms, 557
from laryngitis, chronic, 557
syphilitic, 557
tuberculous, 557
from perichondritis, 557
from tuberculosis of larynx, 312
microscopic, 556
dysphagia in, 555
etiology of, 554
heredity and, 554
hoarseness in, 553, 555
laryngoscopy in, 556
lymphatic glands of neck in, involvement of, 555
operations for, asphyxia in, 575
cardiac reflexes in, 575
inspiration pneumonia and, 575
rectal alimentation and, 575
shock in, 574
sudden death after, 574
voice and, 575
pain in, 553, 555
pathology of, 555
prognosis of, 557, 558, 559
recurrence of, after operation, 576
sex and, 554
social standing and, 554
symptoms of, 555
tobacco and, 554
treatment of, 558, 576
of nose, 281
Carcinoma of nose, diagnosis of, 281
prognosis of, 281
treatment of, 281
of pharynx, diagnosis of, from actinomycosis of pharynx, 328
of subglottic region, diagnosis of, from chronic laryngitis, 473
of tonsils, 444
diagnosis of, from sarcoma, 445
symptoms of, 444
treatment of, 445
Cardiac infection in membranous laryngitis, 461
paralysis in diphtheria, 486
reflexes in operations for laryngeal carcinoma, 575
Caries of attic of ear, 773
of ossicles, defective hearing from, 631
of teeth, sinuitis and, 177
Carotid artery, external, excision of, 377
Carter's nasal splint, 292
Cartilage knife, Ballenger's swivel, 96
septal, reformation of, 101, 102
removal of, 91
Cartilaginous deviations of nasal septum, 60
meatus of ear, collapse of, defective hearing from, 630
Casselberry's operation for amputation of uvula, 358
position for feeding in intubation, 497, 498
Catarrh, epipharyngeal, in adenoids, 337
tubal, 684
Catarrhal diphtheria, 480
lingual tonsillitis, acute, 349
otitis media, acute, 703
chronic moist, 718
pharyngitis, simple acute, 352
sinuitis, treatment of, 190
Catching cold, 396, 449, 450
Catheterization, Eustachian, 695
diagnostic value of, 698
methods of, 695, 696
Binnafont, 695, 696
Kramer, 695, 696
Lowenberg, 696
through mouth, 698
through opposite nasal cavity, 697
therapeutic value of, 698
inflation of tympanic cavity by, 695
Caustics, chemical, 53
Cauterization, submucous, for chronic rhinitis, 141
Cautery for soft hypertrophies of nasal septum, 70
Cavernous sinus, thrombosis of, 817
Cavum tympani. *See* Tympanic cavity.
Cells of Kirschner, 854
Cellulitis, cervical, tonsillectomy and, 392
ethmoid operation and, 247
Central paralysis of pharynx, 366
Cerebellar abscess, surgery of, 982

Cerebellar nystagmus, 872
Cerebral abscess, surgery of, 976
hemorrhage, accessory nasal sinuses and, 187
paralysis in diphtheria, 479
Cerebrospinal fluid in middle ear, 670
rhinorrhea, 265, 266
Cerumen, impacted, 642
diagnosis of, 643
etiology of, 642
nervous cough from, 511
prognosis of, 643
symptoms of, 642
treatment of, 643, 644
Weber's test for, 643
inspissated, defective hearing from, 630
Ceruminous plugs, dizziness from, 643
improper cleansing of ear and, 642
Cervical cellulitis, tonsillectomy and, 392
glands, removal of, 361, 362
suppuration of, 398
tuberculous, 316
Chaleway's spokeshave, 72
Cheyne-Stokes respiration in leptomeningitis, 795
Chilblain, 657
Child-crowing, 509. *See* also Laryngismus stridulus.
Chimani-Moss test for simulated deafness, 1014
Chisel, Ballenger's reverse, 297
Corwin's antrum, 227
Choanæ, adhesions around, 371
Cholesteatoma, 761
abscess of brain and, 763
defective hearing from, 630
diagnosis of, 763
etiology of, 761
extradural abscess and, 792
primary, 761
prognosis of, 763
secondary, 761
sequelæ of, 765
sinus thrombosis and, 763
symptoms of, 762
treatment of, 763
Cholesterin in chronic lacunar tonsillitis, 402
Chondritis of auricle, 601
Chondroma of subglottic space, 546
Chorditis nodosa, 469, 498, 544, 545
etiology of, 470
massage for, 471
pathology of, 470
prognosis of, 470
symptoms of, 470
treatment of, 471
voice in, 470
tuberosa, 469
vocalis hypertrophica inferior, 468
Chorea, laryngeal, 510
Chromic acid method, Goldstein's, of submucous cauterizatiou, 141
Cigarette drain, 362, 378
Circumscribed labyrinthitis, nystagmus in, 904
Cleft palate, singing voice and, 532
Clergyman's sore throat, 353, 531. *See* also Pharyngitis, chronic.
Club foot, adenoids and, 347
Cocaine anesthesia in indirect laryngoscopy, 549
in operations on tonsils, 418, 420, 429
in epistaxis, 285
in tuberculosis of larynx, 313
function of, 613
hyperostosis of, 739
Codeine for cough in acute catarrhal laryngitis, 455
laryngitis in children, 458
in tuberculosis of larynx, 312
Cold, dermatitis of auricle due to, 657
Collapse of alæ nasi, 302
Colloid degeneration of nose, 287
Collodion dressing, Pischel's, 144
Compsomyia macellaria, 636
Concussion of labyrinth, 1010
Condyloma of epiglottis, 319
Consanguinity, deaf-mutism and, 1025
Convulsions in abscess of brain, 803
in acute suppurative otitis media in children, 751
Cooper-Hewitt light for tuberculosis of larynx, 313
Cooper's operation upon maxillary sinus, 230
Corona veneris, 33
Corti's cells, 612, 613
Corwin's antrum chisels, 227
operation upon maxillary sinus, 227
Coryza, acute, 130
edematosa, 264
pus in, 160
sinuitis and, 188
syphilitic, 33
Cough in acute catarrhal laryngitis, 453
laryngitis in children, 455, 456
aneurysmal, in laryngeal paralysis, 519
in atrophic laryngitis, 471
in chronic pharyngitis, 354
in diphtheritic paralysis, 486
in discrete hypertrophic laryngitis, 468
from foreign body in respiratory passages, 577
in hyperkeratosis of tonsil, 409
in laryngeal carcinoma, 555
diphtheria, 483
paralysis, 519
in membranous laryngitis, 460
nervous, 511
in papilloma of tonsil, 442
in phlegmonous laryngitis, 458
reflex, in elongated uvula, 356
in papillomata of larynx, 368
spasmodic laryngeal, 510

Cough in syphilitic stenosis of larynx, 503
Counterirritation, 122
Cretinism, nasal deformity in, 298
Cricoid cartilage, perichondritis of, 457
Cristæ ampullares, 870, 876
Croup, 459. *See* also Laryngitis, membranous.
"ascending," 484
diphtheritic, 483. *See* also Diphtheria, laryngeal.
false, 455, 509. *See* also Laryngismus stridulus.
idiopathic membranous, 459
membranous, 483. *See* also Diphtheria, laryngeal.
pseudomembranous, 459
diagnosis of, from acute laryngitis in children, 457
silent, 460
true, 483. *See* also Diphtheria, laryngeal.
Croupous inflammation of external auditory meatus, 661
laryngitis, 459
Cryptic tonsillitis, 396
Curettage in chronic suppurative otitis media, 760
of tympanic cavity, dangers of, 836
Curette, Ballenger's, 244
Cyanotic congestion, atrophic rhinitis and, 154
engorgement, atrophic rhinitis due to, 155
Cystoma of pharynx, 369
of tonsils, 443
Cysts of auricle, 652
of subglottic space, 546

D

Deaf-mutism, 537, 1023
adenoids and, 1030, 1031
age and, 1026
alcoholism and, 1025
aquæductus vestibuli in, 1028
ankylosis of ossicles in, 1028
articulation in, 1029
brain diseases and, 1026
classification of, 1024
consanguinity and, 1025
deafness in, 1028
death rate in, 1026
diagnosis of, 1031
epilepsy and, 1025
etiology of, 1024
Eustachian tubes in, 1028
hearing in, 1028
hemophilia and, 1025
heredity and, 1025
idiocy and, 1031
infectious diseases and, 1026
labyrinth in, 1028
lungs in, 1030
mental training in, 542
Deaf-mutism, otorrhœa in, 1030
pathology of, 1027
prognosis of, 1031
sequelæ of, 1030
smallpox and, 1027
speech in, 542, 1029
symptoms of, 1028
syphilis and, 1025
testing for, 1029
for "islands of hearing" in, 617
tinnitus in, 1030
treatment of, 1031, 1032
tuberculosis and, 1030
voice in, 1029
Deafness in acute diffuse suppurative manifest labyrinthitis, 942
suppurative otitis media and, 745, 747
adhesive processes in middle ear and, 727
in arteriosclerosis of labyrinth, 1006
chronic moist catarrhal otitis media and, 718, 719
in concussion of labyrinth, 1011
diffuse latent suppurative labyrinthitis, 949
hysterical, diagnosis of, from arteriosclerosis of labyrinth, 1006
islands of, 739
leukemic, 1006
locomotor ataxia and, 1022
in Ménière's disease, 1002
occupation, 1012
perforation of membrana tympani and, 673
physiological law of, 607
simulated, 1013
tests for, 1013, 1014
Chimani-Moos, 1014
Erhard, 1014
stethoscope, 1014
in syphilis of internal ear, 1007, 1008
syphilitic condyloma of external ear and, 321
tumor of brain and, 1021
"Decapitation" of tonsils, 392
Defective hearing, 630
Defects of speech, 536
Deformities of nose, correction of, 289
Delstanche's masseur, 40, 42, 682, 717
rarefacteur, 1005
ring knife, 821
Dementia, othematoma and, 648
Denker's operation upon maxillary sinus, 233
Dermatitis of auricle, 656
due to cold, 657
etiology of, 657
symptoms of, 657
treatment of, 657
treatment of, 656
congelationis auricula, 657
Deviations of nasal septum, 57
De Vilbiss' spray tubes, 44

Diabetes, edema of larynx and, 462
hemorrhage into labyrinth in, 1001
hemorrhagic laryngitis and, 472
otitis media and, 710, 744
Diaphragmatic paralysis in diphtheria, 486
Diffused inflammation of external auditory meatus, 659
laryngitis, chronic, 466
"Dip" of postsuperior wall in simple acute mastoiditis, 766
Diphtheria, 474
adenopathy and, 485
age and, 475
albuminuria in, 485
alimentary canal in, 480
angina in, 482
antitoxin, administration of, 489
albuminuria and, 489
dosage of, 489
effect of, on pseudomembrane, 489
on temperature, 489
immunization by, 488
indications for, 489
injection of, place of, 489
in laryngeal cases, 489
paralysis and, 489
value of, 488
bacteriology of, 476
black, 285, 483
blood-pressure in, 481
bone marrow in, 480
bradycardia in, 481
bronchial, 484
bronchitis and, 486, 487
bronchopneumonia and, 487
catarrhal, 480
complications of, 485
diagnosis of, 485
bacteriological, 477
from acute lacunar tonsillitis, 398
laryngitis in children, 456
from membranous laryngitis, 461
of ear, 484
epistaxis and, 284
of esophagus, 485
etiology of, 474
fruste, 480
gangrenous, 479, 481
glycosuria and, 480
heart in, 479, 486
histopathology of, 478
hyperemia of labyrinth and, 1000
hyperleukocytosis in, 486
infection in, mixed, 478
modes of, 475, 476
intubation in, 485, 486, 492
kidneys in, 480
Klebs-Loeffler bacilli in, 478
laryngeal, 483
antitoxin in, 489
Diphtheria, laryngeal, aphonia in, 483
asphyxia in, 483
cough in, 483
hoarseness in, 483
invasion in, 483
mixed, 484
paralysis and, 515, 520
phlegmonous, 484
respiration in, 483
septic, 484
spasm in, 483
stenosis of larynx in, 483
voice in, 483
"le con proconsulair" symptom in, 485
lesions in spinal cord due to, 479
liver in, 480
lungs in, 479
lymphatic glands in, 480
mixed, 481
mucous membrane in, sloughing of, 479
muscles in, 480
nasal, 481, 482, 485
Neisser-Ernst bodies in, 477
nervous system in, 479, 486
of nose, 482
obstruction of Eustachian tube and, 689
pancreas in, 480
paralysis in, cardiac, 486
cerebral, 479
cough in, 486
diaphragmatic, 486
postdiphtheritic, 486
diffused, 486
general, 486
vagus, 486
of velum palati, 486
phlegmonous, 481
polar granules in, 477
prognosis of, 485
prophylaxis of, 487
pseudodiphtheria bacilli in, 478
pseudomembrane in, 478
"pseudotabes" and, 486
pulse in, 481
race and, 474
septic, 481
septicemia and, 481
sequelæ of, 485
skin in, 486
spleen in, 480
of stomach, 485
stricture of external auditory meatus and, 663
symptomatology of, 481
tachycardia in, 481
temperature in, 481
thymus gland in, 476
of trachea, 484, 578
tracheotomy in, 486, 490
treatment of, 485
alcohol, 488
antitoxin, 488

Diphtheria, treatment of, general, 488
local, 488
surgical, 490
types of, 480
urine in, 485
Diphtheritic croup, 483
laryngitis, 459
paralysis of pharynx, 366
"pseudotabes," 486
Diplacusis, 1017
acoustica in concussion of labyrinth, 1011
binauralis or disharmonica, 615
Direct laryngoscopy, 546, 549, 587
Discrete hypertrophic laryngitis, 468
Disharmony, destruction, in acute diffuse serous labyrinthitis, 938
suppurative manifest labyrinthitis, 941
stimulation in acute diffuse suppurative manifest labyrinthitis, 941
Dislocated nose, 294
Diverticulum of hypopharynx, 595
Dizziness from ceruminous plugs, 643
in hyperesthetic rhinitis, 255
from irrigation of ear, 644
in Ménière's disease, 1002
in meningitis serosa, 790
in obstructive deviations of nasal septum, 63
in syphilis of internal ear, 1007
Dobel's solution, 549
Douche, Birmingham nasal, 274
Dry catarrh of middle ear, 724
chronic rhinitis, 154
gauze treatment of chronic suppurative otitis media, 781
Dunbar's serum in treatment of hyperesthetic rhinitis, 262
Dyscrasias, constitutional, acute rhinitis complicating, 130
Dysphagia in carcinoma of larynx, 555
in laryngeal carcinoma, 555
in paralysis of pharynx complicating facial paralysis, 367
in tuberculosis of larynx, 308
Dyspnea in acute infectious epiglottitis, 448
phlegmonous laryngitis, 458
in atrophic laryngitis, 471
in discrete hypertrophic laryngitis, 468, 469
in edema of larynx, 463
in epiglottitis, 448, 449
in laryngeal paralysis, 519, 521, 522
in membranous laryngitis, 460
in miasmatic epiglottitis, 449
in papilloma of larynx, 547, 548
in syphilitic stenosis of larynx, 503
in tuberculosis of larynx, 312

E

Ear, auricle of. *See* Auricle.
ceruminous plugs in, 642
Ear, clinical anatomy of, 601
diphtheria of, 484
external, 601
clinical anatomy of, 601
eczema of, 665, 666
syphilis of, 320, 321
foreign bodies in, 635, 636
general medicine and, 26
improper cleansing of, ceruminous plugs and, 660
internal, neoplasms of, 1021
syphilis of, 1007
irrigation of, dizziness from, 644
middle, actinomycosis of, 328, 329
adhesive processes in, 603, 724
ankylosis of ossicles in, 728, 729
course of, 727
deafness from, 727
diagnosis of, 728
etiology of, 725
hearing in, 727
hyperesthesia acoustica in, 727
incision of membrana tympani in, 678
inflation of tympanic cavity in, 728, 729
massage in, 730
membrana tympani in, 726
paracusis Willisii in, 727
pathology of, 725
Rinné test in, 728
symptoms of, 728
tinnitus in, 727
treatment of, 729
affections of, defective hearing from, 630
arteries of, 610
catarrh of, chronic moist, 718, 725
cerebrospinal fluid in, 670
clinical anatomy of, 602
disease of, Eustachian tube and, 683
dry catarrh of, 724
foreign bodies in, 641
proliferous inflammation of, 724
sclerosis of, 724
suppuration of, incision of membrana tympani in, 678, 679
tuberculosis of, 314
muscles of, intrinsic, 612
ossicles of, 607
physiology of, 610
relations of, to nose and throat, 31
in tabes dorsalis, 32
Eardrum. *See* Membrana tympani.
Eczema of external ear, acute, 665
chronic, 666, 667
subacute, 666

Edema, angioneurotic, acute, diagnosis of, from acute infectious epiglottitis, 448
of bronchial mucous membrane in membranous laryngitis, 461
chronic, in syphilitic stenosis of larynx, 503
glottidis, 462
of larynx, 462
asphyxia in, 462
voice in, 463
of nose, acute circumscribed, 264
of uvula, 355
Edlemann-Bezold forks, 739
Elastic uvula, 357
Electrocautery, 43
in angioma of nose, 279
in chronic pharyngitis, 355
rhinitis, 139
in epistaxis, 285
in fibroma of pharynx, 372
in hyperkeratosis of tonsil, 413
in hypertrophic rhinitis, 144
incision of membrana tympani with, 680
in lingual varix, 351
in lupus of nose, 306
for neoplasms of subglottic space, 546
for removal of foreign bodies in ear, 641
Electrolysis in actinomycosis of pharynx, 328
in angioma of auricle, 651
of pharynx, 373
of tonsil, 441
in obstruction of Eustachian tube, 689
in pachydermia laryngis, 500
in papilloma of pharynx, 368
Elephantiasis græcorum, 321
Elongation of uvula, 356
Elevators, Hajek-Ballenger mucoperichondria, 96
for submucous resection of nasal septum, 85, 86, 87, 96
Embolic abscesses, thrombosis of ear and, 810
Emphysema of orbital tissues, ethmoid operation and, 248
Empyema of accessory sinuses, 169
nasal secretion in, 31
of ethmoidal cells, 148
in sinuitis, 169
Encephalitis, hyperemia of labyrinth and, 1000
Encephaloscope, Whiting's, 981
Enchondrosis of laryngeal cartilages, diagnosis of, from chronic laryngitis, 473
Endocarditis, infection through tonsils and, 382, 391, 398
Endolaryngeal operations, 560
Endolymph, 613
Enteritis, metastatic, in thrombosis of lateral sinus, 812
Enucleation of tonsils, Beck-West method of, 426
Epiglottis, condylomata of, 319
diseases of, inflammatory, 447
Epiglottitis, dyspnea in, 448, 449
infectious, acute, 447
diagnosis of, 448
from acute angioneurotic edema, 448
dyspnea in, 448
etiology of, 447
pathology of, 448
prognosis of, 448
Staphylococcus albus in, 448
Streptococcus aureus in, 448
symptoms of, 448
syphilis and, 448
tracheotomy in, 449
treatment of, 448
tuberculosis and, 448
miasmatic, 449
dyspnea in, 449
etiology of, 449
symptoms of, 449
Epilepsy, deaf-mutism and, 1025
of nasal origin, 267
Epipharyngeal catarrh in adenoids, 337
diseases, acute catarrhal otitis media and, 708
"dropping," 65
inflammation, adenoids and, 345
space, variations in, 344, 345
tonsils, 349
Epipharyngitis, 467, 510
in acute laryngitis in children, 455
chronic suppurative otitis media and, 758
curettage of Eustachian tube and, 838, 839
obstruction of Eustachian tube and, 689
tubal catarrh and, 684
Epipharynx, adenoids in, 337
adhesions in, 363
affections of, defective hearing from, 633, 634
crusts in, significance of, 168, 173
diseases of, 331
osteoma of, 280
tumors of, fibrous, 338
Epistaxis, 284
adrenalin in, 285
arteriosclerosis and, 285
cocaine in, 285
deviation of nasal septum and, 66
diphtheria and, 284
electrocautery in, 285
etiology of, 284
hemophilia and, 285
treatment of, 285
typhoid and, 284
Epithelioma of auricle, 652
Equinia maliasmus, 323

Erectile tissue of nose, 18
tumors of pharynx, 373
Erhard test for simulated deafness, 1014
Erysipelas, acute phlegmonous laryngitis and, 458
of face, cerebellar nystagmus and, 873
of larynx, 463
Erythema multiforme, acute lacunar tonsillitis and, 399
nodosum, acute lacunar tonsillitis and, 399
Escat's position, 170
Esophagoscopy, 597
Esophagus, diphtheria of, 485
foreign bodies in, 577, 596
paralysis of, 367
strictures of, 596
Ethmoid disease, exophthalmos in, 168
knives, Ballenger's, 244
perpendicular plate of, removal of, in submucous resection of nasal septum, 94
Ethmoidal cells. *See* also Sinuses, ethmoidal.
empyema of, 148
and middle turbinal, Ballenger's operation for complete exenteration of, 242
removal of, Ballenger's operation for, 239
partial, 239
sinuses, 164, 167
Ethmoiditis, acute catarrhal otitis media and, 708
atrophic laryngitis and, 471
chronic moist catarrhal otitis media and, 722
Eustachian "tonsil," 633, 684
tubes, adenoids and, 346
affections of, defective hearing and, 632
catheterization of, 695
clinical anatomy of, 603
closure of, 602
curettage of, in epipharyngitis, 838, 839
in radical mastoid operation, 838
in deaf-mutism, 1028
diseases of, 683
foreign bodies in, 641
function of, 611
infection through, 316, 683, 704
inflammation of, 684
inflation of, 606
massage of, 691
mastoiditis and, 687
obstruction of, 688
bougies in, 689, 690, 731
complete, 688
diagnosis of, 688
dilatation for, 689, 690
diphtheria and, 689
effect of, 602, 604
Eustachian tubes, obstruction of, electrolysis for, 689
epipharyngitis and, 689
etiology of, 688
partial, 688
treatment of, 689, 690, 691
patency of, 688
treatment of, 689, 690, 691
relation of, to hearing, 683
to middle-ear disease, 683
stenosis of, 731
tonsils and, 415
Ewald's law, 875
Examination lamp, 39
Exanthemata, sinus thrombosis and, 746
Exenteration of mastoid process, landmarks after, 835
Exophthalmos in ethmoid disease, 168
Exostosis of external auditory meatus, 661
Extradural abscess, 792
chronic otorrhea and, 792
cholesteatoma and, 792
etiology of, 792
location of, 793
mastoiditis and, 792
prognosis of, 793
symptoms of, 792
treatment of, 793
surgical, 983
vertigo in, 793
Extranasal operations upon maxillary sinus, 232
Extubation, 495
Eye, diseases of, due to nasal lesions, 34, 35
relation of, to nose, throat, and ear, 30, 31

F

Face, adenoid, 339, 345
erysipelas of, cerebellar nystagmus and, 873
Facial paralysis, 993
complicating pharyngeal paralysis, 367
False croup, 455, 509. *See* also Laryngismus stridulus.
Falsetto voice, 540
Faradism in diphtheritic paralysis, 367
for laryngeal paralysis, 520
Farcy, 323
Farlow's tonsil punch forceps, 427
Fauces, affections of, defective hearing from, 633, 634
anatomical landmarks of, 425
diseases of, 379
inflammatory, 352
pillars of, voice and, 530
syphilis of, 317
tuberculosis of, 367
Fenestra cochlea. *See* Round window.
vestibuli. *See* Oval window.

Ferguson-Pynchon mouth-gag, 340
Fetterolf's file saw, 80
Fibro-enchondroma of tonsils, 443
Fibroma of auricle, 651
treatment of, 651
of nose, 276
treatment of, 276
operative, 276
von Langenbeck's, 277
of pharynx, 370
aprosexia in, 370
Ballenger's operation for, 372
diagnosis of, 371
from sarcoma of pharynx, 371
electrocautery in, 372
etiology of, 370
"frog face" in, 370
prognosis of, 372
symptoms of, 370
treatment of, 372
voice in, 370
of tonsils, 442
treatment of, with snare, 443
Finger dissection of tonsils, 442
Finsen light, 125, 313
Fistula in auris congenita, 646
in circumscribed labyrinthitis, 930
in diffuse latent suppurative labyrinthitis, 950
of labyrinth, 648
nystagmus, 874
postauricular, closure of, 992
by the Mosetig-Moorhof method, 992
by the Passow-Trautmann method, 992
salivary, 378
test of vestibular apparatus, 888
Flourens' law, 874
Follicular inflammation of external auditory apparatus, 657
tonsillitis, acute, 396
Foramen, stylomastoid, 995
Forceps, Asch's septum, 78
Foster-Ballenger perpendicular bone, 96
Ostrum's forward cutting, 228
Foreign bodies, defective hearing from, 630
in ear, 635, 636
animated, 640
removal of, 636
by the agglutination method, 637, 638
by electrocautery, 641
by forceps, 638
by foreign body hook, 638
by postauricular incision, 639
by syringing, 637
in tracheobronchoscopy, upper, 593
in tracheoscopy, 484
Foreign bodies in ear, removal of, by Voltolini's method, 641
in esophagus, 577, 596
in Eustachian tube, 641
in larynx, 577
in middle ear, 641
in nose, 289
in respiratory passages, 577
cough from, 577
etiology of, 577
pneumonia and, 578
removal of, 578
skiagraphy for, 578,586
symptoms of, 577
tracheotomy in, 579
treatment of, 578
voice and, 577
spasm of pharynx and, 367
in trachea, 577
Fossa, Rosenmüller's, 606, 684, 685, 696
supratonsillar, 384, 391, 418
Fossula fenestræ cochleæ, 609, 610
Fracture of malleus, 670
of ossicles, 670
Freer's operation for deviations of nasal septum, 100, 101
"Frog face" in fibroma of pharynx, 370
Frontal-ethmoid operation, 247
Frontal sinus, 161
Frontonasal canal, 191, 192
opening of, variations in, 64
probing of, 192
Frostbite, 657
Fuchsinophiles, Russell's, 287
Function tests of auditory apparatus, 618
of hearing, 616
of vestibular apparatus, 877
Furunculosis of external auditory meatus, 657
after-treatment of, 659
course of, 658
etiology of, 657
hearing in, 658
symptoms of, 658
treatment of, 658
of nose, 288
etiology of, 288
symptoms of, 288
treatment of, 289

G

Galton-Edelmann whistle, 626
Galvanic nystagmus, 874
test of vestibular apparatus, 891
Galvanism in degeneration of nerves, 366
in paralysis, chronic bulbar, 366
diphtheritic, 367
hysterical, of auditory nerve, 1016
laryngeal, 516, 520
Galvanocautery. *See* Electrocautery.
Ganglion, geniculate, 367
sphenopalatine, 19, 20

Gangrene, pulmonary, infection through tonsils and, 382
Gangrenous diphtheria, 479, 481
Gargling, von Tröltsch method of, 686
Gavage in intubation, 497
Gellé's test for hearing, 628
Germ centres of Goodsir, 334
of tonsils, 384
Giddiness in circumscribed labyrinthitis, 932
Glanders, 323
diagnosis of, mallein in, 324
etiology of, 323
of larynx, 325
of nose, 323
pathology of, 324
of pharynx, 325
prognosis of, 325
treatment of, 325
Gleason's operation for deviation of nasal septum, 75, 76, 77,
Globus hystericus, hyperesthesia of pharynx and, 364
spasm of pharynx and, 367
Glossodynia in lingual varix, 351
Glosso-epiglottic ligament, 348
Glosso-epiglottidian folds, 408
Glottic spasm, complete, in adult, 511
Glottis, spasm of, 509. *See* also Laryngismus stridulus.
Glycosuria, diphtheria and, 480
Goitre, hyperemia of labyrinth and, 1000
laryngeal paralysis and, 515, 518
nasal disease and, 268
Goldsmith's operation for perforation of nasal septum, 109
Goldstein's chromic acid method of submucous cauterization, 141
plastic flap operation for perforation of nasal septum, 104
Good's operation upon frontal sinus, rasps and chisels, 212, 213
Goodsir, germ centres of, 334
Gothic arch, 57
adenoids and, 345
Gouge, Ballenger's septum, 96
Hajek's, 94, 96
Gout, hyperostosis of external auditory meatus and, 662
Granular pharyngitis, 353. *See* also Pharyngitis, chronic.
Granuloma, chronic, of nose, throat, and ear, 304
syphilitic, diagnosis of, from adenoids, 339
tuberculous, diagnosis of, from adenoids, 339
Graves' disease, nasal disease and, 268
Gustatory functions of nose, 25

H

"Hairy pharyngeal polypi," 369
Hajek-Ballenger mucoperichondria elevators, 96
Hajek-Luc's operation upon frontal sinus, 216
Hajek's gouge, 94, 96
hand burr, 854
incision in submucous resection of nasal septum, 84, 90
Halle's frontal sinus drills, 211
operation upon frontal sinus, 210
Hallucinations in arteriosclerosis of labyrinth, 1005
Halsted's subdermal suture, 220
"Häutige bräune," 459
Hawley's spray tubes, 44, 45
Hay fever, 254. *See* also Rhinitis, hyperesthetic.
Hazletine's plastic operation for perforation of nasal septum, 105, 106
Headache in acute diffuse suppurative manifest labyrinthitis, 945
Head lamp, Kierstein's, 39, 40, 579, 589
pain in abscess of brain, 801
Hearing in adhesive processes of middle ear, 727
in deaf-mutism, 1028
defective, 630
adenoids and, 337, 633, 634
affections of epipharynx and, 633, 634
of Eustachian tubes and, 632
of external meatus and, 630
of fauces and, 633, 634
of labyrinth and, 634
of mastoid and, 634
of membrana tympani and, 630
of middle ear and, 631
ankylosis of ossicles and, 631
of foot-plate of stapes and, 631, 632
caries of ossicles and, 631
cholesteatoma and, 630
collapse of cartilaginous meatus of ear and, 630
foreign bodies and, 630
hypertrophy of tonsils and, 634
infections and, 633
inspissated cerumen and, 630
intracranial tumor and, 1020
lesions of auricle and, 630
myringitis and, 631
polypi and, 631, 632
functional tests of, 616
in furunculosis of external auditory meatus, 658
hyperacuteness of, 1016
alcohol and, 1016
neurasthenia and, 1016
tobacco and, 1016
islands of, 617
testing of deaf-mutes for, 617
in myringitis, 672
neuroses of, 1016
paths through which sound waves reach the labyrinth, 616

Hearing, physiological facts of, 616
range of, 616
relation of Eustachian tubes to, 683
tests, Bing's, 628
functional, 616
Gellé's, 628
monochord in, 626
Rinné's, 623, 624
Schwabach's, 622
voice, 619
watch, 620
Weber's, 621, 622
of voices, 1019
Heart in diphtheria, 479
Heath's operation, 833
Hematoma, paraffin injection and, 300
Hemicrania, hyperesthesia acoustica in, 1018
Hemilaryngectomy, 558, 560, 562, 566, 567
Hemophilia, deaf-mutism and, 1025
epistaxis and, 285
hemorrhagic laryngitis and, 472
operations on tonsil and, 416
Hemoptysis, spurious, 472
Hemorrhage, cerebral, accessory nasal sinuses and, 187
deviation of nasal septum and, 66
into labyrinth, 1001
laryngeal, 472
nasal, 284
Hemorrhagic inflammation of external auditory meatus, 660
laryngitis, 472
Heredity, nasal inflammation and, 115
Herpes of auricle, 655
symptoms of, 655
treatment of, 655
zoster of auricle, 656
treatment of, 656
of membrana tympani, 656
High obstruction of nose, 119
tracheotomy, 490
Highmore, antrum of. *See* Sinus, maxillary.
Hillis' position for feeding in intubation, 497, 499
Hinsberg's labyrinth operation, 965
steps of, 865-968
Hoarseness in acute catarrhal laryngitis, 449, 453
in atrophic laryngitis, 471
in chronic pharyngitis, 354
in laryngeal carcinoma, 553, 555
diphtheria, 483
paralysis, 516, 521
in papilloma of larynx, 547
Hodgkin's disease, lymphadenoma of tonsils in, 192
Hoegyes' law, 874
Holmes' malleable frontal sinus probe, 192
scissors, 150, 151
Horny material in tonsils, 409
Hurd's bone septum forceps, 97
Hydrophobia, spasm of pharynx in, 367
Hydrops laryngis, 453
Hydrorrhea, nasal, 265
symptoms of, 265
treatment of, 266
Hyperacuteness of hearing, 1016
Hyperemia of labyrinth, 1000
Hyperemic laryngitis, 464
Hyperesthesia acoustica, 670, 689, 1018
in adhesive processes of middle ear, 727
in concussion of labyrinth, 1011
in hemicrania, 1018
in trigeminal neuralgia, 1018
of pharynx, 364
Hyperesthetic rhinitis, 254
Hyperkeratosis of tonsil, 408
cough in, 409
electrocautery in, 413
Hyperkinesis of pharynx, 365, 367
Hyperleukocytosis in diphtheria, 486
Hyperosmia, 253
from irritation of olfactory lobe, 253
Hyperostosis of bony capsule of labyrinth, 720
of cochlea, 739
diffused inflammation of external auditory meatus and, 660
of external auditory meatus, 661
etiology of, 661
gout and, 662
symptoms of, 662
syphilis and, 662
treatment of, 662
of oval window, 739
of semicircular canals, 740
of stapes, 739
Hyperplasia of bony capsule of labyrinth, 735
of tonsil, 407, 408
Hyperplastic rhinitis, 142, 147, 267
Hypersensitiveness of pharynx, 364
Hypertrophic laryngitis, chronic, 464
localized, 468
ozena, 142
rhinitis, 142
Hypertrophy of larynx, 499, 502
of tonsils, 407, 408
Hypopharynx, diverticulum of, 595
pouch of, 595
Hyposmia, 253
Hysteria, sensory neuroses of pharynx and, 364
upper respiratory tract and, 28
Hysterical paralysis of auditory nerve, 1016

I

Ichthyol solutions, 50
Idiocy, deaf-mutism and, 1031
Impacted cerumen, 642
Incision of membrana tympani, 674, 675, 676
Incus, attachment of, 606

Incus, destruction of, in chronic mastoiditis, 771
removal of, 609
in ossiculectomy, 821, 822
in radical mastoid operation, 836, 837
Indirect laryngoscopy, 546, 549
Infectious diseases, membranous laryngitis in, 459
epiglottitis, acute, 447
fevers, acute suppurative otitis media and, 742, 746, 750
Infective tonsillitis, 396
Inflammation, 110
acute, 110
microörganisms in, role of, 122
mucous surfaces and, 111
nasal, adenoids and, 115
causes of, exciting, 113
extranasal, 113
intranasal, 115
predisposing, 113
chronic, 112
clothing and, 114
due to obstruction, 115
exposure and, 114
heredity and, 115
reaction of, 110
promotion of, 122
by Bier's hyperemic treatment, 127
by counterirritation, 122
by irrigation, 124
by lavage, 124
by leeching, 123
by leukodescent light, 125
by massage, 124
opsonic index and, 127
by pneumomassage, 125
by poulticing, 123
by wet cupping, 123
types of, 111
Inflation of tympanic cavity, 692
Infundibulum, 191
drainage of, 18, 118, 119, 120
Ingals' operation upon frontal sinus, 214
Inspiration pneumonia, operations for laryngeal carcinoma and, 575
Inspissated cerumen, defective hearing from, 630
Insufflator, powder, 50
Internal secretion in tonsils, 392
Intracranial complications in chronic mastoiditis, 771
in disease of sinuses, 207
lesions, acute simple mastoiditis without, 765
nystagmus, 910
pyogenic diseases of otitic origin, 789
tumor, defective hearing from, 1020
Intradural abscess, 794
Intralaryngeal laryngotomy, 546
Intranasal operations upon maxillary sinus, 224
Introitus esophagi, 596
Intubation in acute catarrhal laryngitis, 454, 455
laryngitis in children, 58
complications of, 496
in diphtheria, 485, 486, 492
feeding in, 497
Casselberry's position for, 497, 498
gavage in, 497
Hillis' position for, 497, 499
indications for, 454, 455, 458, 460, 493
instruments, O'Dwyer's, 493
for laryngeal paralysis, 524
membranous laryngitis, 460
position for, 493, 494, 495
rectal alimentation in, 499
removal of tube in, 495, 496
technique of, 493
Iodonucleoid in syphilitic stenosis of larynx, 503
Irritation, reflex, spasm of larynx from, 598
Ischemia, solutions producing, 56
Islands of hearing, 617

J

Jackson's exhaust pump, 583
extractor, 586
forceps, 593
safety-pin forceps, 592
self-illuminating tracheobroncho-scope, 591
tube spatula, 588
slide speculum, 580
tube, 578, 579, 580
Jansen's mastoid retractor, 858
rongeur forceps, 859
Jansen-Neumann labyrinth operation, 957
closure of wound in, 964
preliminary measures in, 957
steps of, 957–964
Jansen-Stacke incision in radical mastoid operation, 846, 847
Jugular bulb, surgery of, 989, 990, 991
thrombosis of, 816
vein, internal, resection of, 987, 988

K

Keratosis obturans, 644
Kidneys in diphtheria, 480
Kierstein's head lamp, 39, 40, 579, 580, 589
Killian's incision in submucous resection of nasal septum, 84, 85, 90
operation upon frontal sinus, 221
tube, 578, 579
Kirschner, cells of, 854

Klebs-Loeffler bacillus, 461, 474, 476, 478, 480, 481, 483
diagnosis of, from staphylococcus, 478
from streptococcus, 478
Neisser's stain for, 477
Koch's tuberculin in tuberculosis of middle ear, 317
Kramer, method of Eustachian catheterization, 695, 696
Krause-Heryng laryngeal forceps, 550
Krause's nasal snare, 149
trocar, 225
Krönlein's landmarks, 979
Kuhnt-Luc's operation upon frontal sinus, 217
Kuhnt's operation upon frontal sinus, 217
Kuster's operation upon maxillary sinus, 231
osteoplastic, upon frontal sinus, 218
Kyle's crypt knife, 420
malleable tubes, 80
operation for deviations of nasal septum, 80

L

Labyrinth, affections of, defective hearing from, 551
anemia of, 1001
etiology of, 1001
symptoms of, 1001
treatment of, 1001
arteriosclerosis of, 1005
alcoholism and, 1005
deafness in, 1005
diagnosis of, 1006
etiology of, 1005
hallucinations in, 1005
lead poisoning and, 1005
pathology of, 1005
prognosis of, 1006
symptoms of, 1005
syphilis and, 1005
tinnitus in, 1005
treatment of, 1006
bony tissue of, destruction of, in tuberculosis of middle ear, 316
concussion of, 1010
deafness in, 1011
detonations and, 1011
diplacusis acoustica in, 1011
etiology of, 1010
hyperesthesia acoustica in, 1011
nystagmus in, 1011
symptoms of, 1011
tinnitus in, 1011
treatment of, 1012
in deaf-mutism, 1028
destruction of, nystagmus in, 908
traumatic, 907
diseases of, surgical, 926
hemorrhage into, 1001
Labyrinth, hemorrhage in caisson workers, 1001
course of, 1001
in diabetes, 1001
in meningitis, 1001
in nephritis, 1001
termination of, 1001
hyperemia of, 1000
alcohol in, 1000
from amyl nitrite, 1000
diphtheria and, 1000
encephalitis and, 1000
etiology of, 1000
goitre and, 1000
meningitis and, 1000
mumps and, 1000
pneumonia and, 1000
puerperal fever and, 1000
from quinine, 1000
from salicylic acid, 1000
scarlet fever and, 1000
sinus thrombosis and, 1000
symptoms of, 1000
tinnitus in, 1000
tobacco in, 1000
treatment of, 1000
typhoid fever and, 1000
hyperostosis of bony capsule of, 720
hyperplasia of bony capsule of, 735
infection of, in acute suppurative otitis media, 748
inflammation of. *See* Labyrinthitis.
deafness in, 1006
injuries of, 1010
irritation of, nausea from, 670
nystagmus from, 670
mumps in, 1006
necrosis of, 1008
operations on, 953
Bourgnet's, 968
technique of, 968
Hinsberg's, 965
steps of, 965–968
indications for, 953
Jansen-Neumann, 957
closure of wound in, 964
preliminary measures, 957
steps of, 957–964
merits of, 955
Richards', 971
physiology of, 861
spongifying of bony capsule of, 735
static, 866
reaction movements of, 868
spontaneous nystagmus of, 867
suppuration of, 765
Labyrinthitis, 1006. *See* also Labyrinth, inflammation of.
acute diffuse serous, 934
caloric test in, 937
degrees of severity of, 938
destruction disharmony in, 938
etiology of, 934

Labyrinthitis, acute diffuse serous, nausea in, 936
operation in, indications for, 939
spontaneous nystagmus in, 936
symptoms of, 934
treatment of, 939
vertigo in, 936
vomiting in, 936
suppurative manifest, 939
course of, 945
deafness in, 942
destruction disharmony in, 941
etiology of, 939
fever in, 945, 947
headache in, 945
onset of, 946
pointing reaction in, 945
position of patient in, 945
prognosis of, 947
spontaneous nystagmus in, 946
stimulation disharmony in, 941
symptoms of, 940
tinnitus aurium in, 945
treatment of, 948
surgical, 948
vestibular reaction in, 942
circumscribed, 928
area of involvement in, 931
cristæ in, inhibited, 931
stimulated, 931
deductions from, 914
diagnosis of, 933
erosion with fistula in, 904
etiology of, 929
fistula in, absence of, 930
location of, 930
presence of, 930
giddiness in, 932
nausea in, 932
nystagmus in, 904
operation in, indications for, 933
spontaneous nystagmus in, 932
symptoms of, 932
rationale of, 929
traumatic, 906
vertigo in, 906
treatment of, 933
vertigo in, 904, 932
diagnosis of, 903
Barany's fixation apparatus in, 903
diffuse latent suppurative, 949
caloric reaction in, 950
deafness in, 949
diagnosis of, 951
Labyrinthitis, diffuse latent suppurative, diagnosis of, from cerebellar disease, 952
from disease of eighth nerve, 952
from hysteria, 951
from polyneuritis (syphilis) of eighth and facial nerves, 952
etiology of, 949
fistula in, 950
nystagmus in, 909
purulent discharge in, 950
symptoms of, 949
turning reaction in, 950
purulent, inflammatory pathological changes in, 902
mechanical pathological changes in, 902
general remarks on, 926
illustrative cases of, 917
infection in, 899
through oval window, 900
operations for, 953
oval window in, 900
pathology of, 899
round window and, 900
serous, deductions from, 914
site of, 900
static labyrinth and, 900
La Force adentome, 339
La petæ, 321. *See* Leprosy.
Lack's operation for collapse of alæ nasi, 303
Lacunar inflammation, acute, of pharyngeal tonsil, 331
lingual tonsillitis, acute, 349
pharyngitis, 353. *See* also Pharyngitis, chronic.
tonsillitis, acute, 396
Lamp, Kierstein's head, 39, 40
leukodescent, 46
Lancet, laryngeal, 455, 464
Laryngeal apoplexy, 511
carcinoma, 553
chorea, 510
crises, 32
diphtheria, 483
forceps applicator, Sajous', 464
Sajous', 469
hemorrhage, 472
lancet, 455, 458, 464
phthisis, 308
syncope, 511
vertigo, 511
Laryngectomy, complete, 568
after-treatment of, 573
indications for, 568
technique of, 568–574
partial, 567
indications for, 567

Laryngectomy, partial, technique of, 568
Laryngismus stridulus, 509
diagnosis of, from membranous laryngitis, 461
enlarged thymus gland and, 509
etiology of, 509
sexual excesses and, 489
treatment of, 509
uterine disease and, 509
Laryngitis, acute, in children, 555
calomel in, 457
cough in, 455, 456
codeine for, 458
diagnosis of, 456
from diphtheria, 456
from foreign bodies in larynx, 457
from perichondritis, 457
from pseudomembranous croup, 451
epipharyngitis in, 455
etiology of, 455
intubation in, 458
prognosis of, 457
symptoms of, 456
tracheotomy in, 458
treatment of, 457
atrophic, 471
cough in, 471
dyspnea in, 471
ethmoiditis and, 471
etiology of, 471
hoarseness in, 471
pathology of, 471
pilocarpine in, 472
prognosis of, 472
sphenoiditis and, 472
symptoms of, 471
treatment of, 472
catarrhal, acute, 449
age and, 452
aphonia in, 449, 453
arthritis and, 450
"catching cold" and, 449, 450
climate and, 452
cough in, 453
codeine for, 455
deflection of nasal septum and, 450
environment and, 451
etiology of, 449
hoarseness in, 449, 453
hygienic conditions and, 451
intubation in, 454, 455
lymphatic communication in, 451
nasal stenosis and, 450
pathology of, 452
pilocarpine in, 454
preëxisting diseases and, 450
prognosis of, 453
respiration in, 453
Laryngitis, catarrhal, acute, sinuitis and, 450
symptoms of, 452
syphilis and, 450, 453
systemic disturbances and, 449
tonsillitis and, 450
tracheotomy in, 454, 455
trauma and, 452
treatment of, 454
tuberculosis and, 450
voice in, 453
chronic, 464
diagnosis of, 472
from angina laryngis, 474
from carcinoma of subglottic region, 473
from enchondrosis of laryngeal cartilages, 473
from laryngeal carcinoma, 557
from lupus of larynx, 473
from pachydermia laryngis, 473
from papilloma of larynx, 474
from paralysis of posterior crico-arytenoid muscle, 473
from prolapse of ventricle of larynx, 473
from sarcoma of larynx, 473
from syphilis of larynx, 473
from tuberculosis of larynx, 473
mouth-breathing and, 465
tobacco and, 465
unstableness of vasomotor system and, 466
croupous, 459
diphtheritic, 459
hemorrhagic, 472
albuminuria and, 472
diabetes and, 472
etiology of, 472
hemophilia and, 472
leukemia and, 472
symptoms of, 472
syphilis and, 472
treatment of, 473
tuberculosis and, 472
typhoid fever and, 472
variola and, 472
voice in, excessive use of, 472
yellow fever and, 472
hypertrophic, diffused, adenoids and, 465, 467
alcohol and, 465, 467
deflections of nasal septum and, 465, 467
diagnosis of, 467
epipharyngitis and, 467
etiology of, 465
faucial tonsils and, 467
mouth-breathing and, 465

Laryngitis, hypertrophic, diffused, pathology of, 466
polypi and, 465, 467
prognosis of, 467
Sajous' laryngeal forceps in, 467
sinuitis and, 465, 467
smoking and, 465, 467
symptoms of, 466
trades and, 466
treatment of, 467
voice in, 466
discrete, 468
cough in, 468
diagnosis of, 469
from rhinoscleroma, 469
dyspnea in, 466, 469
etiology of, 468
pathology of, 468
symptoms of, 468
tracheotomy in, 469
treatment of, 469
localized, 468
hypoglottica, 468
membranous, 459
aphonia in, 460
Bacillus pyocyaneus in, 460
calomel fumigations in, 462
cardiac infection in, 461
cough in, 460
diagnosis of, 461
from diphtheria, 461
from laryngismus stridulus, 461
from retropharyngeal abscess, 461
from spasmodic laryngitis, 461
dyspnea in, 460
edema of bronchial mucous membrane in, 461
etiology of, 459
infectious diseases and, 459
intubation in, 460
laryngeal spasm in, 460
onset of, 460
pathology of, 460
pneumococcus in, 460
spirillum in, 460
staphylococcus in, 460
streptococcus in, 460
symptoms of, 460
tracheotomy in, 460
treatment of, 461
voice in, 460
phlegmonous, acute, 458
cough in, 458
dyspnea in, 458
erysipelas and, 458
etiology of, 458
pathology of, 459
prognosis of, 459
scarification in, 459
symptoms of, 458
Laryngitis, phlegmonous, acute, tracheotomy in, 459
treatment of, 459
sicca, 471
singing voice and, 534
spasmodic, diagnosis of, from membranous laryngitis, 461
stridulosa, 455. *See* Laryngitis, acute, in children.
subglottic, 456
subjective, chronic, 468
supraglottic, 456
syphilitic, diagnosis of, from laryngeal carcinoma, 557
tuberculous, 308
diagnosis of, from laryngeal carcinoma, 557
pregnancy and, 314
in pregnant women, 314
Laryngocele, 501
Laryngofissure, 546, 548, 558, 560, 561
hemorrhage in, 565
incisions in, 563, 564
indications for, 561, 562
for laryngeal paralysis, 524
rectal anesthesia for, 563
in stenosis of larynx, 505, 506, 565
syphilitic, 503
technique of, 562
Laryngoscopy, direct, 546, 549, 587
anesthesia for, 587
instruments for, 599
position of patient in, 588
slide speculum in, examination through, 589
indirect, 546, 549
technique of, 549
in laryngeal carcinoma, 556
Laryngotomy, intralaryngeal, 546
Larynx, abductor muscles of, paralysis of, 453
abscess of, 463
edema of larynx and, 462
etiology of, 463
perichondritis of larynx and, 462
tracheotomy in, 464
treatment of, 464
tuberculosis and, 463
carcinoma of, 553
diagnosis of, from tuberculosis of larynx, 312
catarrh of, acute, 449
deformities of, 501
diseases of, 445
edema of, 462
abscess of larynx and, 462
asphyxia in, 462
Bright's disease and, 462
diabetes and, 462
dyspnea in, 463
etiology of, 462
Ludwig's angina and, 462
pathology of, 462
perichondritis of larynx and, 462
peritonsillitis and, 462

Larynx, edema of, prognosis of, 463
sclerosis of liver and, 462
symptoms of, 463
syphilis and, 462
tracheotomy in, 463
treatment of, 463
tuberculosis and, 462
valvular lesions of heart and, 462
voice in, 463
erysipelas of, 463
foreign bodies in, 577
diagnosis of, from acute laryngitis in children, 457
glands of, 325
hyperplasia of, 501
hypertrophy of, 501, 502
papillomatous, 502
mouth-breathing and, 502
lupus of, 306
diagnosis of, from tuberculosis of larynx, 312
lymphatic drainage of, 551, 552
malformations of, 501
muscles of, abductor, paralysis of, 515
adductor, paralysis of, 514, 515
aryteno-epiglottic, paralysis of, 516
cricothyroid, paralysis of, 514, 515, 543
intrinsic, paralysis of, 453, 512
tensor, external, of vocal cords, paralysis of, 515
thyro-epiglottic, paralysis of, 516
neoplasms of, 544
benign, diagnosis of, from laryngeal carcinoma, 557
etiology of, 544, 545
irritation and, 545
location of, 544
malignancy of, tendency toward, 546
malignant, 551. *See* also Carcinoma, laryngeal.
mouth-breathing and, 545
syphilis and, 545
tuberculosis and, 545
varieties of, 544
neuralgia of, 510
neuroses of, motor, 508
papilloma of, 546
perichondritis of, 457
edema and, 462, 463
pouches of, dilatation of, 501
singing voice and, 527
spasm of, 460, 508, 509
stenosis of, 501, 502
laryngeal diphtheria and, 483
syphilis and, 501
syphilis of, 319
diagnosis of, from tuberculosis of larynx, 312
tuberculosis of, 309
Lateral sinus, thrombosis of, 810
Lavage, 124
Lavage of ethmoidal sinus, 194
of frontal sinus, 191
of maxillary sinus, 192
of sphenoidal sinus, 194
"Le con proconsulair" symptom in diphtheria, 485
Lead poisoning, arteriosclerosis of labyrinth and, 1005
Leeching, 123
for acute catarrhal otitis media, 715, 716
for simple acute mastoiditis, 768
for suppurative otitis media, 775, 776
Leiter coil, 406, 650, 654, 768, 769
Leontiasis, 321
Lepra anesthetica seu nervosa, 323
Leprosy, 321
anesthetic, 323
etiology of, 322
infection in, modes of, 322
nasal secretion in, contagiousness of, 322
pathology of, 322
prognosis of, 323
Leprous stenosis of larynx, 504
Leptomeningitis, 794
Cheyne-Stokes respiration in, 795
ethmoiditis and, 794
lumbar puncture in, 795
otitic origin of, 794
prognosis of, 795
sphenoiditis and, 794
symptoms of, 795
Westphal's symptoms in, 795
Leptothrix in tonsils, 408
Leukemic deafness, 1006
Leukocytosis in reaction of inflammation, 110, 122
Leukodescent lamp, 46, 125, 136, 190, 306, 313, 356, 406
light, 125
Lingual tonsil, 348, 384
varix, 350
Lip reading, 1032
Lipoma of nose, 280
of pharynx, 373
of tonsils, 442
Lips, paralysis of, in chronic bulbar paralysis, 366
Liver in diphtheria, 480
sclerosis of, edema of larynx and, 462
Localized hypertrophic laryngitis, 468
Localizer, Ostrum's, 223
Locomotor ataxia, deafness from, 1022
laryngeal paralysis and, 521
Loeb's projections of accessory sinuses of nose, 162
"Lop ear," 645
Low tracheotomy, 490, 594
Lowenberg method of Eustachian catheterization, 696
Lower tracheobronchoscopy, 594
Ludwig's angina, edema of larynx and, 462
Lumbar puncture, 790

Lumbar puncture in leptomeningitis, 795
Lumpy jaw, 325
Lungs in deaf-mutism, 1030
in diphtheria, 479
Lupous stenosis of larynx, 504
Lupus, arsenic paste for, 305, 306
of auricle, 306
treatment of, 306
of larynx, 306
diagnosis of, from chronic laryngitis, 473
from tuberculosis of larynx, 312
of nose, 304
electrocautery in, 305
etiology of, 304
radiotherapy for, 305
radium in, 305
Röntgen rays in, 306
symptoms of, 304
treatment of, 305
ultraviolet rays in, 306
pachydermia laryngis and, 500
of pharynx, 306
Lymphadenoma of pharynx, 369
of tonsils in Hodgkin's disease, 444
Röntgen rays in, 444
Lymphatic drainage of larynx, 551, 552
glands in diphtheria, 480
of neck, 388
hypertrophy of, 333
involvement of, in laryngeal carcinoma, 555
suppurative diseases of ear and, 28
infection through tonsils, 379, 381, 382, 387, 388, 390
system, infection through, 32
vessels of neck, 388
tonsillar, 387, 388
Lymphoid tissue in adenoids, 335
of pharynx, 354
of tonsil, faucial, 383, 384
lingual, 348, 351
Lymphoma of nose, 278
of pharynx, 369
Lymphosarcoma of pharynx, 375

M

McKernon's rongeur forceps, 858
Mackenzie's reflex area, 34
Macrotia, 646
treatment of, 647
Malaria, nose and, 29
Mallein in diagnosis of glanders, 324
Malleus, 602
attachment of, 606
fracture of, 670
removal of, 609
in ossiculectomy, 821, 822
in radical mastoid operation, 836, 837
Manometer in inflation of tympanic cavity, 697
Margo supratonsillaris, 408, 419, 424, 425, 430
Massage, 124
in adhesive processes of middle ear, 730
apparatus, 40
Delstanche's, 40, 42
Pynchon's, 41
for chorditis nodosa, 471
of eardrum, 40, 42
of Eustachian tube, 691
external mechanical, inflation of tympanic cavity by, 701
for suppurative otitis media, 776
Masseur, Weaver's intratympanic, 690
Mastoid affections, defective hearing from, 634
antrum, location of, 827
necrosis of, 603, 609, 759
opening of, 828
otitis media and, 770
bone, sclerosis of, in chronic mastoiditis, 771
cells, distribution of, 610
exenteration of, 831, 833, 834
in radical mastoid operation, 833
cortex, removal of, 830, 833
in radical mastoid operation, 833
emissary vein, tenderness over, in thrombosis of lateral sinus, 812
operation, facial paralysis and, 994
perichondritis after, 601, 654
radical, 793, 833
after-treatment of, 846, 847
Ballance flap method in, 840, 854
Ballenger's modification of, 848
incision in, 849, 850
curettage of Eustachian tube in, 838
danger to oval window in, 836
disfigurement after, 848
exenteration of mastoid cells in, 833
facial paralysis and, 834
incision in, Ballance, 841, 843
in infants, 857
Jansen-Stacke, 846, 847
Panse, 846
postauricular, 859
"shepherd's crook," 841, 843
Siebermann's Y, 845
indications for, 760, 764, 773
in infants and children, 859

Mastoid operation, radical, plastic surgery of cutaneous meatus in, 840
removal of incus in, 836, 837
of malleus in, 836, 837
of mastoid cortex in, 833
of necrosed bone from tympanic cavity in, 838
of outer wall of atrium in, 838
of outer wall of attic in, 838
of posterior wall of meatus in, 834
Thiersch grafts after, 844, 853
Trautmann tongue flap method in, 846
simple, 824
after-treatment in, 833
Allport's mallet for, 828
anatomical landmarks for, 826, 827
antrum in, location of, 827
opening of, 828
Ballenger's mastoid periosteal elevator in, 826
cells in, exenteration of, 831
closure of cutaneous wound in, 832
cortex in, removal of, 830
dressing in, 832
elevation of cutaneous periosteal flaps in, 823
incisions in, 824, 825
Whiting's, 825
irrigation in, 832
Russian perforator for, 827, 828
technique of, 824
process, caries of, 772
destruction of, in tuberculosis of middle ear, 316
diseases of, 765
exenteration of, landmarks after, 835
necrosis of, 772
osteosclerosis of, 771
periostitis of, acute, 770
etiology of, 770
treatment of, 770
Wilde's incision for, 770
tuberculosis of, 314
Mastoiditis. *See* also Otitis media, suppurative.
acute, Bier's hyperemic treatment in, 769
Bezold's, 857
treatment of, surgical, 859, 860
chronic, 770, 833
caries of mastoid process in, 772
diagnosis of, 770
Mastoiditis, chronic, incus in, destruction of, 771
intracranial complications in, 771
membrana tympani in, 771
necrosis of mastoid process in, 772
neuralgia in, 771
operation for, radical mastoid, 773
prognosis of, 772
sclerosis of mastoid bone in, 771
Siegle's otoscope in, 771
symptoms of, 770
treatment of, 773
Eustachian tube and, 687
extradural abscess and, 792
incision for, Whiting's, 825
Wilde's, 824
otitis media and, suppurative acute, 747
pathology of, 784
primary, acute, 823
meningitis and, 824
thrombosis of lateral sinus and, 824
sequelæ of, 765
simple, acute, 765
Bier's treatment for, 769
cold applications for, 768
"dip" of postsuperior wall in, 766
without intracranial lesions, 765
leeching for, 768
pain in, 766
perforation in, 767
Schwartze's point of tenderness in, 766
spontaneous cures of, 767
symptoms of, 765
torticollis in, 766
treatment of, 767
subacute, 769
surgical intervention in, indications for, 823, 824
Mathieu's tonsillotome, 417
Maxilla, superior, resection of, for nasal polypus, 277
Maxillary sinuitis, hyperesthetic rhinitis and, 255
sinus, 166
Mayer's nasal tube splints, 79
Meatomastoid operation, 848
indications for, 756, 759, 760, 764, 768
Meatus, auditory, external, 601
in acute suppurative otitis media, 744
affections of, defective hearing from, 630
epithelial plugs in, 644
exostosis of, 661
follicular inflammation of, 657

Meatus, auditory, external, furunculosis of, 657
hyperostosis of, 661
inflammation of, croupous, 661
prognosis of, 661
treatment of, 661
diffused, 659
duration of, 660
etiology of, 659
hyperostosis in, 660
periostitis and, 660
prognosis of, 660
symptoms of, 659
treatment of, 660
hemorrhagic, 660
treatment of, 660
mycosis of, 663
stricture of, 663
diphtheria and, 663
cartilaginous, of ear, collapse of, defective hearing from, 630
cutaneous, plastic surgery of, in radical mastoid operation, 840
Mediastinum, tumors of, laryngeal paralysis and, 515, 518, 520, 521, 522
Medulla oblongata, lesions of, laryngeal paralysis from, 521
Membrana flaccida, perforation of, significance of, 783
tectoria, 614, 615
tympani, abscess of, 671
adenoids and, 346
adhesions of, operations for, 734
adhesive processes of middle ear and, 726
affections of, defective hearing from, 630
atrophy of, 679
calcareous deposits in, 679, 721, 788
in chronic mastoiditis, 771
moist catarrhal otitis media, 720, 721
dermic layer of, ulceration of, 673
diseases of, 669
function of, 611
herpes zoster of, 656
incision of, 674, 675, 676, 749, 767, 768, 775
in adhesive processes of middle ear, 678
curved bistoury for, 675
with electrocautery, 680
indications for, 677
knife for, 675
with lancet, 681
location for, 675
methods of, 680
in myringitis with abscess formation, 677
in ossiculectomy, 819, 820
Membrana tympani, incision of, in otitis media, 677
acute catarrhal, 678
suppurative, 678
chronic catarrhal, 677
postoperative considerations, 682
in suppuration of middle ear, 678, 679
in tenotomy of tensor tympani muscle, 677
inflammation of, 671, 673. *See also* Myringitis.
injuries of, 669
etiology of, 669
nausea in, 670
nystagmus in, 670
prognosis of, 670
symptoms of, 670
treatment of, 671
malformations of, 669
massage of, 40, 42
Beck's mercury, 43
operations for, 733
perforation of, 603, 673, 754, 755
in acute suppurative otitis media, 744, 745
central, 755, 757
deafness from, 673
marginal, 759
significance of, 669
spontaneous, 676
retraction of, 683
rupture of, 670
tuberculous, 315
Membranous croup, 483
idiopathic, 459
laryngitis, 459
Ménière's disease, 634, 1002
deafness in, 1002
diagnosis of, 1002
from Ménière's symptom complex, 1002
dizziness in, 1002
nausea in, 1002
nystagmus in, spontaneous, 1002
prognosis of, 1003
tinnitus in, 1002
treatment of, 1003
vertigo in, 1002
vomiting in, 1002
symptom complex, 1002
diagnosis of, from Ménière's disease, 1002
pneumomassage in, 1004
Rinné test in, 1004
tinnitus in, 1003
treatment of, 1004
Meningitis, 789
primary mastoiditis and, 824
suppurative otitis media and, 746, 748, 752

Meningitis, ethmoid operation and, 247
hemorrhage into labyrinth in, 1001
hyperemia of labyrinth and, 1000
infection of tympanic cavity and, 608
metastatic, in thrombosis of lateral sinus, 812
after middle turbinotomy, 153
nystagmus in, 910
serosa, 790
dizziness in, 790
etiology of, 790
nystagmus in, 790
symptoms of, 790
torticollis in, 790
treatment of, 791, 983
sinuitis and, 164, 180
following turbinotomy, 242
Mentality in adenoids, 337
Menthol in tuberculosis of larynx, 313
Mesopharynx, inflammatory diseases of, 352
Meyer's ring curette, 339, 344
Michel's metal clamp suture, 857, 859
suture clip forceps, 859
Microtia, 646, 647
Middle ear, inflation of, in acute suppurative otitis media, 749
suppuration, bacteriology of, 786, 787
tuberculosis of, 314
Mikulicz's cells, 287
Miller's asthma, 455, 509. *See* also Laryngismus stridulus.
Mixed diphtheria, 481
infection in diphtheria, 479
laryngeal diphtheria, 484
Mogiphonia, 511
Monophasia, 1020
Morve, 323
Mosetig-Moorhof plastic operation, 990, 992
Mosher's frontal-ethmoid operation, 247
safety-pin holder, 592
Moure's operation for deviations of nasal septum, 81
upon ethmoidal sinuses, 250
Mouth-breathing in adenoids, 337
chronic laryngitis and, 465
diffused hypertrophic laryngitis and, 465
neoplasms of larynx and, 545
papillomatous hypertrophy of larynx and, 502
Mucin in nasal hydrorrhea, 265
Mucopurulent rhinitis, 154
Mucosa knife, Ballenger's, 96
Mucous membrane, sloughing of, in diphtheria, 479
rhinitis, simple, 154
Multiple sclerosis, laryngeal paralysis and, 521
throat in, 32
Mumps, hyperemia of labyrinth and, 1000
labyrinth in, 1000
Muscle, constrictor, superior, of pharynx, 385, 391, 420, 425
digastric, in excision of external carotid artery, 378
palatoglossus, 384, 391
palatopharyngeus, 384, 391
stapedius, 607, 612
sternocleidomastoid, lymphatic relations of, 387
stylohyoid, in excision of external carotid artery, 378
stylopharyngeus, 995
tensor tympani, 607, 612
tenotomy of, 677
Muscles in diphtheria, 480
of larynx, 512, 513, 515
Mutism, 1029
Mycosis of external auditory meatus, 664, 665
leptothricia of tonsils, 408
Myles' operation upon maxillary sinus, 225
Myringitis, 671
chronic, 673
defective hearing from, 631
diagnosis of, 672
eruptions in, 671
etiology of, 671
hearing in, 672
prognosis of, 672
symptoms of, 671
treatment of, 672
Myxoma of nose, 269
classification of, surgical, 272
etiology of, 269
pathology of, 270
prognosis of, 272
removal of, with snare, 274
symptoms of, 270
treatment of, 272, 273
of pharynx, 370
Myxosarcoma of pharynx, 375

N

Nasal chambers, 17
singing voice and, 532
deformity in cretinism, 298
diphtheria, 481, 482, 485
disease, goitre and, 268
Graves' disease and, 268
singing voice and, 526, 534
hemorrhage, 284
hydrorrhea, 265
lesions, diseases of eye and, 34, 35
neuroses, 253
obstruction, 115
acute catarrhal otitis media and, 707
respiration, 23
secretion in empyema of accessory sinuses, 31
in leprosy, contagiousness of, 322

Nasal secretion in obstructive deviations of nasal septum, 65
septum, 17
cartilage of, reformation of, 101, 102
deflection of acute catarrhal laryngitis and, 450
deformities of, 57
deviations of, 57
cartilaginous, 60
operations for, 67, 68
classification of, 59
correction of, 62, 67
diffused hypertrophic laryngitis and, 465, 467
epistaxis and, 66
etiology of, 57, 58
hemorrhage and, 66
hyperesthetic rhinitis and, 255
obstructive, asthma in, 65
dizziness in, 63
headache in, 63
nasal secretions in, 65
stenosis in, 65
turbinated bones in, 63
vertigo in, 63
operations for, Asch-Mayer's, 77, 78, 79
Bosworth's, 70, 71, 72
Freer's, 100, 101
Gleason's, 75, 76, 77
Kyle's, 80
Moure's, 81
open method of, 100, 101
Price-Brown's, 81
Roe's, 77
Sluder's, 74, 75
Watson's, 73, 74
osseous, 60
operations for, choice of, 68, 69
symptoms of, objective, 66
subjective, 63
hypertrophies of, soft, 70
cautery for, 70
lesions of, obstructive, 62
operations for, 67, 68, 69
perforation of, 103
etiology of, 103
operations for, 104
Ballenger's mucosa swivel knife for, 105
Goldsmith's, 109
Goldstein's plastic flap, 104
Hazletine's plastic, 105, 106
Yankauer's intranasal suture, 107, 108
in scarlet fever, 103
symptoms of, 103
treatment of, 104
tuberculosis of nose and, 307
Nasal septum, spurs on, removal of, 69, 72
submucous resection of, 82–100
accidents in, 99, 100
after-treatment in, 98
anesthesia in, 82, 84
dressing for, 98
elevation of mucoperichondrium in, 90
of periosteum in, 85, 90
elevators for, 85, 86, 87
Foster-Ballenger forceps in, 93, 94, 96
Freer's method, 100, 101
gouge for, 94, 96
incision in, through cartilage, 91
Hajek's, 84, 90
Killian's, 84, 85, 90
inspection of field of operation in, 97, 98
open method of, 100, 101
position of patient in, 82
splints for, 98
spokeshave in, use of, 93
swivel knife in, use of, 91, 92, 96
sinuses, accessory, cerebral hemorrhages and, 187
optic neuritis and, 187
splint, Carter's, 292
stenosis, respiration and, 17
suppuration, 160
tachycardia, 268
tube splints, Mayer's, 79
Nausea in acute diffuse serous labyrinthitis, 936
in circumscribed labyrinthitis, 932
in injuries of membrana tympani, 670
from irritation of labyrinth, 670
in Ménière's disease, 1002
Neck, tumors of, laryngeal paralysis and, 521
Negative air pressure, treatment of sinuitis by, 196
pressure apparatus, 45
Neisser-Ernst bodies in diphtheria, 477
Neisser's stain for Klebs-Loeffler bacillus, 477
Neoplasms of larynx, 544
of nose, 269
of pharynx, 368, 374
of subglottic space, 546
of tonsils, 442, 444
Nephritis, hemorrhage into labyrinth in, 1001
Nerve, acusticus, paralysis of, 515

Nerve, auditory, 613
distribution of, 613
paralysis of, 1015, 1016
auricularis posterioris profunda, 995
chorda tympani, relations of, 607, 608
facial, anastomosis of, with hypoglossal nerve, 995–999
anatomy of, 994, 995
paralysis of, 515, 993, 994
relation of, to aditus ad antrum, 834
to annulus tympanicus, 835
to horizontal semicircular canals, 835
to tympanic cavity, 836
surgery of, plastic, 993–995
glossopharyngeal, paralysis of, 515
hypoglossal, anastomosis of, with facial nerve, 995–999
paralysis of, 993, 994
surgery of, plastic, 993, 994
infra-orbital, relation of, to maxillary sinus, 167
laryngeal, inferior, paralysis of, 516, 517
recurrent, paralysis of, 516, 517, 520
superior, paralysis of, 515
optic, relation of, to sphenoidal sinus, 169
pneumogastric, 513, 515, 516, 518
irritation of, shock from, 575
relations of, 518
spinal accessory, 515, 518, 521
Nervous cough, 511
system in diphtheria, 479, 486
Neumann's law, 875
noise apparatus, 627
Neuralgia in acute suppurative otitis media, 744
in chronic mastoiditis, 771
of larynx, 510
of pharynx, 365
Neurasthenia, hyperacuteness of hearing and, 1016
laryngeal paralysis and, 522, 523
nystagmus in, 907
vertigo in, 907
Neuritis, facial paralysis and, 993
optic, accessory nasal sinuses and, 187
sinuitis and, 187
Neuroses of larynx, 508
Nitrate of silver, solutions of, 48
Nitrous oxide anesthesia in tonsillotomy, 436
Nodules, singer's, 469
Noises, subjective, 1018
Nose, accessory sinuses of, 19, 161
actinomycosis of, 325
adenoma of, 278
alternating stenosis of, 137
anatomy of, clinical, 17
anemia and, 28
Nose, angioma of, 278
aquiline or hump, operation for, Ballenger's intranasal, 296
external, 295
attic of, 254
blood supply of, 21
carcinoma of, 281
chambers of, 17
colloid degeneration of, 287
deformities of, correction of, 291
paraffin injections for, 297
Thiersch graft for, 293
digestive tract and, 29
diphtheria of, 482, 483
dislocated, 294, 295
edema of, acute circumscribed, 264
erectile tissue of, 18
excretory organs and, 30
fibroma of, 276
as a filter, 24, 25
foreign bodies in, 289
functions of, 23, 25
furunculosis of, 288
general medicine and, 26
glanders of, 323
hemorrhage from, 284
hypersensitive area in, nervous cough from, 511
inflammatory diseases of, 110
lipoma of, 280
long or drooping, operation for, Ballenger's, 297
lupus of, 304
lymphoma of, 278
malaria and, 29
meatuses of, 18
middle, 18
myxoma of, 269
superior, 19
neoplasms of, 269
nerve supply of, 19
nerves of, sensory, 19
neuroses of, 253
obstruction of, 115, 117, 119, 120
osteoma of, 279
papilloma of, 275
physiology of, 22
polypi of, 269
rheumatic fever and, 29
saddle, paraffin injections for, 298
sarcoma of, 281
screw-worms in, 289
septum of, 17
stenosis of, 31
streptodiphtheria of, 483
submucous resection of. *See* Nasal septum, submucous resection of.
"swell bodies" of, 18, 24
syphilis of, 33, 317
turbinated bones of, 17, 18
twisted or crooked, Ballenger's operation for, 291, 292, 293
"vicious circle" of, 199
"key" to, 199

Nose, "vicious circle" of, polypi in, 269
Nystagmus in acute destruction of one labyrinth, 908
cerebellar, 872
erysipelas of face and, 873
of scalp and, 873
in circumscribed labyrinthitis, 904
in concussion of labyrinth, 1011
in diffuse latent suppurative labyrinthitis, 909
induced, after-, 873
after-after-, 873
caloric, 874
characteristic of, 873
fistula, 874
galvanic, 874
primary, 873
in injuries of membrana tympani, 670
of intracranial origin, 910
from irritation of labyrinth, 670
laws of, 874
Ewald's, 875
Fleurens', 874
Hoegye's, 874
Neumann's, 875
in meningitis, 910
serosa, 790
in neurasthenics, 907
spontaneous, in acute diffuse serous labyrinthitis, 936
suppurative manifest labyrinthitis, 946
in circumscribed labyrinthitis, 932
in Ménière's disease, 1002
in suppurative otitis media, 752
toxemia and, 907
vestibular, deductions from, 913
spontaneous, 870
characteristics of, 871

O

Obstructive lesions of nasal septum, 62
rhinitis, 142
Occupation deafness, 1012, 1013
Ocular symptoms in sinuitis, 164, 169, 181, 186, 187
O'Dwyer's intubation instruments, 493
Odor, subjective, significance of, 173
Odynophagia, 449
Œdema. *See* Edema.
Office equipment, 36–47
Ointments, 52
Olfaction, neuroses of, 253
Olfactory fissure, obstruction of, 21, 119, 168
anosmia and, 253
pus in, 19, 160
lobe, irritation of, hyperosmia from, 253
nerve, 21
Ollier's operation for sarcoma of nose, 282
Open method of operation for deviations of nasal septum, 100, 101
Operating chair, 36, 37
Ophthalmic veins, infection through, 32
Opsonic index, 127
Optic neuritis, accessory nasal sinuses and, 187
sinuitis and, 187
Orbito-ethmoid operation upon ethmoidal sinuses, 251
Orthoform in tuberculosis of larynx, 313
Osseous deviations of nasal septum, 60
Ossicles of ear, 607
ankylosis of, in adhesive processes of middle ear, 728
in deaf-mutism, 1028
defective hearing from, 631
caries of, defective hearing from, 631
fracture of, 670
loss of, in acute suppurative otitis media, 748
Ossiculectomy, 609, 759, 819
after-treatment of, 823
anesthetic for, 819
Delstanche's ring knife for, 821
dressing in, 823
hemorrhage in, 823
incision of membrana tympani in, 819, 820
preparation of ear for, 819
removal of incus in, 821, 822
of malleus in, 821, 822
Sexton's knives for, 821
Osteoma of epipharynx, 280
of nose, 279
diagnosis of, 279
pathology of, 279
symptoms of, 279
treatment of, 279
Osteoplastic operation, Beck's double, upon frontal sinus, 219
Küster's, upon frontal sinus, 218
Osteosclerosis of mastoid process, 771
Ostrum's forward cutting forceps, 228
localizer, 223
Othematoma, 648
dementia and, 648
diagnosis of, 650
from angioma of auricle, 651
etiology of, 647
Leiter coil for, 650
pain in, 649
prognosis of, 650
resemblance of, to perichondritis of auricle, 654
symptoms of, 649
treatment of, 650
Otitis externa, 657
crouposa, 661
diffusa, 659
hæmorrhagica, 660
parasitica, 654
interna, parotitica, 1006
media, acute lacunar tonsillitis and, 398

Otitis media, adenoids and, 338
bacteriology of, 785, 786
catarrhal, acute, 703
auscultation in, 712
bacteriology of, 703, 704
bone conduction in, 711, 713
climate and, 706
diabetes and, 710
diagnosis of, 709
duration of, 713
epipharyngeal diseases and, 708
ethmoiditis and, 708
etiology of, 703
faucial tonsils and, 708
incision of membrana tympani in, 678
inflation of tympanic cavity for, 714, 715
intracranial complications in, 710
leeching for, 715, 716
membrana tympani in, 711
nasal diseases and, 707
obstruction and, 707
onset of, 710
pathology of, 709
pneumomassage for, 715
prognosis of, 713
Rinné test in, 711
sphenoiditis and, 708
symptoms of, 709, 710
syphilis and, 708
treatment of, 713, 714
tubal disease and, 526
tuberculosis and, 708
weather and, 704, 705, 706
Weber test in, 711
chronic moist, 718
adenoids and, 723
autophony in, 720
deafness from, 718
deafness in, 719
ethmoiditis and, 722
etiology of, 718
membrana tympani in, 720, 721
paracusis Willisii in, 720
passive hyperemia in, 724
prognosis of, 722
sphenoiditis and, 722
symptoms of, 719
tinnitus aurium in, 719
Otitis media, catarrhal, chronic moist, treatment of, 722, 723
catarrhalis chronica, 724
sicca, 724
incision of membrana tympani in, 677
insidiosa, 735
mastoid antrum and, 770
pathology of, 784
sclerotica, 724
sinus thrombosis and, 748, 752
suppurative, acute, 742
abscess of brain and, 748
adhesions in, 747
compression of air in meatus in, 749
course of, 745
deafness from, 745, 747
death from, 748
diabetes and, 744
diagnosis of, 748
drainage in, 749
etiology of, 742
external auditory meatus in, 744
hearing in, 745
incision of membrana tympani in, 678
infectious fevers and, 742, 746, 750
inflation of middle ear in, 749
labyrinth in, infection of, 748
mastoiditis and, 747
membrana tympani in, 744, 748
meningitis and, 746, 748, 752
mucous membrane in, loss of, 748
neuralgia in, 744
ossicles in, loss of, 748
pain in, 743, 748
perforation in, 744, 746
predisposing causes of, 743
probe in, use of, 748
prognosis of, 749
as to hearing, 756
Rinné test in, 747
Rivinian segment and, 743
secretions in, 745
sequelæ of, 746
subjective noises in, 745
symptoms of, 743
temperature in, 744, 748
terminations of, 746
treatment of, 749, 756, 757, 759

Otitis media, suppurative, acute, tympanic murmur in, 749
Weber test in, 745
in children, 750
convulsions in, 751
symptoms of, 751
treatment of, 752
adenoids and, 346
chronic, 752
adenoids and, 757
dry gauze dressings for, 758
epipharyngitis and, 758
forms of, 753
otorrhea in, 753
pain in, 753
perforations in, 754
sinuitis and, 758
symptoms of, 752
treatment of, 781
alcohol, 760, 781
with boric acid powder, 782
with camphoroxol, 782
with compound tincture of benzoin, 782
by curettage, 760
dry gauze, 781
by irrigation, 782
via Weber-Liel catheter, 758
free drainage for, 779
incision of membrana tympani in, 775
leeching in, 775, 776
massage for, 776
nystagmus in, 790
radiant light for, 775
removal of granulations in, 779
morbid material in, 779
sequelæ of, 765
treatment of, 774
vaccine therapy for, 776
syphilitic, 1007
tuberculous, 316
Otomycosis, 664
Otopiesis, 1030
Otorrhea, chronic, in abscess of brain, 752
dangers of, 787
extradural abscess and, 792
life insurance and, 752, 836
treatment of, 757, 759
in chronic suppurative otitis media, 753
in deaf-mutism, 1030
Otosclerosis, 735
diagnosis of, 738
etiology of, 735
pathology of, 737
prognosis of, 740
symptoms of, 738
Otosclerosis, synonyms of, 735
treatment of, 740
mechanical, 741
medical, 740
prophylactic, 741
surgical, 741
Otoscope, Siegle's, 745, 750
Oval window, 608
hyperostosis of, 739
combined with otitis media, 740
membrane of, 607
Oxyecoia, 1016
Ozena, 154
in atrophic rhinitis, 156
hypertrophic, 142
in syphilis of internal ear, 1007

P

Pachydermia laryngis, 500
accessory, 500
diagnosis of, from chronic laryngitis, 473
electrolysis for, 500, 501
lupus and, 500
papilloma of larynx and, 546
perichondritis and, 500
potassium iodide in, 500
syphilis and, 500
tuberculosis and, 500
Pachymeningitis circumscripta, treatment of, surgical, 983
externa circumscripta, 792. *See* also Extradural abscess.
interior circumscripta, 794
Pancreas in diphtheria, 480
Panotitis, 747
Panse incision in radical mastoid operation, 846
Papilloma of larynx, 546
aphonia in, 547
diagnosis of, from chronic laryngitis, 474
dyspnea in, 547, 548
etiology of, 546
hoarseness in, 547
pachydermia laryngis and, 546
pathology of, 548
prognosis of, 548
reflex cough in, 368
symptoms of, 547
syphilis and, 546
treatment of, 548, 549
tuberculosis and, 546
of nose, 275
treatment of, 275
of pharynx, 368
electrocautery in, 368
treatment of, 368
of tonsils, 442
cough in, 442
Papillomatous hypertrophy of larynx, 502

Paquelin cautery for angioma of auricle, 651
Paracentesis, 677
Paracusis, 1016
duplex, 1017
Willisii, 739, 1017
in adhesive processes of middle ear, 727
in chronic moist catarrhal otitis media, 720
Paraffin injections in atrophic rhinitis, 158
for deformities of nose, 297
hematoma and, 300
in rhinolalia pata, 298
Paralysis of abductor muscles of larynx, 453
angioneurotic, of auditory nerve, 1015
chronic bulbar, galvanism in, 366
paralysis of lips in, 366
pneumonia and, 366
from sunstroke, 366
diphtheria antitoxin and, 489
diphtheritic, 486
galvanism in, 367
of esophagus, 367
facial, 993
etiology of, 993
mastoid operation and, 994
neuritis and, 993
of otitic origin, 993
paralysis of pharynx and, 367
perineuritis and, 993
radical mastoid operation and, 834
surgery of, plastic, 993, 994, 995
treatment of, 994
tumors and, 994
of hypoglossal nerve, 993
hysterical, of auditory nerve, 1016
galvanism for, 1016
treatment of, 1016
of intrinsic muscles of larynx, 453
laryngeal, 512, 524
anesthesia of larynx in, 516
aneurysm and, 522
of aorta and, 517, 520
of subclavian and, 518, 521
aphonia in, 516, 519, 521
bilateral, 519
abductor, 522
"cadaveric" position of vocal cords in, 519, 521
cough in, 519
diphtheria and, 515, 520
from disease of superior laryngeal nerve, 515
dyspnea in, 519, 521, 522
enlarged thyroid gland and, 521
etiology of, 515
faradism for, 520
galvanism for, 516, 520
goitre and, 515, 518
hoarseness in, 516, 521
Paralysis, laryngeal, indication of aneurysm of arch of aorta, 515
intubation for, 524
laryngofissure for, 524
from lesions of medulla oblongata, 521
of nuclei of spinal accessory nerve, 521
lobar pneumonia and, 516
locomotor ataxia and, 521
multiple sclerosis and, 521
neurasthenia and, 522, 523
pericarditis and, 515, 518
phonation in, 519
pleurisy and, 515, 518
pneumonia and, 516
progressive bulbar paralysis and, 521
scoliosis and, 518
syphilis and, 519, 521, 522
thyroidectomy in, 521
tracheotomy in, 520, 522, 523, 524
traumatisms and, 515, 518
tuberculosis and, 515
tumors and, 515, 518, 520
of brain and, 521
of mediastinum and, 515, 518, 520, 521, 522
of neck and, 521
typhoid fever and, 515
unilateral, 517, 520
voice in, 516, 522
of pharynx, 365, 366, 367
of posterior crico-arytenoid muscle, diagnosis of, from chronic laryngitis, 473
progressive bulbar, laryngeal paralysis and, 521
rheumatic, of auditory nerve, 1015
from tumor of brain, 1021
Parosmia, 253
in sinuitis, 186
Paresthesia of pharynx, 364
Passow-Trautmann plastic operation, 989, 990
Patency of Eustachian tube, 688
Perception of tone, 618
Perforation of membrana tympani, 673
of nasal septum, 103
Pericarditis, laryngeal paralysis and, 515, 518
Perichondritis of auricle, 601, 654
mastoid operation and, 599, 654
resemblance of, to othematoma, 654
symptoms of, 654
treatment of, 654
of cricoid cartilage, 457
of larynx, 457
diagnosis of, from acute laryngitis in children, 457
from laryngeal carcinoma, 557
edema of larynx and, 462, 463

Perichondritis, pachydermia laryngis and, 500
Perilymph, 613
Perineuritis, facial paralysis and, 993
Periosteum, elevation of, in submucous resection of nasal septum, 85, 90
Periostitis acute, of mastoid process, 770
diffused inflammation of external auditory meatus and, 659
Peritonsillar abscess, 404. *See* also Peritonsillitis.
Peritonsillitis, 404
Ballenger's operation for, 407
complications of, 405
edema of larynx and, 462
etiology of, 404
sequelæ of, 405
symptoms of, 404
thrombophlebitis and, 405
treatment of, 405, 406
Perpendicular plate of ethmoid, removal of, in submucous resection of nasal septum, 94
Pes anserinus, 378, 995
Pharyngeal adenoids, 333
scissors, 347
tonsils, development of, 384
Pharyngitis, catarrhal, simplea cute, 352
chronic, 353
cough in, 354
electrocautery in, 355
etiology of, 353
hoarseness in, 354
pathology of, 354
prognosis of, 355
rheumatic diathesis and, 354, 355
symptoms of, 354
tobacco and, 353, 354
treatment of, 355
granular, 353. *See* also Pharyngitis, chronic.
lacunar, 353. *See* also Pharyngitis, chronic.
singing voice and, 531
Pharyngotomy, subhyoid, 565
indications for, 565
technique of, 565, 566, 567
Pharynx, akinesis of, 365
anesthesia of, 364
angioma of, 373
cystoma of, 369
erectile tumors of, 373
fibroma of, 370
glanders of, 325
hyperesthesia of, 364
globus hystericus and, 364
hyperkinesis of, 365, 367
hypersensitiveness of, 364
lipoma of, 373
lupus of, 306
lymphadenomata of, 369
lymphoid tissue of, 354
lymphoma of, 369
malformations of, 362
Pharynx, muscles of, paralysis of, 366
myxomata of, 370
neoplasms of, 368, 374
neuralgia of, 365
neuroses of, 364, 565
rheumatism and, 365
tobacco and, 364
papillomata of, 368
paralysis of, bulbar, acute, 365, 366
chronic, 366
central, 365, 366
diphtheritic, 366
symptoms of, 366
treatment of, 367
facial paralysis and, 367
peripheral, 366
paresthesia of, 364
during menopause, 365
sarcoma of, 374
diagnosis of, from fibroma of pharynx, 371
sensory neuroses of, hysteria and, 364
singing voice and, 531
spasm of, 367
brain tumor and, 367
foreign bodies and, 367
globus hystericus and, 367
in hydrophobia, 367
prognosis of, 367
symptoms of, 367
treatment of, 367
stenosis of, 362
syphilis of, 317
teratoma of, 368
tuberculosis of, 307
Phlegmonous angina in diphtheria, 482
diphtheria, 481
laryngeal diphtheria, 484
laryngitis, acute, 458
lingual tonsillitis, acute, 349
rhinitis, 289
tonsillitis, 404
Phonation, 23
in laryngeal paralysis, 519
Phonatory spasm, 509
Phthisis, pharyngeal, 308
"Pigeon chest," adenoids and, 347
Pilocarpine in acute catarrhal laryngitis, 472
in atrophic laryngitis, 472
Pischel's collodion dressing, 144
Pleurisy, laryngeal paralysis and, 515, 518
Plica salpingopharyngeus, 696
supratonsillaris, 385, 396, 402, 408
tonsillaris, 385, 396, 402, 408, 425
triangularis, 387, 402, 419
Plicotomy, 682
Pneumococcus in membranous laryngitis, 460
Pneumomassage, 40, 42, 125
for acute catarrhal otitis media, 715
in hyperostosis of bony capsule of labyrinth, 741
in Ménière's symptom complex, 1004

Pneumonia, chronic bulbar paralysis and, 366
foreign bodies in respiratory passages and, 578
hyperemia of labyrinth and, 1000
inspiration, operations for laryngeal carcinoma and, 575
laryngeal paralysis and, 516
lobar, laryngeal paralysis and, 516
metastatic, in thrombosis of lateral sinus, 812
tracheoscopy and, 584
Polar granules in diphtheria, 477
Politzerization, 694, 699
Politzer's acoumeter, 1029
bag, 699, 700
method of inflation of tympanic cavity, 699
modified method of inflation of tympanic cavity, 700
Pollen of plants, hyperesthetic rhinitis and, 257
Polypi, defective hearing from, 631, 632
diffused hypertrophic laryngitis and, 465, 467
hyperesthetic rhinitis and, 256
nasal, 269. *See* also Myxoma of nose.
sinuitis and, 177, 180, 184, 189
within "vicious circle" of nose, 269
Postauricular fistula, closure of, 992
for removal of foreign bodies in ear, 639
Postdiphtheritic paralysis, 486
respiration in, 486
Postnasal "dropping," significance of, 173
Potassium iodide in actinomycosis, 328
in pachydermia laryngis, 500
subglottic stenosis and, 507
Pouch of hypopharynx, 595
of larynx, dilatation of, 501
Poulticing, 123
Powder insufflator, 50
Pregnancy, tuberculous laryngitis and, 314
Pressure, atrophic rhinitis due to, 155
Price-Brown's operation for deviations of nasal septum, 81
Primary mastoiditis, acute, 823
Prolapse of ventricle of Morgagni, 502
Prominentia canalis facialis, 608
semicircularis lateralis, 609
Promontorium, 608
Prussak's space, 610, 717
suppuration of, 612
Pseudocroup, 455
Pseudodiphtheria bacilli in diphtheria, 478
Pseudomembrane in diphtheria, 478
appearance of, 479
effect of antitoxin on, 489
formation of, 479
Pseudomembranous angina, 400
croup, 459
diagnosis of, from acute laryngitis in children, 457
Pseudomembranous sore throat, 366
"Pseudotabes, diphtheritic," 486
Puberty, influence of, on voice, 525
Puerperal fever, hyperemia of labyrinth and, 1000
Pulmonary gangrene from infection through tonsils, 381, 382
Puncture, lumbar, 790
Pynchon-Golding-Bird's curette, 344
Pynchon's cautery dissection of tonsils, 429
massage apparatus, 41
submerged tonsil, 408
tonsil hemostat, 422
Pyogenic diseases, intracranial, of otitic origin, 789

Q

Quinine, hyperemia of labyrinth from, 1000
Quinlan's forceps, 344
Quinsy, 404. *See* also Peritonsillitis.

R

Radiant light in treatment of suppurative otitis media, 775
Radical mastoid operation, 760, 764, 773, 833
Radiotherapy in lupus of nose, 305
for tuberculosis of nose, 308
Radium in lupus of nose, 305
in tuberculosis of larynx, 315
Ray fungus, 325
Reaction of inflammation, 110
Recessus epitympanicus, 608, 609
Rectal alimentation in intubation, 499
operations for laryngeal cancer and, 575
anesthesia for laryngofissure, 563
Reflex, cardiac, in operations for carcinoma, 565
neuroses, nasal, 254
phenomena of nasal origin, 21
toxemic, 29, 30
Refraction, disorders of, in sinuitis, 185
Reissner's membrane, 615
Resection in adenoids, 336, 345
submucous, of nasal septum, 82, 100
Respiration in acute catarrhal laryngitis, 453
in laryngeal diphtheria, 483
methods of, improper, 532
nasal, 23
in postdiphtheritic paralysis, 486
Respiratory functions of nose, 23
passages, foreign bodies in, 577
tract, upper, epithelium of, 450
function of, 26
infections of, 32
inflammation in, 450
inoperable cancer of, 377

Retropharyngeal abscess, 359
boric acid in, 361, 362
diagnosis of, 359
from aneurysm, 359
from membranous laryngitis, 461
etiology of, 359
iodoform emulsion in, 360, 361, 362
operation for, 360, 361
prognosis of, 360
symptoms of, 359
treatment of, 360
Reverdin's needle, 859
Rheumatic diathesis, chronic pharyngitis and, 354, 355
fever from infection through tonsils 381, 382
nose and, 29
paralysis of auditory nerve, 1015
Rheumatism, acute articular, acute lacunar tonsillitis and, 398
operations on tonsils and, 416
chronic lacunar tonsillitis and, 403
neuroses of pharynx and, 365
Rhinal hydrorrhea, 265
Rhinitis, acute, 130
constitutional dyscrasias and, 130
etiology of, 130
pathology of, 133
prognosis of, 134
specific fevers and, 130
"swell bodies" in, 131
symptoms of, 133
treatment of, 134
with leukodescent lamp, 136
atrophic, 154
cyanotic congestion and, 154, 155
due to pressure, 155
etiology of, 155
prognosis of, 155
symptoms of, 155
treatment of, 155
suppurative sinuitis, 156
etiology of, 154
ozena in, 156
paraffin injections in, 158
simple, 155
symptoms of, 157
treatment of, 157
chronic, with collapse of erectile tissue, 153, 154
dry, 154
with turgescence, 137
electrocauterization for, 139
etiology of, 137
pathology of, 139
prognosis of, 139
submucous cauterization for, 141
symptoms of, 138
treatment of, 139
Rhinitis, hyperesthetic, 254
alcohol injections for, 263
climatic influence and, 256
deflection of septum and, 255
dizziness in, 255
etiology of, 254
exciting causes of, 256
geographical distribution of, 256
maxillary sinuitis and, 255
pathology of, 257
pollen of plants and, 257
polypi and, 256
predisposing causes of, 254
prognosis of, 258
symptoms of, 257
treatment of, 259
with Dunbar's serum, 262
of dyscrasias in, 259
of local marked lesions in, 259
of neuroses in, 259
palliative, 261
protective, 260
serum, 262
Stein's, 263
hyperplastic, 142, 147, 267
etiology of, 147
prognosis of, 149
rhinoscopy in, anterior, 148
symptoms of, 148
treatment of, 151
hypertrophic, 142
electrocautery, 144
etiology of, 142
pathology of, 142
prognosis of, 143
"swell bodies" in, 143
symptoms of, 143
treatment of, 144
mucopurulent, 154
mucous, simple, 154
obstructive, 142
phlegmonous, 289
suppurative, 160
Rhinolalia pata, paraffin injection in, 298
Rhinorrhea, cerebrospinal, 265, 266
etiology of, 266
treatment of, 267
Rhinoscleroma, 286
bacteriology of, 286
definition of, 286
diagnosis of, from discrete hypertrophic laryngitis, 469
etiology of, 286
pathology of, 287
Röntgen rays in, 288
symptoms of, 287
thiosinamin in, 288
treatment of, 288
Rhinoscopy, anterior, in hyperplastic rhinitis, 148
Rhodes' tonsil punch forceps, 427
Richards' method of adenectomy, 344
operation upon labyrinth, 971–974

Richards' operation upon vestibule, 971
Right-angle knife, Ballenger's, 417
Rinné test in acute catarrhal otitis media 711
suppurative otitis media, 747
in adhesive processes of middle ear, 728
for hearing, 623, 624
in hyperostosis of bony capsule of labyrinth, 739
in Ménière's symptom complex, 1004
in syphilis of internal ear, 1007
Rivinian segment, 610
acute suppurative otitis media and, 743
Robertson's operation on tonsils, 428
tonsil scissors, 428
Roe's operation for deviation of nasal septum, 77
Röntgen rays, 127
in lupus of nose, 305
in lymphadenoma of tonsils, 444
in rhinoscleroma, 288
in tuberculosis of larynx, 313
Rosenmüller's fossa, 333, 338, 340, 606, 684, 685, 696
Rose's position, 282, 372, 418, 594
Round window, 608
Round-cell sarcoma of pharynx, 374
Ruault's tonsil punch forceps, 428
Rupture of membrana tympani, 670
Russell's fuchsinophiles, 287
Russian perforator, 827

S

Saccule, cristæ of, 870
Saddle nose, paraffin injections of, 298
Sajous' laryngeal forceps, 467, 469
applicator, 464
Salicylic acid, hyperemia of labyrinth from, 1000
Salivary fistula, 378
Salpingitis, 684
Salpingopharyngeal fold, 332
Santorini, fissures of, 601
Sarcoma of auricle, 652
diagnosis of, 653
prognosis of, 653
treatment of, 653
of larynx, diagnosis of, from chronic laryngitis, 473
of nose, 281
diagnosis of, 281
Ollier's operation for, 282
prognosis of, 281
treatment of, 281
of pharynx, 374
diagnosis of, from actinomycosis of pharynx, 327
varieties of, 374, 375
Satyriasis, 321
Saw, antrum, Vail's, 226
Bosworth's, 71
Fetterolf's, 80
Scalp, erysipelas of, cerebellar nystagmus and, 873
Scarification in acute phlegmonous laryngitis, 459
Scarlet fever, hyperemia of labyrinth and, 1000
perforation of nasal septum in, 103
tonsils and, 381
Scheibel's suture forceps, 859
Schroetter's laryngeal tubes, 505
Schwabach's test for hearing, 739
Schwartze's point of tenderness in simple acute mastoiditis, 766
Scissors, Holmes', 150, 151
Sclerosis of mastoid bone in chronic mastoiditis, 771
of middle ear, 724
multiple, laryngeal paralysis and, 521
Scoliosis, laryngeal paralysis and, 518
Screw-worms in nose, 289
Secretion, internal, in tonsils, 392
Seiler's solution, 55, 355, 549
Semicircular canals, physiology of, 861
function of, 613
relation of, to facial nerve, 835
hyperostosis of, 740
Semon's law, 519, 556
Sensory aphasia, 1020
nerves of nose, 19
neuroses, nasal, 254
Septic angina in diphtheria, 482
diphtheria, 481
laryngeal, 484
Septicemia, diphtheria and, 481
Septum forceps, Hurd's bone, 97
gouge, Ballenger's, 96
nasal, 17
nasi. See Nasal septum.
speculum, Ballenger-Foster, 97
Serous meningitis, 790
Serum treatment of hyperesthetic rhinitis, 262
Sexton's foreign body forceps, 639
ossiculectomy knives, 821
"Shepherd's crook" incision in radical mastoid operation, 841, 843
Shock in operations for laryngeal carcinoma, 574
Shrapnell's membrane. *See* Membrana flaccida.
Shutz adenotome, 344
Siebermann's Y-incision in radical mastoid operation, 845
Siegle's otoscope, 603, 610, 717, 728, 745, 750, 771, 783, 788, 1004
Silent croup, 460
Simpson's nasal sponge splint, 97
Simulated deafness, 1013
Singer's nodules, 469, 544, 545
Singing voice, 525

Sinuitis, 161, 162, 164, 166, 168
acute catarrhal laryngitis and, 450
anosmia in, 186
auditory functions and, disturbance of, 188
caries of teeth and, 177
catarrhal, acute, treatment of, 190
chronic, treatment of, 190
cerebral hemorrhage and, 187
chronic suppurative otitis media and, 758
coryza and, 188
diagnosis of, differential, 169
diffused hypertrophic laryngitis and, 465, 467
digestive disturbances from, 27
dizziness in, 163, 186
due to pressure in middle turbinated region, 188
empyema in, 169
Escat's position in, 170
etiology of, 176
exciting causes of, 177
headache in, 163, 171, 185
hypothetical cases of, 170, 175
intracranial complications in, 164
maxillary, hyperesthetic rhinitis and, 255
meningitis and, 164, 180
nasal causes of, 177
ocular symptoms in, 164, 169, 186, 187
optic neuritis and, 187
pain in, 163, 168, 185
parosmia in, 186
pathology of, 178
polypi and, 177, 180, 184, 189
pus in, 163, 165, 168, 170, 171, 184
refraction in, disorders of, 185
singing voice and, 534
skiagraphy in, 162, 163, 182, 183
special senses in, disturbance of, 186
strabismus and, 174
suppurative, atrophic rhinitis due to, 156
chronic, due to obstructive lesions, 189
treatment of, 191
symptoms of, 162, 163, 181, 184, 185
syphilis and, 176
tenderness on pressure in, 162, 163, 171, 185
transillumination in, 171, 181, 182
treatment of, 188
indications for, 188
by irrigation, 191–195
by lavage, 191–195
by negative air pressure, 196
operative, 199, 207
tuberculosis and, 176
vaccine therapy for, 196, 197
vertigo in, 163, 186
Sinus, accessory, of nose, 19, 161. *See* also Sinus, ethmoidal, frontal, etc.
Sinus, accessory of nose, disease of, vaccine therapy in, 196
division of, 161, 167
empyema of, 169
nasal secretions in, 31
inflammation of. *See* Sinuitis.
irrigation of, 195
Loeb's projections of, 162
operations for, 202
surgery of, 199
indications for, 205
cavernous, thrombosis of, 817
disease of, asthenopia in, 205
hay fever and, 207
intracranial complications in, 207
ocular symptoms in, 205, 206
skiagraphic indications in, 206
ethmoidal, anterior, 164
anatomy of, 164
inflammation of, 164. *See* also Sinuitis.
operations for, choice of, 204, 205
pus in, 165
skiagraphy in, 165
suppuration in, 164, 165
lavage of, 194
operations for, 239, 247
Ballenger's, 239, 242
external, 250
Moure's external, 250
orbito-ethmoid, 251
posterior, 167
anatomy of, 167
inflammation of, 168. *See* also Sinuitis.
operations for, choice of, 203
frontal, 161
anatomical variations of, 161
inflammation of, 161. *See* also Sinuitis.
dizziness in, 163
headache in, 163
intracranial complications in, 164
meningitis and, 164
mucous discharge in, 163
ocular symptoms in, 164
pain in, 163
pus in, 163
redness in, 163
skiagraphy in, 162
swelling in, 163
symptoms of, 162, 163
tenderness to pressure in, 162, 163
vertigo in, 163
irrigation of, 171
lavage of, 191
operations for, 207
after-treatment in, 209, 212, 213, 220, 223

Sinus, frontal, operation for, Ballenger's intranasal, 207
Beck's double osteoplastic, 219
choice of, 204, 205
external, 215
Good's, 212
Hajek-Luc's, 216
Halle's, 210
Ingals', 214
Killian's, 221
Kuhnt-Luc's, 217
Kuhnt's, 217
Kuster's osteoplastic, 218
probing of, 172, 192
surgery of, 202, 207
lateral, thrombosis of, 789, 810, 984
maxillary, anatomy of, 166
inflammation of, 166. *See* also Sinuitis.
etiology of, 166, 167
teeth and, 167
irrigation of, 170
lavage of, 192
technique of, 193
through alveolar process, 194
through canal external to teeth, 194
operations for, 224
alveolar, 230
Caldwell-Luc's, 231
Canfield-Ballenger's antrum, 233
choice of, 203
Cooper's, 230
Corwin's, 227
Denker's, 233
extranasal, 230
intranasal, 224
Kuster's, 231
Myles', 225
Vail's, 226
relation of intra-orbital nerve to, 167
to teeth, 230
transillumination of, 277
sphenoidal, 168
anatomy of, 168
inflammation of, 168. *See* also Sinuitis.
ocular symptoms in, 169
pain in, 168
pus in, 168
lavage of, 194
operations for, Ballenger's, 249
choice of, 203
relation of optic nerve to, 169
thrombosis, cholesteatoma and, 763
exanthemata and, 746
hyperemia of labyrinth and, 1000
otitis media and, 748, 752
tonsillaris, 387
Skiagraphy in disease of sinuses, 206
Skiagraphy for foreign bodies in respiratory passages, 578, 586
in sinuitis, 162, 165, 182, 183
Skin in diphtheria, 486
Sloughing of mucous membrane in diphtheria, 479
Sluder's guillotine operation on tonsils, 430
contra-indications to, 436
operation for deviation of nasal septum, 74, 75
Smallpox, deaf-mutism and, 1027
Smell, sense of, 22
Smoking, diffused hypertrophic laryngitis and, 465, 467
Snare, Krause's nasal, 149
removal of myxoma of nose with, 272
Soft hypertrophies of nasal septum, 70
Sore throat, clergyman's, 353. *See* also Pharyngitis, chronic.
pseudomembranous, 366
Spasm, glottic, complete, in adult, 511
of glottis, 509. *See* also Laryngismus stridulus.
from irritation, reflex, 508
of trunk of recurrent laryngeal nerve, 508
of larynx, 508, 509
adductor, 509. *See* also Laryngismus stridulus.
in membranous laryngitis, 460
in tetanus, 508
tonic, of central origin, 508
of pharynx, 367
phonatory, 509
of tensor muscles of vocal cords, 509
Spasmodic laryngeal cough, 510
Spasmus glottidis, 509. *See* also Laryngismus stridulus.
Speculum, Allen's nasal, 97
Ballenger-Foster septum, 97
Jackson's slide, 580
Spedalskhed, 321. *See* also Leprosy.
Speech in deaf-mutism, 1029
defects of, 536
adenoids and, 335, 539
aprosexia and, 538, 539
classification of, 537
deaf-mutism and, 542
emotion and, 536
mentality and, 536, 537
origin of, abdominal, 540
epipharyngeal, 539
faucial, 539
laryngeal, 540
lingual, 539
nasal, 538
thoracic, 540
Sphenoidal sinus, 168
Sphenoiditis, acute catarrhal otitis media and, 708
atrophic laryngitis and, 471
chronic moist catarrhal otitis media and, 722

Sphenoiditis, leptomeningitis and, 794
Sphenomaxillary fissure, growths in, 371
Sphenopalatine ganglion, 19, 20
Spinal accessory nerve, nuclei of, lesions of, laryngeal paralysis from, 521
cord, lesions in, due to diphtheria, 479
Spindle-cell sarcoma of pharynx, 375
Spirillum in membranous laryngitis, 460
Spitting blood, 472
Spleen in diphtheria, 480
Splints, Mayer's nasal tube, 79
Simpson's nasal sponge, 97
Spokeshave, 146
Chaleway's, 72
in submucous resection of nasal septum, 93
Spray tubes, 43, 44
De Vilbiss', 44
Hawley's, 44, 45
Spurious hemoptysis, 472
Stacke protector, 609, 994
Stammering, 537
pulmonary tuberculosis and, 540
Stapedectomy in hyperostosis of bony capsule of labyrinth, 741
Stapes, attachment of, 606
foot-plate of, ankylosis of, defective hearing from, 648, 649
hyperostosis of, 739
Staphylococcus albus in acute infectious epiglottitis, 448
diagnosis of, from Klebs-Loeffler bacillus, 478
in membranous laryngitis, 460
Stein's treatment of hyperesthetic rhinitis, 263
Stenosis, alternating, of nose, 137
of Eustachian tube, 731
of larynx, 501, 502
in laryngeal diphtheria, 483
laryngofissure in, 505, 506, 565
leprous, 504
lupous, 504
Schroetter's laryngeal tubes in, 505
subglottic, 507
syphilitic, 502
chronic edema in, 503
cicatricial contraction in, 503
cough in, 503
dyspnea in, 503
hyperplastic growths in, 503
iodonucleoid in, 503
laryngofissure in, 503
papillary growths in, 503
voice in, 503
webs in, 503
tracheotomy in, 504, 505, 506
traumatic, 504
treatment of, 504
tuberculous, 504
ventricular eversion and, 504
Stenosis, nasal, 31
acute catarrhal laryngitis and, 450
respiration and, 17
in obstructive deviations of nasal septum, 65
of pharynx, 362
subglottic, potassium iodide and, 507
Sterilizer for instruments and gauze, 47
Stethoscope test for simulated deafness, 1014
Stomach, diphtheria of, 485
Strabismus, sinuitis and, 174
Street's tonsil hypodermic syringe, 418
Streptococcus aureus in acute infectious epiglottitis, 448
diagnosis of, from Klebs-Loeffler bacillus, 478
in infection of tonsils, 438
in membranous laryngitis, 460
Streptodiphtheria, 481
of nose, 483
Streptodiphtheritic angina in diphtheria, 482
Stricture of external auditory meatus, 663
Stoerk's blennorrhea, 286, 468
Stubbs' operation for adenoids, 343
Stuttering, 537
Stylomastoid foramen, 995
Stylopharyngeus muscle, 995
Subacute mastoiditis, 769
Subdermal suture, Halsted's, 220
Subglottic laryngitis, 456
space, chondromata of, 546
cysts of, 546
importance of, 507
neoplasms of, 546
electrocautery for, 546
Subhyoid pharyngotomy, 565
Subjective laryngitis, chronic, 468
noises, 1018
"Submerged tonsils," 634
of Pynchon, 408
Submucous resection of nasal septum, 82–100
Subperiosteal mastoid abscess, 770
Sudden death after operations for laryngeal carcinoma, 574
Sulcus tympanicus, 610
Sunstroke, chronic bulbar paralysis from, 366
Suppuration of atrium of ear, 781
of attic of ear, 781, 783
of cervical glands, 398
of labyrinth, 1008
Suppurative diseases of ear, lymphatic glands and, 28
otitis media, acute, 742
rhinitis, 160
sinuitis, treatment of, 191
Supraglottic laryngitis, 456
Supratonsillar fossa, 384, 391, 419
"Swell bodies," 18, 24. *See* also Turbinated bones.
in acute rhinitis, 131

"Swell bodies" in hypertrophic rhinitis, 143
Swivel knife, 151
Ballenger's mucosa, 105
in submucous resection of nasal septum, 91, 92, 96
turbinotome, Ballenger's, 146
Syncope, bronchial, 511
laryngeal, 511
Syphilis, acquired malformations of larynx and, 501
acute catarrhal laryngitis and, 450, 453
otitis media and, 708
infectious epiglottitis and, 448
arteriosclerosis of labyrinth and, 1005
deaf-mutism and, 1025
destruction of uvula and, 362
diagnosis of, from simple acute pharyngitis, 352
edema of larynx and, 462
of external ear, condyloma in, 321
of fauces, 317
hemorrhagic laryngitis and, 472
hyperostosis of external auditory meatus and, 662
of internal ear, 1007
course of, 1008
deafness in, 1008, 1009
diagnosis of, 1008
from hyperostosis of bony capsule of labyrinth, 1008
dizziness in, 1007
nystagmus in, 1007
ozena in, 1007
pathology of, 1007
prognosis of, 1008
Rinné test in, 1007
symptoms of, 1007
tinnitus in, 1007
treatment of, 1008
Weber test in, 1007
laryngeal paralysis and, 519, 521, 522
of larynx, 319
diagnosis of, from chronic laryngitis, 473
from tuberculosis of larynx, 312
pathology of, 319
symptoms of, 319
treatment of, 320
malformations of pharynx and, 362
neoplasms of larynx and, 545
of nose, 33, 317
pachydermia laryngis and, 500
papilloma of larynx and, 546
of pharynx, 317
diagnosis of, from actinomycosis of pharynx, 328
from tuberculosis of the pharynx, 308
prolapse of ventricle of Morgagni and, 502
Syphilis, sinuitis and, 176
stenosis of larynx and, 501, 502
throat in, 33
of tonsils, 317
Syphilitic coryza, 33
otitis interna, 1007
Syringe, Beck's paraffin, 299

T

Tabes dorsalis, ear in, 32
throat in, 32
Tachycardia in diphtheria, 481
nasal, 268
Teasing needle for Thiersch's grafting, 855
Teeth, adenoids and, 345
caries of, sinuitis and, 177
relation of, to maxillary sinus, 230
singing voice and, 532
Tegmen tympani, drainage through, in surgery for abscess of brain, 976
necrosis of, 838
relations of, 606, 608
Temporal bone, surgery of, 819
cells, distribution of, 610
Tenotomy of tensor tympani muscle, 677
Teratoma of pharynx, 368
treatment of, 369
Tetanus, spasm of larynx in, 508
"Theatre pain," 185
Thiersch grafts, application of, after radical mastoid operation, 854, 855, 856
for deformity of nose, 293
razor for, 854
spatula for, 855
Thimble gag, 597
Thiosinamin in rhinoscleroma, 288
Thornwaldt's disease, 331, 347
Throat, bleeding in, 472
general medicine and, 26
in multiple sclerosis, 32
in syphilis, 33
in tabes dorsalis, 32
Thrombophlebitis, infection through tonsil and, 382
Thrombosis, 809
bacteriology of, 809
of cavernous sinus, 817
symptoms of, 818
treatment of, 818
of ear, embolic abscesses and, 810
of jugular bulb, 816
Whiting's test for, 816
of lateral sinus, 789, 810
acute primary mastoiditis and, 824
bacteremia and, 814
blood count in, 814
cultures in, diagnostic value of, 813
diagnosis of, early, 812

Thrombosis of lateral sinus, blood in etiology of, 810
headache in, 812
metastatic enteritis in, 812
meningitis in, 812
pneumonia in, 812
prognosis of, 816
stages of, 812
symptoms of, 812
tenderness over mastoid emissary vein in, 812
treatment of, surgical, 983–986
pathology of, 810
Thymic asthma, 509. *See* also Laryngismus stridulus.
Thymus gland in diphtheria, 480
enlarged, laryngismus stridulus and, 509
Thyroid extract in hyperostosis of bony capsule of labyrinth, 740
gland, enlarged, laryngeal paralysis and, 521
Thyroidectomy, laryngeal paralysis and, 521
Thyrotomy, 502, 546, 558, 561, 564
Timbre of voice, 528, 530
Tinnitus in adhesive processes of middle ear, 727
in arteriosclerosis of labyrinth, 1005
aurium, 615, 1018
in acute diffuse suppurative manifest labyrinthitis, 945
alcohol and, 720, 727
in chronic moist catarrhal otitis media, 719, 720
prognosis of, 1020
treatment of, 1020
in concussion of labyrinth, 1011
in deaf-mutism, 1030
from disease of cochlea, 63
in hyperemia of labyrinth, 1000
in hyperostosis of bony capsule of labyrinth, 739
in Ménière's disease, 1002
physiological law of, 607
in syphilis of internal ear, 1007
syphilitic condyloma of external ear and, 321
Tobacco, chronic laryngitis and, 465
pharyngitis and, 353, 354
hyperacuteness of hearing and, 1016
in hyperemia of labyrinth, 1000
laryngeal carcinoma and, 554
neuroses of pharynx and, 364
vestibular nystagmus and, 335
Tone blindness, singing voice and, 533
"islands," 615
perception of, 618
Tongue, base of, diseases of, 331
singing voice and, 527
Tongue-tie, 527, 532
Tonsil, absorptive properties of, 385
actinomycosis of, 326
Tonsil, adhesions of, 387
anatomy of, 383
angioma of, 442
bacteriolysis in, 386
as barriers to microörganisms, 380, 381
blood supply of, 390
calculus of, 403
carcinoma of, 444
crypts of, 384
cystoma of, 443
"decapitation" of, 392
diseases of, 379
écraseur, Ballenger's, 421
enucleation of, Beck-West method of, 426, 427
epithelium of, 385
Eustachian, 633
tube and, 415
extirpation of, by external route, 445, 446
faucial, acute catarrhal otitis media and, 708
diffused hypertrophic laryngitis and, 467
lymphoid tissue of, 383, 384
fibro-enchondroma of, 443
fibroma of, 442
forceps, Ballenger's, 418
function of, 392
"germ centres" of, 384
hemostat, Boetcher's, 422
Pynchon's, 422
horny material in, 409
hyperkeratosis of, 408
tonsillitis and, 410
hyperplasia of, 408
hypertrophy of, 408
defective hearing from, 634
treatment of, 408
hypodermic syringe, Street's, 418
infection of, rheumatic fever and, 381, 382
streptococcus in, 438
thrombophlebitis and, 382
through, endocarditis and, 382, 391, 398
pulmonary gangrene and, 381, 382
inflammatory diseases of, 395
general considerations of, 395
internal secretion in, 392
invasion of, by microörganisms, 379, 380, 381
knife, Ballenger's, 424
laryngeal, in acute lacunar tonsillitis, 398
leptothrix in, 408
lingual, 348
hypertrophy of, 349, 350
lymphoid tissue of, 348, 351
removal of, 350
lipoma of, 442
local lesions of, acute lacunar tonsilitis and, 396

Tonsil, lymphadenoma of, in Hodgkin's disease, 444
lymphatic infection through, 379, 381, 382, 387, 388, 390
lymphatics of, 387
mycosis leptothricia of, 408
neoplasms of, 442, 444
operations on, 416
acute articular rheumatism and, 416
Ballenger's, with scalpel, 423
cocaine anesthesia in, 418, 420, 429
complete, 416
Ballenger's, with right-angle knife and écraseur, 417
with capsule intact, 431
with tonsillotome and punch forceps, 427
complications of, 435
extirpation by external route, 445, 446
by finger dissection, 441
hemophilia and, 416
hemostat for, Boetcher's, 422
Pynchon's, 422
indications for, 414
infection following, 436, 437
mouth-gag in, 418, 437
reverse Trendelenburg, 440
Pynchon's cautery dissection, 429
Robertson's, 428
sequelæ of, 437
Sluder's guillotine, 431
Street's hypodermic syringe for, 418
tonsillectomy, 423, 441
tonsillotomy, 431, 436
papilloma of, 442
pharyngeal, in acute lacunar tonsillitis, 398
angina lacunaris of, 331
lacunar inflammation of, acute, 331, 332
as portals of infection, 379
punch forceps, Farlow's, 427
Rhodes', 427
Ruault's, 427
removal of, 383, 392
hemorrhage and, 392, 393
sarcoma of, diagnosis of, from carcinoma, 445
scarlet fever and, 381
scissors, Robertson's, 428
"submerged," 634
of Pynchon, 408
surgery of, 414
syphilis of, 317
syringe, Ballenger's, 441
tuberculosis of larynx and, 309
latent, 379
primary, 379
as vestigial organs, 380
Tonsillar lymphatic vessels, 387, 388
Tonsillar ring, 331, 338, 381
Tonsillectomy, 423, 441
anesthesia in, 417
cervical cellulitis and, 392
with scalpel, 423
Tonsillitis, acute catarrhal laryngitis and, 450
cryptic, 396
follicular, acute, 396
hyperkeratosis of tonsils and, 410
infective, 396
lacunar, acute, 396
acute articular rheumatism and, 398
age and, 396
bacteriology of, 396
catching cold and, 396
complications of, 396
diagnosis of, 398
from diphtheria, 398
erythema multiforme and, 399
nodosa and, 399
etiology of, 396
laryngeal tonsils in, 398
local lesion of tonsil and, 396
pathology of, 397
pharyngeal tonsils in, 398
sequelæ of, 398
specific fevers and, 397
surgical trauma and, 396
symptoms of, 397
objective, 397
subjective, 397
treatment of, 397
chronic, 402
cholesterin in, 402
etiology of, 402
rheumatism and, 403
symptoms of, 402
treatment of, 403
lingual, acute catarrhal, 349
lacunar, 349
phlegmonous, 349
phlegmonous, 404. *See* also Peritonsillitis.
ulcerative, 400
Tonsillotome, 427
Mathieu's, 417
Tonsillotomy, 431, 436
Torticollis in meningitis serosa, 790
Toxemia, nystagmus and, 907
Toxic angina in diphtheria, 482
Trachea, diphtheria of, 484
foreign bodies in, 577
Tracheal diphtheria, 484, 578
Tracheobronchoscope, Jackson's self-illuminating, 591
Tracheobronchoscopy, 579
lower, 594
indications for, 594
position of patient in, 594
upper, 589
anesthesia for, 589

Tracheobronchoscopy, upper, introduction of slide speculum in, 591
of tracheobronchoscope in, 591
position of head for, 590
preparation of patient in, 589
removal of foreign bodies in, 593
of secretions in, 593
topical applications in, 593
Tracheoscopy, 581
anesthesia for, 581
Boyce's position for, 581, 582
complications of, 584
inspection in, 584
introduction of tube in, 582
pneumonia and, 584
position of patient in, 581
preparation of patient for, 581
removal of foreign bodies in, 584
Tracheotomy, 490
in abscess of larynx, 464
accidents in, 491
in acute phlegmonous laryngitis, 559
catarrhal laryngitis, 454, 455
infectious epiglottitis, 449
laryngitis in children, 458
after-effects of, 491
after-treatment of, 492
complications of, 491
in diphtheria, 486, 490
in discrete hypertrophic laryngitis, 469
in edema of larynx, 463
high, 490
incision in, 490
indications for, 449, 454, 455, 458, 459, 460, 463, 464, 469, 490, 505, 506, 520, 523, 524, 546, 548, 579,
in laryngeal paralysis, 520, 522, 523, 524
low, 490, 594
after-treatment in, 595
introduction of tracheobronchoscope in, 594
in membranous laryngitis, 460
in stenosis of larynx, 504, 505, 506
tube, 490
introduction of, 491
Trachoma of vocal cords, 469
Transillumination, 171, 172, 174
of maxillary sinus, 279
in sinuitis, 171, 181, 182
Traumatic circumscribed labyrinthitis, 906
Trautmann tongue flap method in radical mastoid operation, 846
Trendelenburg position, reverse, for operations on tonsils, 440
Trephine, Bishop's, 229
circular, 978
Trocar, Krause's, 225
Trousseau's dilator, 594
Trypsin treatment for malignant neoplasms, 376
Tubal catarrh, 684
adenoids and, 684, 685
epipharyngitis and, 684
etiology of, 684
prognosis of, 685
symptoms of, 685
treatment of, 685, 686, 687
disease, acute catarrhal otitis media and, 708
Tuberculosis, acquired malformations of larynx and, 501
acute catarrhal laryngitis and, 450
otitis media and, 708
infectious epiglottitis and, 448
deaf-mutism and, 1030
edema of larynx and, 462, 463
of fauces, 307
hemorrhagic laryngitis and, 472
laryngeal paralysis and, 515
of larynx, 308
aphonia in, 311
"ashen-gray" color in, 310, 311
cocaine in, 313
Cooper-Hewitt light for, 313
cough in, codeine for, 312
curettage for, 314
diagnosis of, 312
from carcinoma of larynx, 312
from chronic laryngitis, 473
from lupus of larynx, 312
from syphilis of larynx, 312
dysphagia in, 308
dyspnea in, 308
etiology of, 309
menthol in, 313
orthoform in, 313
pathology of, 310
prognosis of, 312
radiotherapy for, 313
radium in, 315
Röntgen rays in, 313
symptoms of, 311
tonsils and, 310
treatment of, 312
ultraviolet rays in, 313
of mastoid process, 314
of middle ear, 314
destruction of bony tissue of labyrinth in, 316
of mastoid process in, 316
diagnosis of, 315
Koch's tuberculin in, 315
prognosis of, 316
symptoms of, 315
treatment of, 316
of nose, 307
perforation of septum and, 307
radiotherapy for, 307
pachydermia laryngis and, 500
papilloma of larynx and, 546
of pharynx, 307
diagnosis of, 308

Tuberculosis of pharynx, diagnosis of, from syphilis, 308
prognosis of, 308
symptoms of, 308
treatment of, 308
prolapse of ventricle of Morgagni and, 502
pulmonary, stammering and, 540
sinuitis and, 176
of tonsils, 379
Tuberculous cervical glands, 316
disease of pharynx, diagnosis of, from actinomycosis of pharynx, 327
laryngitis, 308
in pregnant women, 314
membrana tympani, 315
otitis, 316
stenosis of larynx, 504
Tuning forks, Bezold-Edelmann, 617
Turbinal region of nose, middle, obstruction of, 119
Turbinated bones, hypertrophy of, 142
middle, enlargement of, asthenopia due to, 35
hemorrhage from, 153
obstruction of nose due to, 129
removal of, with Ballenger's turbinotome, 150
with Holmes' scissors, 150
with scissors and snare, 150
with swivel knife, 151
of nose, 17, 18
in obstructive deviations of nasal septum, 63
region, middle, sinuitis due to pressure in, 188
Turbinectomy with Ballenger's knife, 239
Turbinotome, Ballenger's, 150
swivel, 146
Turbinotomy, meningitis following, 242
middle, meningitis after, 153
Turning reactions in diffuse latent suppurative labyrinthitis, 950
test of vestibular apparatus, 877
Twisted or crooked nose, Ballenger's operations for, 291, 292, 293
Tympanic cavity, 606
auscultation of, 696, 697, 702
contents of, 606
curettage of, dangers of, 836
function of, 611
infection of, brain abscess and, 608
meningitis and, 608
inflammatory diseases of, 703
inflation of, 692
in acute catarrhal otitis media, 714, 715
in adhesive processes of middle ear, 728, 729
Tympanic cavity, inflation of, diagnostic value of, 694, 698, 701
manometer in, 697
methods of, 694
American, 700
by catheterization, 695
by external mechanical massage, 701
Politzer's, 699
modified, 700
Valsalva's, 694
principles of, 692, 693
relations of, 606
to facial nerve, 835
removal of necrosed bone from, in radical mastoid operation, 838
walls of, 608
murmur in acute suppurative otitis media, 749
Tympanum, 606
Typhoid, epistaxis and, 284
fever, hemorrhagic laryngitis and, 472
hyperemia of labyrinth and, 1000
laryngeal paralysis and, 515

U

Ulcerative tonsillitis, 400
Ultraviolet rays, 313
in lupus of nose, 306
in tuberculosis of larynx, 313
Uncinate cells, obstruction of nose due to, 119
process, 118, 174, 199
Upper tracheobronchoscopy, 589
Urine in diphtheria, 485
Uterine disease, laryngismus stridulus and, 509
Utricle, cristæ of, 870
Uvula, amputation of, 357, 358
Casselberry's operation for, 356
edema of, 355
elastic, 357
elongation of, 356
reflex cough in, 356
singing voice and, 531
symptoms of, 356
treatment of, 357
syphilitic destruction of, 362
voice and, 525, 526

V

Vaccine therapy for disease of accessory sinuses of nose, 196
for sinuitis, 196
for suppurative otitis media, 776
Vagus paralysis in diphtheria, 486
Vail's antrum saw, 226
operation upon maxillary sinus, 226

Valsalva's method of inflation of tympanic cavity, 694
Valvular lesions of heart, edema of larynx and, 462
Variola, hemorrhagic laryngitis and, 472
Varix, lingual, 350
electrocautery in, 351
glossodynia and, 351
pathology of, 351
symptoms of, 351
treatment of, 351
Vasomotor neuroses, nasal, 254
system, unstableness of, chronic laryngitis and, 466
Vegetations, adenoid, 333
Vein, jugular, internal, resection of, 987, 988
Velum palati, paralysis of, in diphtheria, 486
Ventricles of larynx, prolapse of, diagnosis of, from chronic laryngitis, 473
of Morgagni, prolapse of, 502
syphilis and, 502
tuberculosis and, 502
Vertigo in acute diffuse serous labyrinthitis, 936
circumscribed labyrinthitis, 904, 906, 932
in extradural abscess, 793
intractable and unbearable, 975
laryngeal, 511
in Ménière's disease, 1002
in neurasthenics, 907
in obstructive deviations of nasal septum, 63
in sinuitis, 163, 186
Vestibular apparatus, function of, 613
qualitative estimation of, 896
quantitative estimation of, 896
tests of, 629
tests of, caloric, 881
with cold water, 883
significance of, 887
with warm water, 886
clinical problems in, 893
fistula, 888
hindrances to, 890
significance of, 890
functional, 877
galvanic, 891
significance of, 893
turning, 877
significance of, 898
nystagmus, 870
reaction in acute diffuse suppurative manifest labyrinthitis, 942
Vibrator, mechanical, 45
"Vicious circle" of nose, 199
Victor massage apparatus, 716
Vincent's angina, 400
Vocal bands, removal of, 524
cords, "cadaveric" position of, 519, 521
Vocal cords, color of normal, 466
paralysis of. *See* Paralysis, laryngeal.
spasm of tensor muscles of, 509
trachoma of, 469
Voice in acute catarrhal laryngitis, 453
in chorditis nodosa, 470
in deaf-mutism, 1029
in diffused hypertrophic laryngitis, 466
in edema of larynx, 463
excessive use of, in hemorrhagic laryngitis, 472
falsetto, 540
in fibromata of pharynx, 370
foreign bodies in respiratory passages and, 577
influence of puberty on, 525
in laryngeal diphtheria, 483
paralysis, 516, 522
in membranous laryngitis, 460
operations for laryngeal carcinoma and, 575
pillars of fauces and, 530
production, 525
range of, 525
resonators of, 529, 530
singing, adenoids and, 526, 531
chronic pharyngitis and, 531
cleft palate and, 532
defects in, causes of, 525
treatment of, 533
elongation of uvula and, 531
improper methods of respiration and, 532
laryngitis and, 534
larynx and, 527
nasal chambers and, 532
diseases and, 526, 534
papillomata and, 527
pharynx and, 531
sinuitis and, 534
teeth and, 532
tone blindness and, 533
tongue and, 527, 531
tongue-tie and, 527, 532
in syphilitic stenosis of larynx, 503
test for hearing, 619
throatiness of, 530
timbre of, 528, 530
uvula and, 525, 526
Voltolini's method for removal of foreign bodies in ear, 641
Vomer, removal of, in submucous resection of nasal septum, 94, 95
Vomiting in acute diffuse serous labyrinthitis, 936
in Ménière's disease, 1002
von Langenbeck's operation for fibroma of nose, 277
von Tröltsch's method of gargling, 686

W

Waldeyer's ring, 329, 336, 379
Walsham's operation for collapse of alæ nasi, 302
"Warty diathesis," 545
Watson's operation for deviation of nasal septum, 73, 74
Weaver's intratympanic masseur, 690
Weber test in acute catarrhal otitis media, 711
suppurative otitis media, 745
on bone conduction, 616
for hearing, 616, 621, 622
for impacted cerumen, 643
in syphilis of internal ear, 1007
in unilateral deafness, 622
Weber-Liel catheter, 758, 819
Wells' trocar cannula, 229
Westphal's symptoms in leptomeningitis, 795
Wet cupping, 123
Whistle, Galton-Edelmann, 321, 626
Whiting's encephaloscope, 981
incision, 825
test for thrombosis of jugular bulb, 816
Wilde's incision, 824
for acute periostitis of mastoid process, 770
Wooden tongue, 325
Word-deafness, 1020

X

X-rays. *See* Röntgen rays.

Y

Yankauer's intranasal suture for perforation of nasal septum, 107, 108
Yellow fever, hemorrhagic laryngitis and, 472

W. S. MEACHAM.